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RANTES comparison in patients with periodontal disease - A prospective clinical study

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1. Introduction

Research into the pathogenesis of disease has traditionally involved a reductionist approach in which discrete inflammatory pathways and processes are investigated to elucidate underlying mechanisms. With advances in genomic, epigenetic, proteomic, and metabolomic capabilities, an increased interest has emerged in a biologic systems approach to define the complex regulatory networks that result in health or disease [1] (see Figs. 2–6).

Periodontitis is a complex disease in which disease expression involves intricate interactions of the biofilm with the host immunoinflammatory response and subsequent alterations in bone and connective tissue homeostasis [2–4].

The basic conceptual model of periodontitis was revised in 1997 (Fig. 1) [5], in great part to acknowledge that various risk factors operated by modifying host responses led to changes in disease expression. In this model, host immunoinflammatory mechanisms are activated by bacterial product. In addition, cytokines and prostanoids, as well as matrix metalloproteinases activated through the host response, may stimulate damage to connective tissue and bone and shape the clinical presentation of disease [6].

Though specific microorganisms are cited as a cause for periodontitis, various other aspects of tissue alterations are also known to modify the periodontal status adversely. Based on this concept, presently serum, saliva, tissue biopsy specimens and gingival crevicular fluid have been investigated for their biochemical constituents [7,8].

Gingival crevicular fluid is generally considered as an initial transudate/interstitial fluid which later changes to exudates in the presence of inflammation [9]. It contains a vast array of biochemical factors, offering potential use as a diagnostic or prognostic biomarker of the biologic state of the periodontium in health and disease [10].

Chemokines selectively attract and activate different leukocyte subpopulations which in turn induce inflammation [11]. RANTES, a member of the CC chemokine family, displays a significant chemotactic activity for eosinophils, monocytes and CD + T cells [12].

RANTES expression has also been demonstrated in a variety of other diseases characterized by inflammation, including asthma, atherosclerosis, endometriosis and fibrosis [13].

Considering the multifunctional ability of monocytes/macrophages, these could be directly involved in initiation of development of the inflammatory response and alveolar bone loss observed in periodontitis.

Therefore, analysis of RANTES mechanism that induces monocyte recruitment into periodontal tissues represents an important step towards understanding the pathogenesis of this disease.

This study envisages to determine the presence of RANTES in GCF samples in healthy subjects, patients with gingivitis and periodontitis before and after initial periodontal therapy. The objectives of the study include assessment and comparisons of the levels of RANTES in GCF of healthy subjects, patients with gingivitis, chronic periodontitis and aggressive periodontitis, before and after 2 months following initial periodontal therapy, to correlate the levels of RANTES in GCF with various periodontal parameters, to determine whether RANTES can serve as a marker in the identification of active phase of periodontal disease.

2. Materials and methods

40 subjects who visited the Department of Periodontics, The Oxford Dental College, Hospital and Research center, Bangalore were included in the study. An informed consent was obtained

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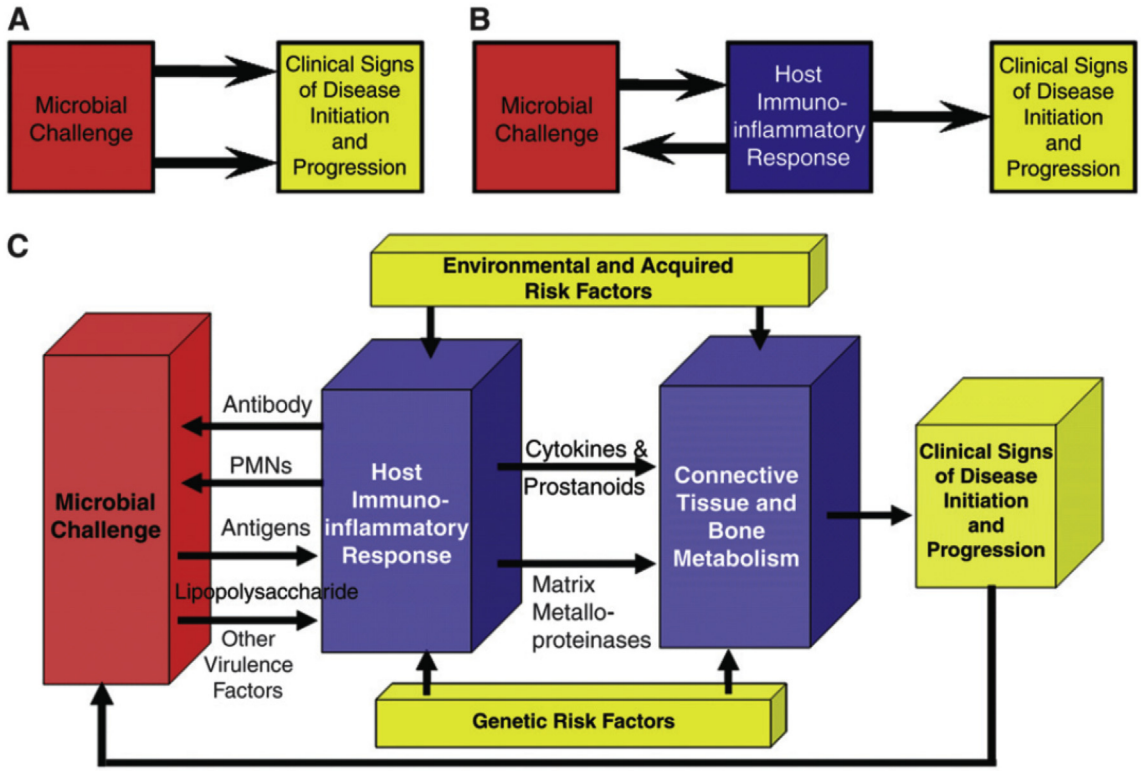

Fig. 1. The evolution of conceptual models of periodontal disease. A) An early linear model depicting the principal etiologic role for bacteria in the initiation and progression of periodontal disease. B) Circa 1980s model emphasizing a central role for the host immunoinflammatory response in the clinical development and progression of periodontal disease. C) A 1997 model demonstrating various factors contributing to the pathogenesis of human periodontitis based on pathways and processes known at the time.

GROUP-I

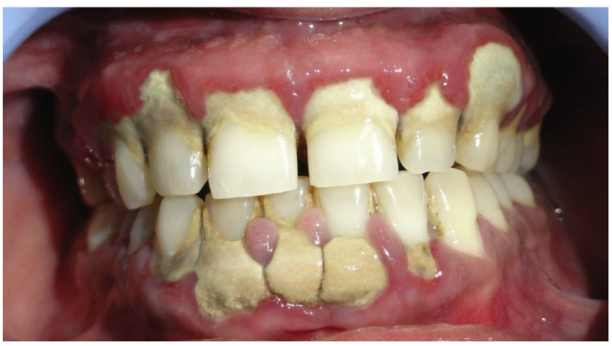

Fig. 2. Chronic periodontitis. Before NON surgical periodontal therapy.

GROUP-II

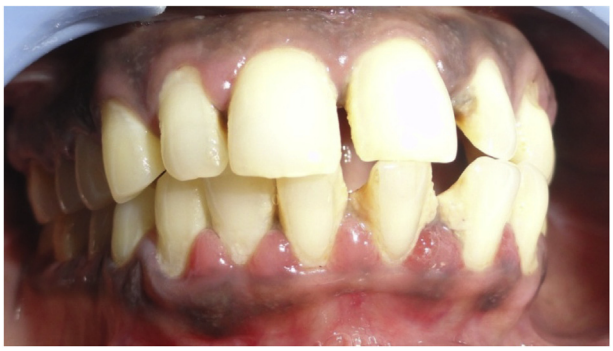

Fig. 3. AGGRESSIVE periodontitis. Before NON surgical periodontal therapy.

from all the subjects. The study period was of 10 months.

Inclusion Criteria: Subjects aged between 18 and 55years, Systemically healthy subjects, No antibiotic/NSAIDs usage in previous 3 month, No history of periodontal treatment in the last 6 months.

2.1. Exclusion criteria

Patients on medications (eg. Corticosteroids, anti inflammatory drugs, immune modulators), Patients with infectious conditions other than periodontitis, Medically compromised patients.

2.2. Study design

A total of 40 subjects were recruited and were divided into 4 groups.

Group 1 included 10 patients diagnosed with chronic periodontitis with probing pocket depth > 5 mm and clinical attachment loss > 3 mm with radiographic evidence of bone loss. In this group 5 patients having localized periodontitis and 5 with generalized periodontitis were included.(Photograph1).

Group 2 included ten patients with aggressive periodontitis. In this group 5 patients having localized aggressive periodontitis and 5 with generalized aggressive periodontitis were included.(Photograph2).

Group 3 included 10 patients with gingivitis. In this group 5 patients having localized gingivitis and 5 with generalized gingivitis were included.(Photograph 3).

Group 4 consisted of 10 systemically and clinically healthy

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