Rohi Rashid
MDS Student, Department of Periodontology, Institute of Dental Studies and Technologies, Modinagar, UP, India

**ABSTRACT:**
Periodontal diseases are initiated by bacterial species living in polymicrobial biofilms at or below the gingival margin and progress largely as a result of the inflammation initiated by specific subgingival species. In the past few decades, efforts to understand the microbiota of periodontal diseases have led to an exponential increase in information about biofilms associated with periodontal health and disease.

**Key words:** Diseases, Microbiology, Periodontal

**BACTEREMIA**
The incidence of bacteremia following dental procedures such as tooth extraction, endodontic treatment, periodontal surgery, and root scaling has been well documented. Bacteremia after dental extraction, third-molar surgery, dental scaling, endodontic treatment, and bilateral tonsillectomy has been studied by means of lysis-filtration of blood samples with subsequent aerobic and anaerobic incubation. Bacteremia was observed in 100% of the patients after dental extraction, in 70% after dental scaling, in 55% after third-molar surgery, in 20% after endodontic treatment, and in 55% after bilateral tonsillectomy. Anaerobes were isolated more frequently after endodontic treatment, and in 55% after third-molar surgery, in 20% after dental scaling, in 100% of the patients after dental extraction, in 70% after third-molar surgery, in 20% after endodontic treatment, and in 55% after bilateral tonsillectomy. Anaerobes were isolated more frequently than facultative anaerobic bacteria.

**Biofilm**
In natural settings, biofilms generally take the form of polymicrobial communities attached to biotic or abiotic surfaces. As a surface becomes colonized with individual cells, the bacteria form microcolonies which then secrete a sticky extracellular polymeric substance. The extracellular polymeric substance consists of polysaccharides, proteins, lipids, nucleic acids, and other polymers, and it helps the bacteria adhere to the surface, as well as to each other. Upon secretion of the extracellular polymeric substance, the biofilm matures by becoming larger and taking on a distinctive architecture. Usually, this structure includes separate regions of fast- and slow-growing cells, the presence of water channels which circulate metabolites, and the establishment of nutrient gradients.

**Gingivitis**
The simplest form of gingivitis is associated with the accumulation of supragingival plaque along the gingival margins of the teeth. This form of gingivitis has been extensively studied in human volunteers, and the sequence of events is well described. In these studies, individuals are brought to a state of health and then refrain from all forms of oral hygiene for a 3- to 4-week period. The initial colonizers of the teeth are streptococci,
which proliferate and in turn become colonized by other bacteria present in saliva, such as various Actinomyces species and Veillonella.  

Localized Juvenile Periodontitis (LJP)

LJP is different from all other periodontal infections, as it is not associated with plaque accumulations or calculus, is localized to certain anterior or front teeth and first molars, and is seen following puberty. Bacterial examinations of subgingival plaque from affected teeth and adjacent healthy teeth, revealed that the diseased teeth were colonized by an essentially Gram-negative flora dominated by organisms subsequently identified as various Capnocytophaga and Wolinella species and Actinobacillus actinomycetemcomitans.  

A microscopic examination cannot distinguish the species of bacteria present unless one uses an immunologic staining reagent specific for the organism in question. Such immunodiagnostic reagents have been used to detect and quantify the levels of P gingivalis, P intermedia, T denticola, and Aactinomycetemcomitans in the plaque. Cultural methods can, if the appropriate nonselective and selective media are used, provide information on the levels of Aactinomycetemcomitans, black pigmented species, Campylobacter species, and other periodontopathogens. Also, because viable organisms are available, antibiotic sensitivities of the isolated organisms can be determined, which may be useful in certain instances.  

Because of the various risk factors that contribute to periodontitis, it is possible that there will be no “magic bullet” treatment. It is also likely true that the underlying cause of periodontitis is different in different patients. For instance, one patient’s periodontitis may be due to a shift in the oral microflora due to poor hygiene, while another patient’s periodontitis may be due to an underlying genetic abnormality that leads to a destructive immune response. In light of this, periodontitis is perhaps better described not as a disease but as a symptom of an underlying condition.  

Conclusion

The dental literature is replete with scientific data supporting the concept that periodontal diseases are infectious diseases and that the infectious components are contained in the dental plaque. There is also convincing evidence that good oral care at home, combined with professional cleaning and/or the use of antimicrobial compounds can control dental plaque, and therefore reduce or eliminate dental disease.

References


Source of support: Nil
Conflict of interest: None declared
This work is licensed under CC BY: Creative Commons Attribution 3.0 License.