IMMUNOLOGICAL INDICATORS AND THEIR ROLE IN THE PATHOGENESIS OF PERIODONTAL LESIONS IN COMBINATION WITH CHRONIC TONSILLITIS

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Abstract: The interrelationship between periodontal diseases and chronic tonsillitis represents a significant clinical and scientific concern due to shared immunopathological mechanisms. This study explores immunological indicators and their contribution to the pathogenesis of periodontal lesions when associated with chronic tonsillitis. Special attention is given to systemic and local immune responses, including cytokine imbalance, immunoglobulin levels, and cellular immunity dysfunction. The findings highlight how chronic infection in the tonsils may exacerbate periodontal tissue destruction through immune dysregulation. Understanding these mechanisms provides a basis for improved diagnostic approaches and integrated treatment strategies, ultimately contributing to better management of patients with combined inflammatory conditions.

Keywords: periodontitis, chronic tonsillitis, immunological indicators, cytokines, immune response, inflammation, oral health, pathogenesis, immunoglobulins, systemic infection

The study of periodontal diseases has evolved significantly in recent decades, with increasing attention being paid not only to local microbial factors but also to systemic influences, particularly immunological mechanisms. Among the conditions that exhibit a strong association with periodontal pathology, chronic tonsillitis occupies a unique position due to its persistent infectious nature and its impact on systemic immunity. The coexistence of periodontal lesions and chronic tonsillitis creates a complex pathological environment in which immunological indicators play a central role in disease progression and severity.

Periodontal diseases, particularly periodontitis, are characterized by inflammation of the supporting structures of the teeth, including the gingiva, periodontal ligament, and alveolar bone. While bacterial biofilm is the primary etiological factor, the host immune response determines the extent of tissue destruction. In patients with chronic tonsillitis, the immune system is continuously exposed to

antigenic stimulation from the tonsillar infection, which can lead to systemic immune alterations. These alterations significantly influence the pathogenesis of periodontal lesions, often resulting in more aggressive and persistent forms of the disease.

One of the key immunological aspects involved in this interaction is the imbalance of cytokines. Cytokines are signaling molecules that regulate immune and inflammatory responses. In both chronic tonsillitis and periodontitis, there is an overproduction of pro-inflammatory cytokines such as interleukin-1 (IL-1), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). These cytokines contribute to the destruction of periodontal tissues by stimulating osteoclast activity and promoting bone resorption. At the same time, the production of anti-inflammatory cytokines is often insufficient to counterbalance this effect, leading to a chronic inflammatory state.

Another important immunological indicator is the level and activity of immunoglobulins. Immunoglobulins, particularly IgA, IgG, and IgM, play a crucial role in the defense against microbial pathogens. In patients with chronic tonsillitis, alterations in immunoglobulin levels are frequently observed. For example, decreased levels of secretory IgA in saliva may impair the local immune defense in the oral cavity, facilitating the colonization and proliferation of pathogenic bacteria in periodontal pockets. Conversely, elevated levels of IgG and IgM in the bloodstream may reflect a systemic immune response to chronic infection, which can contribute to tissue damage through immune complex formation and complement activation.

Cellular immunity also plays a significant role in the pathogenesis of periodontal lesions associated with chronic tonsillitis. T-lymphocytes, particularly helper T cells (CD4+) and cytotoxic T cells (CD8+), are essential for orchestrating the immune response. In chronic tonsillitis, there is often a dysregulation of T-cell function, characterized by an imbalance between different T-cell subsets. This imbalance can lead to an exaggerated inflammatory response in periodontal tissues, as well as impaired regulation of immune processes. Additionally, the activity of macrophages and neutrophils, which are key players in innate immunity, may be altered, resulting in increased production of reactive oxygen species and proteolytic enzymes that contribute to tissue destruction.

The role of neutrophils in this context deserves particular attention. Neutrophils are the first line of defense against bacterial infection and are abundant in periodontal tissues. However, in the presence of chronic systemic inflammation, such as that caused by tonsillitis, neutrophil function may become dysregulated. This can manifest as either hyperactivity, leading to excessive tissue damage, or impaired function, resulting in

inadequate bacterial clearance. Both scenarios can exacerbate periodontal disease and contribute to its progression.

Furthermore, the presence of chronic tonsillitis can influence the systemic inflammatory status of the patient, which in turn affects periodontal health. Elevated levels of acute-phase proteins, such as C-reactive protein (CRP), are commonly observed in patients with chronic infections. These proteins can enhance inflammatory responses and contribute to the destruction of periodontal tissues. The systemic spread of inflammatory mediators from the tonsils to the periodontal tissues may create a feedback loop that perpetuates and intensifies the disease process.

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