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PERIODONTITIS AND INTERLEUKIN- 18

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ABSTRACT
The occurrence and progression of the periodontal disease depend on periodontal microflora and the multifaceted response of the host, and these interactions are mediated by cytokines and chemokines. Interleukin-18 (IL-18) is a proinflammatory cytokine of the IL-1 superfamily. The aim of the present review was the assessment IL-18 level in periodontal disease.

KEYWORDS: microflora, cytokines and chemokines.

INTRODUCTION

Interleukins (ILs) are a group of cytokines that have complex immunological functions comprising proliferation, migration, growth and differentiation of cells.[1-3] They can be pro- or anti-inflammatory, and some interleukins also function as chemokines or chemoattractants for other cells.[5] Various interleukins are involved at different levels in the inflammatory pathway that ultimately leads to tissue destruction and the clinical presentation of Aggressive periodontitis (AgP). Due to these key roles that interleukins play in inflammation and periodontal disease pathogenesis, they make important potential therapeutic targets.[3,4]

Chronic periodontitis, an inflammatory disorder of the tooth supporting structures arises as a result of the complex interaction between periodontopathic bacteria and cells of host immune system. Resident and nonresident cells in the inflammation site are responsible for production of cytokines, which play a role in pathogenesis of periodontal disease.[1,3,5] The cellular immune response is characterized by infiltration of T cell into the periodontal tissues and differentiation into diverse subsets, which is influenced by the cytokine milieu.[5,6] The T cells subsets can be classified as helper T cells, cytotoxic T cells, and regulatory T cells. Among the T helper cell subsets, Th1 and Th2 cells have been most extensively researched upon. The Th1/Th2 balance is pivotal in immunoregulation of periodontal disease and is influenced by genetic factors, the characteristic of antigen (s), antigen presenting cell (APC), the immune response, and T cell receptor interactions.[1-4] It has been proposed that the stable lesion in periodontitis is mediated by Th1 cells, whereas progression of the lesion reflects a shift toward Th2 subset of cells.[4] The cytokines interleukin IL-12, IL-18, interferon gamma, and tumor necrosis factor alpha are involved in Th1 immune response and in contrast IL-4, IL-5, and IL-13 are involved in Th2 immune response and promoting humoral immunity.[5] The "protective Th1/destructive Th2" model is disputed by some studies.[6,7,8] A innovative subset of CD4 + T cells which clarifies many of the discrepancies in the classic Th1/Th2 model, has been identified and termed "Th17" based on the secretion of the cytokine IL-17. Cytokines characteristic of this subset have been found in inflamed periodontal tissue, suggesting their potential role in periodontal pathogenesis.[1-5] The presence of Th17 cells has been demonstrated in gingiva of patients with chronic periodontitis and there is an increasing evidence that Th17 cytokine plays a dominant role in progression of periodontal disease.[3]

Periodontitis is a complex, multifactorial disease that may be classified as two main types, chronic periodontitis (CP) and aggressive periodontitis (AgP), among other subclasses in the 1999 world workshop classification of periodontal diseases and conditions.[1-4]

A large number of studies have been conducted in different populations groups reporting genetic polymorphisms in various interleukins and their associations with both CP and AgP. The aim of this review was to summarize the findings of studies that reported associations or potential associations of

polymorphisms in the interleukin- 18 family of cytokines, with CP and AgP.

Interleukin- 18

Interleukin-18 a member of the IL-1 ligand superfamily is primarily produced by APCs,[13,14] and also by osteoblasts, adrenal cortex cells and oral epithelial cells.[7] It has been found to be up regulated in various chronic inflammatory diseases, including periodontal disease.[7-10] IL-18 could play a significant role in progression of periodontal disease because of its chemotactic, proinflammatory, and angiogenic properties and this cytokine also increases the rates of neutrophil activation.[2,4-7]

Periodontitis is a disease that may be classified as two main types, chronic periodontitis (CP) and aggressive periodontitis (AgP), among other subclasses in the 1999 world workshop classification of periodontal diseases and conditions. The destruction of periodontal tissues occurs by a complex interaction of the bacterial biofilm and host response and is also influenced by genetic, systemic and environmental factors. AgP, previously known as juvenile periodontitis (JP) or early onset periodontitis (EOP) occurs in a younger age group (≤35 years) and is associated with the rapid destruction of periodontal attachment and supporting bone. AgP can be either generalized (GAgP) or localized to the first molars and incisors localized (LAgP).[2] The terms AgP, LAgP and GAgP have been used throughout this manuscript to refer to this disease.[6,7]

Genetic polymorphisms are allelic variants that occur in at least 1% of the population. Single nucleotide polymorphisms (SNPs) are the most commonly found type of genetic polymorphisms. Such changes in genes could potentially alter the function of the proteins that they encode. In complex diseases like periodontitis, interaction of several different alleles may lead to an increased susceptibility to the disease. AgP is associated with familial aggregation, suggesting a strong genetic influence in this form of periodontitis.[3] Associations of several genetic polymorphisms in various genes with AgP have been previously reported, including the FcGammaR2, Vitamin D receptor and neutrophil formyl peptide receptor.[11]

Discussion and Clinical Implications of IL-18

Periodontitis is provocative and distract the connective tissue attachment and supporting bone present surrounding the teeth. It usually results from interactions between periodontal microflora and the complex response of the host.[1,3] Interleukins (ILs) are a group of cytokines that have complex immunological roles including proliferation, migration, growth and differentiation of cells.[4,6] Interleukin-18 is a pro-inflammatory cytokine and associate of the IL-1 family that has the ability to encourage either Th1 or Th2 cells in reaction to gram-negative infections.[8] It has only been scrutinized in a small number of studies to date, looking at different loci. Considerate the advancement of gingivitis to periodontitis has been a topic of strong search for several eras. An exploration for conceivable key cytokines tangled in this process might elucidate the mechanisms intricate in the start of the collagen breakdown, and consequently the loss of attachment.

Interleukin‑18 is recognised as a cytokine that rouses both Th1 and Th2 response reliant on the presence or absence of IL‑12. In the presence of IL‑12, IL‑18 mainly persuades the manufacture of interferon gamma producing Th1 cells and in absenteeism of IL‑12, it shifts the response to IL‑4 manufacturing Th2 cells.[9-11] Hence, IL‑18 appears to be a latent factor that can play a key role in amendable the immune responses intricate in the initiation and advancement of periodontal disease.[3-12-14]

Formerly Johnson & Serio revealed that the connotation between IL-18 and the pathogenesis of periodontal disease.[6] They confirmed that IL-18 concentration was higher in gingival biopsies adjacent to sites where the probing depth was >6 mm when compared to healthy sites.[12] In study conducted by Figueredo et al., it has been evidenced that IL-1b might play role as a factor between gingivitis in periodontitis patients and gingivitis in otherwise healthy patients.[15]

Pradeep et al., carried out the research with the exploration of the role of the Th1 cytokines with concomitant surge in Interleukin-17 and interleukin-18 in periodontitis as compared to the healthy individuals.[10] Figueredo et al stated that there is elevated concentration of interleukin-18 in gingival crevicular fluid of periodontitis patients.[15] Orozco et al., found IL-18 to be increased in shallow inflamed sites in periodontitis patients when compared with gingivitis sites in control patients.[16]

In the research conducted by Udagawa et al., and Horwood et al., demonstrated that Interleukin‑18 seems to have a pleiotropic activity, wherein IL‑18 acts as an inhibitor of osteoclast manufacture through indirect effects enabled by T cells production of granulocyte macrophage colony stimulating factor.[17] De Campos BO et al., evaluated the IL-18 level in gingival crevicular fluid following non-surgical periodontal therapy.[18] The study found that the IL-18 level is higher in periodontitis and decreases with concomitant non-surgical periodontal approach. Nair et al., concluded that, as the inflammation increased, there was a rise in the level of IL-18 and level of IL-18 decreases following periodontal therapy.[15] This may clarify the abridged levels of IL‑18 in gingival tissues from periodontitis patients in group III after periodontal therapy as perceived in present study. Our results are in accord with the results of these previous studies.[12-15]

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