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Review Article

Microbial Flora and Periodontitis: A Comprehensive Review

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ABSTRACT:
Periodontitis can be defined as the presence of gingival inflammation at sites where there has been a pathological detachment of collagen fibers from the cementum and the junctional epithelium has migrated apically. The present review was undertaken to highlight some of the important aspects and correlation of microbial flora and periodontitis.
Key words: Microbial flora, Periodontitis.

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Introduction

Periodontitis is defined as an inflammatory disease of the supporting tissues of the teeth caused by specific microorganisms or group of specific microorganisms resulting in progressive destruction of periodontal ligament and alveolar bone with pocket formation, recession or both. The disease prevalence is 13–57% worldwide and 65–80% in India accounting to a major health problem. Bacterial DNA sequencing study has suggested that nearly 19,000 bacterial phylotypes exist in the oral cavity. Nearly, 500 bacterial strains have been recovered from subgingival plaque, but only a small number are potential pathogens. Immunological, DNA probe assessment, bacterial enzyme detection and culture are the methods available for isolation of oral bacteria, among which culture remains economical and gold standard.[1, 2]

Microbial shift and disease-associated biofilms

Just as entire microbial communities can be associated with health; current research also indicates that entire microbial communities can be associated with disease. Since more than one bacterial species may be associated with a particular disease, the traditional concept of "one germ, one disease" can be rejected or may need modification. The idea that the lack of a beneficial organism in a biofilm may be just as important as the presence of a pathogen in the contribution to disease, a hypothesis has been developed linking certain diseases to a shift in membership of the local microbiota. Microbial shift, more commonly known as dysbiosis, refers to the concept that some diseases are due to a decrease in the number of beneficial symbionts and an increase in the number of pathogens. The long-standing paradigm is that, as periodontitis develops, the oral microbiota shifts from one consisting primarily of gram-positive aerobes to one consisting primarily of gram-negative anaerobes. Recent research has indicated that dysbiosis in the oral cavity can lead to periodontitis. The development of oral dysbiosis is likely to occur over an extended period of time, gradually changing the symbiotic host–microbe relationship to a pathogenic one.[3, 4]

Role of microbial flora in pathogenesis of periodontal diseases

The microbial role in periodontal pathogenesis although is increasingly realized, to our great chagrin nothing substantive has emerged. A play of words, semantics, it is all a charade. Although the presence of *M. tuberculosis* is an indication of tuberculosis and *T. pallidum* a positive diagnosis of syphilis, there is no single microorganism, which is attributable to chronic periodontitis (CP). About 20 years ago Socransky aptly summed up the microbial etiology of periodontal disease with a disarming statement "specific bacteria of right clonal type with

essential genetic elements in numbers for that host with appropriate additional species in the right environment."[5]

The following are essentially associated with periodontitis: *P. gingivalis, T. forsythia, Aggregatibacter actinomycetemcomitans, Prevotella intermedia, Prevotella melaninogenica, Fusobacterium nucleatum, Parvimonas micra, Eikenella corrodens, Prevotella nigrescens, Capnocytophaga gingivalis, Treponema denticola, Treponema socranskii, Eubacterium nodatum* and *Campylobacter rectus*.[6]

The following organisms have also been implicated as periodontal pathogens: *Porphyromonas endodontalis, Prevotella denticola, Filifactor alocis, Cryptobacterium curtum, Eubacterium saphenum, Mogibacterium timidum, Prevotella corporis, Prevotella disiens, Peptostreptococcus magnus, Slackia exigua, Treponema maltophilum, Treponema sp. Smibert-3, Treponema lecithinolyticum, Treponema putidum sp. nov., Enterococcus faecalis, Escherichia coli* and *Bartonella sp*. Nevertheless, the relevance of certain species as etiological agents of periodontal diseases remains controversial, even among microbiologists.[7]

While it is not possible to dwell on each of the above organisms, few of them are mentioned here and the discerning reader is recommended to wade through a maze of information and draw necessary inferences accordingly.[6]

The microbiota associated with periodontal health and disease has been intensely studied for well over a century by several generations of skilled scientists and clinicians. Oral microbiota is an enormously complex and dynamic entity that is profoundly affected by perpetually changing local environments and host-mediated selective pressures. The presence of a commensal microbiota, including potential pathogens, is essential for the proper development of mucosal immunity.[8]

The normal oral flora is hence in a balance between pathogens and commensals that requires regular cleaning to be maintained. A decrease in oral hygiene is quickly followed by the build-up of oral biofilms on tooth surfaces and, if left untreated, will progress to gingival inflammation and possibly periodontitis, alveolar bone loss and loss of teeth. It is likely that differences in host-defence mechanisms, including antimicrobial protein profiles, determine whether bacterial colonization progresses to overt disease.[9]

The clinical parameters used to assess the efficacy of treatment are arguably the most important because they directly measure the health of the patient. When it comes to patient care, improving oral (and overall) health and the patient's quality of life are of paramount concern. Biological considerations are meaningful from a scientific and academic viewpoint, but they are of secondary importance. The clinical parameters typically measured—probing depth reduction, clinical attachment level gain, bleeding-on-probing reduction, and prevention of tooth loss—are both the most common and most clinically meaningful measures of the efficacy of treatment.[8,9] Benachinmardi KK et al evaluated the nature of oral microbiota in chronic periodontitis in this region of India and also the semiquantitative study in pre- and post-treatment group and to determine antibiotic susceptibility pattern for aerobic isolates. The study was conducted on 60 cases. Material was collected from the subgingival pockets in patients with chronic periodontitis attending the Periodontology, Outpatient Department. Clinical samples were transported to the laboratory in fluid thioglycollate medium. Initially Gram's stain and Fontana stains were done. Aerobic, anaerobic, and microaerophilic culture were put up. Antibiotic sensitivity test was done for aerobic isolates. Sixty samples yielded 121 isolates of which 78.34% were polymicrobial, 11.66% were monomicrobial and oral commensals were grown in 10% cases. Out of 121 isolates 91.74% were anaerobic, 7.43% were aerobic and 0.83% were microaerophilic. Fusobacterium species was the most common isolate among anaerobes. Using "paired t-test" "P" value was significant indicating significant reduction in colony count after phase-I periodontal therapy. This study showed that anaerobic bacteria are important cause of chronic periodontitis, along with aerobes and microaerophilic organisms. Fusobacterium spp, Bacteroides fragilis, Porphyromonas spp and Prevotella intermedia are the most common anaerobic pathogens. Bacterial culture methods are still economical and gold standard.[10]

Conclusion

The goal for both researchers and clinicians is to find the best treatment. From a biological perspective, the most successful treatments will likely need to attack the integrity of the periodontal biofilm and suppress the destructive host inflammatory response. However; further studies are recommended.

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