International Journal of Scientific Trends- (IJST)
ISSN: 2980-4299
Volume 3, Issue 4, April, 2024
Website: https://scientifictrends.org/index.php/ijst
Open Access, Peer Reviewed, Scientific Journal

The Role of Cytokines in The Pathogenesis of Periodontal Disease

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Abstract

The pathogenesis of periodontitis is complex and involves a combination of innate and acquired immune mechanisms. The immune system responds to the presence of periodontopathic microorganisms by initiating an inflammatory response. This response involves the release of various cytokines and immune cells, which contribute to tissue damage.The inflammatory process in periodontal tissues progresses through different stages, starting with the infiltration of immune cells into the gingival tissues. The immune cells release pro-inflammatory cytokines, such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α), which further promote inflammation and tissue destruction.

Keywords: Incidence and discriminant models of chronic periodontitis in children.

Introduction

The continuous release of these cytokines disrupts the delicate balance of cytokines in the periodontal tissues. Inflammatory periodontal diseases, such as periodontitis and gingivitis, are prevalent worldwide and represent a significant problem in modern dentistry. Among periodontal diseases, inflammatory conditions like periodontitis and gingivitis are more common in the 40-50 age group, whereas mixed diseases, such as periodontosis with inflammation of the gingival margin, tend to prevail in older age groups. According to a report by the WHO Scientific Group based on a survey of 53 countries, the prevalence of periodontal disease in individuals over the age of 40 ranges from 65% to 98%. These statistics highlight the substantial need for effective treatment of periodontitis in the population.Current understanding of the etiology and pathogenesis of periodontitis is based on two main factors: bacterial colonization and the disruption of local and systemic immune mechanisms in the body. The latter includes the sensitization of the body's immune system to antigens produced by specific types of pathogenic microorganisms. However, it is important to note that the severity of the disease and the extent of bone tissue destruction do not always correlate directly with the quantity of pathogenic microflora present. Additionally, the progression of periodontitis is influenced by factors such as the patient's age and the presence of concurrent systemic diseases.

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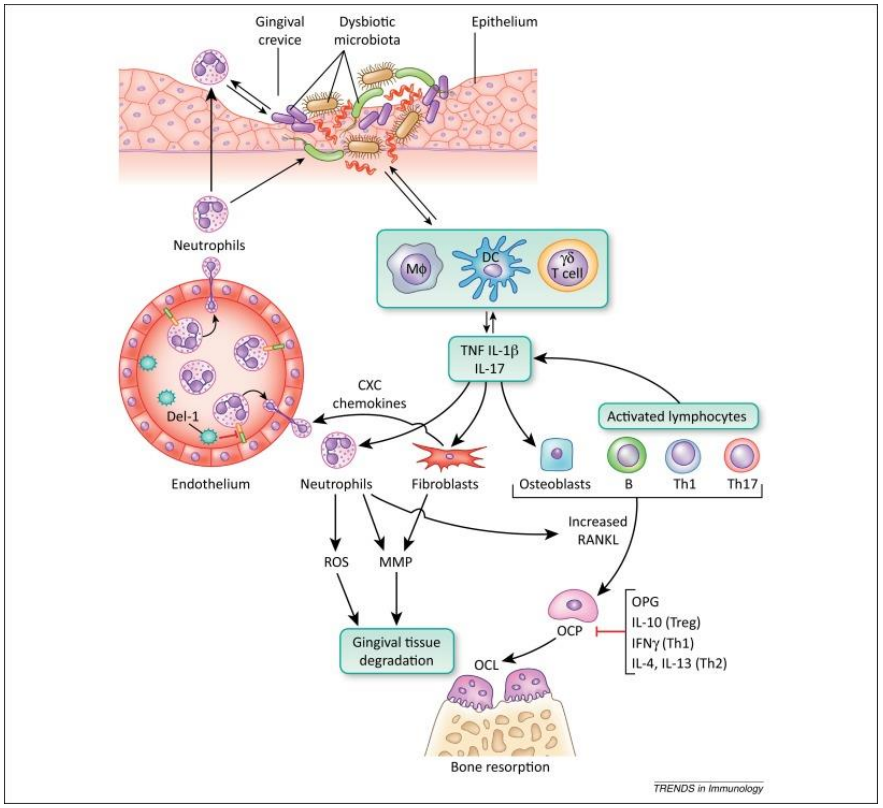
Materials and Methods

The 87 patients between the ages of 24 and 37 who had periodontal disease. The researchers measured various cytokines in both peripheral blood and gingival fluid samples. The cytokines analyzed included TNF-alpha, IFN-γ, IL-17, IL-18, IL-4, IL-10, sRANKL, and OPG. The measurements were performed using the solid-phase ELISA method with commercially available test systems. Prior to testing, the samples were stored under isothermal conditions. The specific commercial kits used for the analysis of IFN-γ, IFN-α, IL-4, IL-10, and TNF-α were from Protein-Best and Proteinovyi Kontur, utilizing solid-phase immunoenzymatic analysis. The results of the study showed that the pro-inflammatory cytokines TNF-α, IFN-γ, IL-18, and IL-17 had the highest levels in the gingival fluid of the patients. Among these cytokines, TNF-α had the highest level, measuring at 268±37.16 pkg/ml, which was more than seven times higher than in practically healthy individuals.



(Figure 1) The levels of other pro-inflammatory cytokines, including IL-18, IL-17, and IFN-γ, were 2-1.6 times higher in the gingival fluid of patients with chronic periodontitis compared to control subjects.

On the other hand, the anti-inflammatory cytokine IL-4 had a 2-1.6 times higher level in the gingival fluid of patients with chronic periodontitis compared to control subjects. The IL-4 content in the gingival fluid of patients with chronic periodontitis was 1.92±1.3 pkg/ml, which was more than 6.6 times lower than in virtually healthy individuals. Similarly, the IL-10 content in the gingival fluid of patients with chronic periodontitis was 1.36±0.92 pkg/ml, which was 2 times lower than its content in the gingival fluid of normal subjects. These findings suggest an imbalance

in cytokine levels in the gingival fluid of patients with chronic periodontitis, with elevated levels of pro-inflammatory cytokines and reduced levels of anti-inflammatory cytokines compared to healthy individuals.

Cytokine	Level in Gingival Fluid during Periodontitis
TNF-α	268±37.16
FN-γ	Elevated (2-1.6 times higher than in healthy individuals)
IL-18	Elevated (2-1.6 times higher than in healthy individuals)
IL-17	Elevated (2-1.6 times higher than in healthy individuals)
IL-4	Elevated (2-1.6 times higher than in healthy individuals)
IL-10	Reduced (2 times lower than in healthy individuals)

(Table 1) reflects the main changes in cytokine levels observed in the gingival fluid during periodontitis. The level of TNF-α is particularly notable, being significantly elevated compared to healthy individuals. The levels of other pro-inflammatory cytokines (IFN-γ, IL-18, IL-17) are also increased, while the levels of anti-inflammatory cytokines (IL-4, IL-10) are reduced compared to healthy individuals.

Results and Discussion

The levels of TNF-α and IL-17 in CP patients are 10 and 8.5 times higher, respectively, than in healthy individuals. The specific levels measured are 5.0±1.2 pg/ml for TNF-α and 17.1±2.3 pg/ml for IL-17. The levels of IFN-γ and IL-18 in the serum of CP patients were also increased compared to control subjects. The measured levels were 2.92±2.3 pg/ml for IFN-γ and 293±80.75 pg/ml for IL-18, which were 1.83 times higher than in control subjects. On the other hand, the levels of anti-inflammatory cytokines IL-4 and IL-10 in the serum of CP patients were lower compared to practically healthy subjects. The level of IL-4 was 1.00±0.85 pg/ml, which was 2.7 times lower, and the level of IL-10 was 4.14±1.09 pg/ml, which was 1.9 times lower than in healthy individuals. In summary, the serum of CP patients exhibited an increase in pro-inflammatory cytokines in the following order: TNF-α > IL-17 > IL-18 > IFN-γ, and a decrease in anti-inflammatory cytokines relative to the control group: IL-4 < IL-10. Additionally, there was a correlation between the levels of TNF-α and IL-4 in the serum of CP patients. An increase in TNF-α was accompanied by a decrease in IL-4, with a coefficient of 5.0. In practically healthy individuals, the reverse relationship was observed, with IL-4 levels (2.7±1.7 pg/ml) being higher than TNF-α levels (0.5±0.4 pg/ml) with a coefficient of 5.4. Some correlation was also observed between the increase in IL-18 (2.93±80.75 pg/ml) and the decrease in IL-10 (4.14±1.09 pg/ml) with coefficients of 1.8 and 1.4, respectively, compared to the control levels (160.51±41.6 pg/ml for IL-18 and 5.12±2.7 pg/ml for IL-10).

It is worth noting that 92% of patients with chronic periodontitis (CGP) exhibited increased blood levels of sRANKL, a cytokine that inhibits osteoclastogenesis. However, the increase was only 1.5 times higher than the normal levels, and it corresponded to a parallel increase in the level of the cytokine OPG, which promotes osteoclastogenesis. This increase in OPG in the blood of CGP patients was found in only 68% of cases. On the other hand, approximately the same percentage of CGP patients (75% and 72%) had increased levels of both RANKL and OPG in the gingival fluid of the periodontal pockets. However, the coefficient of increase in sRANKL content relative to physiological levels was 4.8 times in these patients, whereas the coefficient of increase in OPG was only 2.27 times. Therefore, the degree of damage to the periodontal tissues appears to depend on the balance between the strength of damaging factors and the level of protective-adaptive mechanisms, such as the immune system of oral tissues, which is connected to general immunity but also possesses considerable autonomy and self-regulation. The development of periodontitis is accompanied by an imbalance in the cytokine system, with an increase in pro-inflammatory cytokines. Osteoclastogenic cytokines are also increased in both plasma and gingival fluid. However, the sRANKL/OPG ratio does not reliably show an increase.

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