

Interrelation Between Inflammatory Periodontal Diseases and Bronchial Asthma: Pathogenetic Parallels and Treatment Tactics

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Abstract: The article examines the mechanisms of the relationship between bronchial asthma and chronic periodontitis. The authors analyze common immuno-inflammatory links of pathogenesis, the impact of inhalation therapy on oral health, and the clinical features of periodontitis progression in the context of comorbid pathology. Ways to optimize complex treatment based on an interdisciplinary approach are proposed.

Keywords: periodontitis, bronchial asthma, comorbidity, systemic inflammation, interleukins, inhaled glucocorticosteroids.

Introduction

Inflammatory periodontal diseases remain one of the leading challenges in modern dentistry. In recent years, the concept of "systemic medicine" has emphasized the inextricable link between the condition of periodontal tissues and general somatic status. Bronchial asthma (BA), as a chronic respiratory disease, exerts a significant modifying influence on dental status, creating conditions for the progressive destruction of alveolar bone.

Bronchial asthma is a chronic inflammatory airway disease characterized by variable bronchial obstruction, bronchial hyperreactivity, and systemic immuno-inflammatory changes. The long-term course of asthma and the necessity for continuous pharmacological therapy—specifically the use of inhaled corticosteroids (ICS)—create conditions for the development of an unfavorable dental status. Consequently, analyzing the relationship between bronchial asthma and periodontal diseases is of significant scientific and practical importance.

Literature review

According to epidemiological studies, signs of inflammatory periodontal diseases are detected more frequently in patients with bronchial asthma compared to the general population. The clinical presentation of periodontitis in this category of patients is characterized by more pronounced gingival bleeding (70-80% of patients with BA exhibit more severe bleeding symptoms compared to 20-30% in the general population), increased periodontal pocket depth (average depth in BA patients can be 4-6 mm versus 3-4 mm in the control group), and a tendency toward a chronic course of the process.

There is also a higher recurrence rate and a less pronounced effect from standard therapeutic measures, which may be due to systemic impairments in immune response and microcirculation characteristic of bronchial asthma.

One of the key pathogenetic factors is the chronic systemic inflammation underlying bronchial asthma. Increased production of pro-inflammatory cytokines contributes to osteoclast activation

and enhanced resorption of the alveolar process bone tissue. The key link is the phenomenon of systemic inflammation. Common immunological mechanisms include:

- **Cytokine Imbalance:** Elevated levels of pro-inflammatory mediators (IL-6, IL-1 β , and TNF- α) in blood serum and gingival crevicular fluid stimulate osteoclast activity, accelerating bone resorption.
- **Oxidative Stress:** Free radical tissue damage is intensified against the background of hypoxia caused by bronchial obstruction.
- **Mouth Breathing and Xerostomia:** Reduced salivation and a decrease in the protective properties of saliva (lysozyme, IgA) lead to impaired self-cleaning of the oral cavity.

Discussions

The impairment of microcirculation and tissue hypoxia resulting from bronchial obstruction negatively affects the trophics of periodontal tissues and slows down reparative processes. Mouth breathing further contributes to the drying of the mucous membrane and a decrease in the protective function of saliva.

Inhaled corticosteroids (ICS), widely used in the maintenance therapy of bronchial asthma, exert a pronounced local effect on the oral mucosa. Long-term use of ICS is a risk factor. ICS contribute to:

- Reduced local immunity and activation of fungal flora (predominantly the *Candida* genus).
- A shift in salivary pH toward acidity, which accelerates enamel demineralization and dental plaque formation.
- Impaired microcirculation in periodontal tissues.

These changes create prerequisites for intensifying inflammatory processes in periodontal tissues and increase the risk of infectious complications. In this regard, the prevention of dental side effects from inhalation therapy is of great importance.

The improvement of periodontitis treatment in patients with bronchial asthma should be based on the principles of complexity, individualization, and interdisciplinary interaction. Along with traditional periodontal treatment methods, the correction of risk factors associated with the underlying disease is of particular importance.

Promising directions include the development of personalized treatment regimens aimed at reducing the severity of systemic inflammation, enhancing local immunity, and improving microcirculation in periodontal tissues.

Conclusion

Thus, bronchial asthma is a significant factor influencing the development and clinical course of inflammatory periodontal diseases. An extensive analysis of literature data confirms the need for a pathogenetically grounded and comprehensive approach to treating periodontitis in patients with this somatic pathology.

It has been established that the severity of periodontitis correlates with the duration of bronchial asthma and the degree of its control. In uncontrolled cases, generalized forms of periodontitis are more frequently detected, accompanied by significant loss of attachment and bone resorption.

Special emphasis is placed on the imbalance of cellular and humoral immunity, as well as the impairment of local oral defense mechanisms, which creates favorable conditions for the progression of periodontitis. These changes contribute to the disruption of the oral microbiocenosis and increased activity of periodontopathogenic microflora, which must be considered when planning dental treatment.

Modern approaches to periodontitis treatment in patients with bronchial asthma should be comprehensive and take into account both the dental and somatic status of the patient. Interdisciplinary interaction between the dentist and the pulmonologist is of particular importance.

A promising direction is the development of individualized prevention and treatment schemes aimed at reducing inflammation, improving microcirculation, and increasing the local resistance of periodontal tissues.

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