Various complexes of the oral microbial flora in periodontal disease

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Abstract

Periodontal disease is the commonest oral disease affecting about 30% of the human population [1]. The involvement of the periodontal pathogens is being more and more realized [2]. These pathogens constitute the “Oral microbiome”, a term that was coined to emphasize the ecological community consisting of the commensal, symbiotic, and pathogenic organisms that are present in the oral cavity and are capable of determining oral health and disease [3].

The oral biofilm primarily comprises of microbes and host proteins adhering to the teeth immediately after oral prophylaxis. The healthy gingival sulcus has flora predominantly consisting of Gram positive cocci, such as Streptococcus spp, and Actinomycetes sp in the equal concentrations [4]. A transition in the composition of the gingival sulcus from gram–positive, facultative type, μ–organisms with fermentation potential to predominantly gram–negative, anaerobic in nature, chemo–organotrophic, organisms and with proteolysis potential leads to the progression of the periodontal disease [2].

Subsequently, the plaque undergoes maturation that leads to a μ–flora having more of the anaerobic μ–organisms which are facultative, spirochetes and motile rods. With increase in the severity of the disease, the strict anaerobic, Gram–negative and motile organisms increase proportionately. The periodontal disease progresses from the slow, chronic, progressive destruction to short and acute sudden bursts that vary in intensity and duration [4].

Microbiologists have recently discovered an unexpectedly high level of coordinated multi–cellular behaviors which have made the perception that biofilms are like “cities” of μ–organisms. The bio–films are regulated by the signaling mechanism such as the “quorum sensing”, in which, the bacterial cells do communication with each other by releasing, sensing and responding to small diffusible signal molecules. As communication is the major factor for interpreting discrepancies, the biofilms forming bacteria adopt specialized roles and communicate with one another [5].

Research over a period of years on the sub–gingival μ–flora has helped in classifying μ–organisms into certain form of color complexes [2] that have variation from red to yellow as per their composition. Red complex is extremely
pathogenic and yellow is more of commensal. A number of micro-organisms that are supposed to be responsible for disease like Porphyromonas endodontalis, P. denticola, Filifactor alocis, Cryptobacterium curtum, Eubacterium saphenum, Mogibacterium timidum, P. corporis, P. disiens, Peptostreptococcus magnus, Slackia exigua, Trep. maltophilum, Trep. sp. Smibert-3, Trep. lecitihinolyticum, Trep. putidum sp. nov., Enterococcus faecalis, Escherichia coli and Bartonella sp [4].

Red and orange complexes have been classified as late colonizer in the development and maturation of subgingival plaque and they have been closely related to the pathological conditions of periodontal tissue [6].

Recently, the role of organisms such as D. pneumosintes, F. alocis, T. lecitihinolyticum, S. moorei, Cryptobacterium curtum, Peptostreptococcus micros and Fusobacterium nucleatum have been implicated with periodontal disease. In one of the reviews by Hajishengallis and Lamont, polymicrobial synergy and dysbiosis model of periodontal disease was suggested which suggested that some definite species, known as the “keystone pathogens,” modulate the response of the host which leads to the impairment of immunity and change the homeostatic balance in favour of dysbiosis [5,7].

Chen and colleagues [8] showed that 6 genera of μ-organisms: Porphyromonas, Tannerella, and Eubacterium in salivary sample of periodontitis patients have shown quite abundance compared to the salivary samples of the healthy controls.

The evidence till date is not conclusive of any single micro-organism being implicated in the etiology of the periodontal disease but several micro-organisms might play a role in the occurrence and progression of the periodontal disease. Many of the unrecognized periodontal pathogens are to be unmasked so that their possible role in the pathogenesis of the periodontal disease could be understood.

References


