Genetics and Periodontitis: A Strong Linkage

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Sir,

Mouth acts as window to a lot of systemic diseases and serves as a port of entry of the various infections that can alter and affect the immune status of the person. The oral cavity has the potential to harbor at least 600 different bacterial species, and in any given patient, more than 150 species may be present, surfaces of tooth can have as many as billion bacteria in its attached bacterial plaque and good oral hygiene is the fundamental for oral integrity as it greatly affects the quality of life.[1] Periodontitis is a destructive inflammatory disease of the supporting tissues of the teeth and is caused by specific microorganisms or group of specific microorganisms resulting in progressive destruction of periodontal ligament and alveolar bone with periodontal pocket formation, gingival recession or both. The host responds to the periodontal infections with an array of events involving both innate and adaptive immunity.[2] Periodontal diseases are recognized as infectious processes that require bacterial presence and a host response and are further affected and modified by other local, environmental, and genetic factors. The key organisms that cause periodontal disease were anaerobes including Aggregatibacter actinomycetecomitans, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythia, Fusobacterium nucleatum, Peptostreptococcus micros, and Campylobacter rectus.[3]

The link between periodontal disease and systemic diseases has been scientifically proven over last two decades.[4] Association of periodontal infection with organ systems like cardiovascular system, endocrine system, reproductive system, and respiratory system etc. makes periodontal infection a complex multiphase disease. Inflamed periodontal tissues produce significant amounts of pro-inflammatory cytokines, mainly interleukin 1 beta (IL-1ß), IL-6, PGE2, and tumor necrosis factor alpha (TNF-a), which may have systemic effects on the host. Periodontitis initiates systemic inflammation and can be monitored by inflammatory markers like C-reactive protein or fibrinogen levels.[5]

This relationship is illustrated in detail in Figure A.

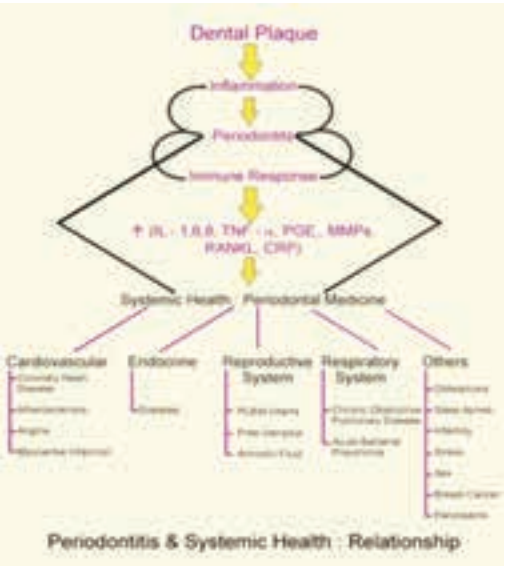

Fig A: Oral-Systemic Relation

Inflammatory-Genetic Linkage

The scientific literature during the last ten years has seen an exponential increase in the number of reports claiming links for genetic polymorphisms with a variety of medical diseases, particularly chronic immune and inflammatory conditions. Recently, periodontal research has contributed to this growth area. This new research has coincided with an increased understanding of the genome which, in turn, has permitted the functional interrelationships of gene products with each other and with environmental agents to be understood. As a result of this knowledge explosion, it is evident that there is a genetic basis for most diseases, including periodontitis. This realization has fostered the idea that if we can understand the genetic basis of diseases, genetic tests to assess disease risk and to develop etiology-based treatments will soon be reality. Consequently, there has been great interest in identifying allelic variants of genes that can be used to assess dis-

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ease risk for periodontal diseases.[6]

Investigations have utilized family studies of probands with chronic (adult) periodontitis or younger subjects with mild/ incipient periodontitis. The results suggested that there may be a genetic basis for the less severe forms of periodontitis.[7] The twin model is probably the most powerful method to study genetic aspects of periodontal diseases. The largest twin study included 4908 twin pairs of which, on the basis of questionnaire data, 349 (116 MZ and 233 DZ) pairs reported a history of periodontal disease in one or both pair members. The concordance rates ranged from 0.23 to 0.38 for MZ twins and 0.08 to 0.16 for DZ twins.[8] Michalowicz and co-workers evaluated the periodontal condition (attachment loss, pocket depth, gingival index and plaque index) of 110 adult twin pairs with a mean age of 40 years. The results indicated that between 38% and 82% of the population variance for these measures may be attributed to genetic factors.[9] In a subsequent study the authors estimated that chronic (adult) periodontitis had approximately 50% heritability, which was unaltered following adjustments for behavioural variables including smoking and utilization of dental care. [10] In epidemiological studies in the Dutch population, it has been suggested that periodontitis aggregates in families.[11, 12] Kornman et al reported that a "composite" IL-1 genotype consisting of at least one copy of the more rare allele at both an IL-la and IL-1ß loci was associated with severe periodontitis.[13] Karimbux et al (2012), in their meta-analysis, reported that IL1A and IL1B genetic variations are significant contributors to chronic periodontitis in Caucasians.[14] From both the twin studies and familial studies it can be concluded that the basis for familial aggregation of periodontitis appears to be genetic rather than bacterial, environmental or behavioral in nature. The heritable component for periodontitis was not associated with behaviors such as smoking, utilization of dental care and oral hygiene habits. This implies that genes controlling biologic mechanisms, and not behaviors, mediate the genetic influence on disease.

With the new available technologies and the fast growing body of related knowledge, the prospects are very promising. The genetic basis of periodontal disease is moving from experimental evidence to a more consistent translation effect on diagnosis and development of new strategies to modulate the host.

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