

# Periodontal Condition in Patients with Bronchial Asthma: Influence of the Disease and Its Pharmacotherapy

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**Abstract.** Bronchial asthma is a chronic inflammatory respiratory tract disease which affects more than 300 million people worldwide and is expected to increase substantially. Its treatment mainly includes inhaled bronchodilators, corticosteroids and anticholinergic agents that improve patency of airways. Prolonged use of these medications has been linked to negative oral health effects such as decreased salivary secretion, enamel demineralization and susceptibility to periodontal inflammation and candidiasis. However, the mechanisms and degree of periodontal impairment in asthmatic patients under long term pharmacotherapy are insufficiently explored. In spite of the association of asthma drugs with oral changes, few studies have comprehensively assessed the multifactorial effect of asthma drugs on periodontal tissues and salivary function, especially in the populations of Central Asia. The purpose of this study was to analyze the impact of bronchial asthma and its pharmacological treatment on periodontal health taking into consideration the connection between decreased salivation, changes of immunity and periodontal inflammation. Studies have shown that beta-2-agonists and corticosteroids have significant effects in reducing the secretion and protective function of saliva, which results in the accumulation of plaques, enamel demineralization and inflammatory periodontal disease. Inhaled corticosteroids have also been associated with decreased bone mineral density and increased IgE-mediated inflammatory responses. The study shows the regional aspect from Uzbekistan and CIS countries, with a focus on the clinical evidence of periodontal changes in asthmatics and offers recommendations for special preventive measures. Understanding the interaction between asthma treatment and oral health can be used to guide individual preventive and therapeutic approaches to ensure periodontal integrity of patients on long-term inhalation therapy.

**Keywords:** Bronchial asthma, Beta-2-agents, Dental caries, Dental erosion, Inhaled corticosteroids, Candidiasis, Periodontal diseases, Salivary secretion.

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## 1 Introduction

Bronchial asthma qualifies as one of the most common chronic respiratory illnesses, and one which has a major role to play as far as the quality of life of the consonant is concerned [10,13]. According to the World Health Organisation, the number of asthma patients exceeds 300 million people, with a projected increase of another 100 million by 2025 (Global Initiative for Asthma, 2020). The pathophysiological basis of the disease is persistent inflammation of the bronchial mucosa, accompanied by increased sensitivity of the airways to various stimuli. The revelation comprises the presence of wheezing, episodes of coughing, a tight sensation in the chest and dyspnoea [2,8]. Asthma is characterised by intermittent bronchial obstruction, the severity

of which varies both overnight and over a long period of time, remaining potentially reversible both spontaneously and under the influence of medication.

The main goals of bronchial asthma treatment remain the suppression of inflammatory processes in the airways and restoration of their patency. As a consequence, treatment is traditionally divided in two directions: management of exacerbations and maintenance of long-term control of the disease. The drugs of the first group (life saving medications) are short-acting bronchodilators ( $\beta_2$  agonists), systemic corticosteroids and anticholinergic agents. Control o/t LT is maintained using ICS, LABA and LTRAs[1].

Most medications used for asthma are delivered by inhalation, via aerosol inhalers, powder inhalers or nebulisers. To achieve optimal therapeutic effect, it is essential to teach patients the correct inhalation technique and monitor compliance. In some cases, the frequency of inhalation can be as high as 4 times a day and the therapy can last for many years, making the effects of the drugs on the oral tissues long-lasting and significant[3,4,7].

Because of the gradual rise in the occurrence of bronchial asthma, the dental fraternity is continually facing the aftermath of chronic inhalation medication among the management of such patients. Several researchers have reported that salivary secretion rate reduces with the use of beta-2-agonists and glucocorticosteroids resulting to the impaired salivary defence. In patients with bronchial asthma receiving regular drug therapy, salivary secretion was reduced by 26% for total saliva and 36% for parotid saliva compared to healthy individuals. In addition to a decrease in volume, a decrease in protein, amylase, hexosamine, lysozyme and secretory IgA concentrations in stimulated saliva was noted [9]. Such a change in the qualitative and quantitative composition of saliva disrupts the processes of remineralisation of hard dental tissues, promotes colonisation of cariogenic and pathogenic microorganisms, creating conditions for the development of multiple caries, erosive lesions of enamel, candidiasis and inflammatory periodontal diseases.

These processes are especially relevant in patients with chronic inflammatory diseases of the gingival mucosa, in particular chronic generalised catarrhal gingivitis. Reduced salivation in combination with constant medication load in bronchial asthma increases local inflammatory reactions, impairs the barrier function of the mucosa, increasing its susceptibility to microorganisms and external irritants. As a result, such patients often have more pronounced swelling, hyperaemia of gums, tendency to bleeding, which requires a special approach to both treatment and prevention of inflammatory periodontal diseases [5,11]

Since the issue of bronchial asthma has a very pronounced influence on the state of periodontal tissues, and the features of its treatment on the background of chronic catarrhal gingivitis require careful consideration, there is the necessity to analyze the existing literature sources in detail and derive the best possible options on the prevention and treatment of inflammatory pair of planning the disease in patients belonging to the given category [2].

## **2 Salivated and Unsalivated Oral Health in Bronchial Asthmatics Patients**

Saliva fulfils a number of vital functions, providing maintenance of oral homeostasis, protection of hard and soft tissues, as well as participating in the prevention of inflammatory processes. In norm its composition is balanced, which contributes to the preservation of biochemical balance and prevents the aggressive effects of external factors on the mucous membrane and teeth [11].

One of the key functions of saliva is to moisturise and protect the oral mucosa, which is achieved by mucins, glycoproteins and water. These components form a thin seromucinous layer that prevents microbial adhesion and provides a barrier effect, preventing mechanical damage and irritation of the oral epithelium. In chronic generalised catarrhal gingivitis, especially in patients with bronchial asthma, adhesion of pathogenic bacteria against the background of reduced salivary secretion can increase inflammatory changes, exacerbating swelling and hyperaemia of the gums.

The ability of saliva to cleanse and flush is also playing a crucial role. Its water removes food debris, necrotic epithelium, non-viable cells, and microorganisms, which leads to a decrease in the bacterial load on periodontal tissues. Lack of saliva in patients with bronchial asthma, taking inhaled beta-2-agonists, leads to a decrease in bathing, plaque, and products of their vital activity of the drug, which creates conditions for chronic inflammation in the gingival margin [4].

The buffering function of saliva ensures the maintenance of acid-alkaline balance, which is extremely important for the prevention of enamel demineralisation and regulation of remineralisation processes. The main components responsible for this are bicarbonates, phosphates, calcium, statin and fluoride. These elements maintain the mineral structure of teeth, contribute to the repair of enamel micro-damage and neutralise acids formed by plaque bacteria. In patients with asthma, a decrease in saliva secretion, especially with prolonged use of inhaled corticosteroids, leads to impaired buffering properties, creating conditions for accelerated development of dental caries and inflammatory periodontal diseases such as catarrhal gingivitis.

The antimicrobial activity of saliva is due to the content of secretory immunoglobulin A, lysozyme, lactoferrin, lactooxidase, mucins, cysteine and histatins. These biologically active substances prevent adhesion of microorganisms to mucosal and dental surfaces and inhibit the growth of pathogenic bacteria and fungi. Nevertheless, in the patients with bronchial asthma diseases, with the active use of inhaled corticosteroids regularly, the concentration of secretory IgA, and other protective factors of saliva diminishes and is contributed to the formation of the candidiasis and inflammatory processes in the periodontal tissues[6,8].

Decrease in the above mentioned functions of saliva in patients taking bronchodilators and glucocorticosteroids for a long time creates special conditions for the development of chronic generalised catarrhal gingivitis. Gingival inflammation in this group of patients often occurs against the background of xerostomia, increased salivary viscosity, impaired remineralisation of enamel and pathogenic microflora activation, which requires an individual approach to the prevention and treatment of inflammatory periodontal diseases.

### **3 Factors Contributing to Oral Health Deterioration in Patients with Bronchial Asthma and Preventive Measures**

Dental disease is more prevalent among patients with bronchial asthma because there are several factors contributing to such a condition. As an illustration, the reduction in the salivary flow as a result of beta 2-agonists serves as part of the decline of the natural cleaning action of the tooth cavity, carbohydrate build-up, reduced buffering strength as well as subsequent exposure to dental caries. Moreover, not all antiasthmatic medications are free of fermentable carbohydrates that also induces the growth of *Streptococcus mutans* and *Lactobacillus* spp. that is known in the pathogenesis of dental caries.

In chronic catarrhal gingivitis, these changes increase the inflammatory process in periodontal tissues, making the mucosa more vulnerable to infection. In addition, patients with asthma often suffer from gastroesophageal reflux, which further increases the risk of erosive lesions of the teeth and may irritate the gingival margin, aggravating the course of gingivitis [13].

Decrease in the concentration of secretory IgA and changes in the immune response against the background of constant use of corticosteroids weaken the barrier function of the mucosa, which contributes to its dehydration, especially in patients who breathe mainly by mouth. This creates favourable conditions for the development of candidiasis and the formation of chronic gingival inflammation.

To prevent these complications, patients with asthma are recommended to:

- regular professional oral hygiene;
- use of fluoride-containing pastes and rinses;
- use of antiseptic mouthwash solutions (e.g. chlorhexidine) after using an inhaler;
- learning proper inhalation techniques and using spacers to reduce contact of the aerosol with the oral mucosa;
- taking sufficient fluids to reduce dry mouth;
- prescribing sugar substitutes and chewing gums to stimulate salivation;
- use of saliva substitutes in case of severe xerostomia.

An individual approach to the management of patients with bronchial asthma suffering from chronic catarrhal gingivitis should include not only therapy of inflammatory periodontal diseases, but also a set of measures aimed at normalising saliva secretion, restoring its protective properties and reducing the effect of drug load on the oral cavity.

### **4 Asthma and Dental Erosion**

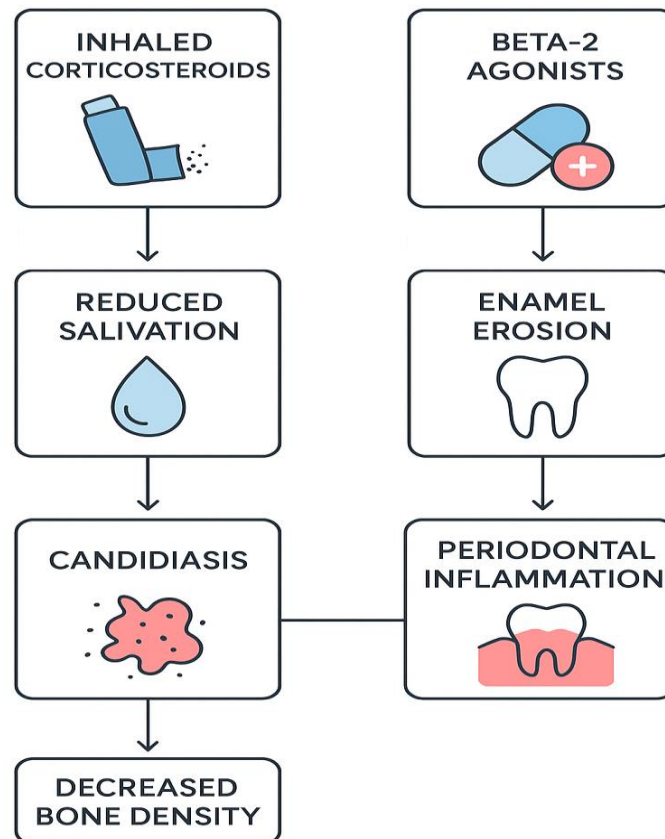
A number of clinical studies indicate an increased risk of dental erosion in patients with bronchial asthma, particularly among children. Children with bronchial asthma were significantly more likely to have acid erosion of dental hard tissues compared to healthy peers. Significant thinning of enamel on the vestibular and occlusal surfaces of molars and premolars, as well as increased tooth sensitivity were observed [9].

Contradictory results were obtained, and no statistically significant association was identified between the presence of asthma and denture use, which underscores the multifactorial and complex nature of this process [10].

The primary pathogenesis cause of the enamel damage in patients having bronchial asthma is linked with the diminishment of the protective ability of saliva. It is known that saliva acts as a natural buffer and helps to neutralise acids coming from outside or secreted during gastroesophageal reflux. However, the administration of beta-2-agonists, which are widely used to control bronchospasm, reduces salivary secretion and impairs its buffering properties. In addition, patients with bronchial asthma often have oral breathing, which also causes xerostomia, increasing the risk of erosion and caries.

An additional risk factor is the acidity of the inhaled medications themselves. Powdered forms of bronchodilators have a pH below the critical level of 5.5 at which enamel demineralisation begins. Such preparations, especially with repeated daily use, may contribute to the gradual dissolution of the mineral structures of teeth. A decrease in saliva and plaque pH was found after the use of a lactose-based powder inhaler, confirming its potential role in enamel demineralisation[8].

## ASTHMA THERAPY EFFECTS ON ORAL HEALTH



**Fig. 1.** Multifactorial Effects of Asthma Therapy on Oral Health

This multifactorial schematic diagram shows the various ways in which asthma pharmacotherapy can impact on oral health. Inhaled corticosteroids and beta-2 agonists have been demonstrated to diminish salivary flow, induce enamel erosion, and disrupt the microbial equilibrium, thereby facilitating candidiasis and periodontal inflammation. Long-term exposure further reduced bone density, highlighting the systemic and local oral effects of long-term asthma treatment (Figure 1).

The high prevalence of gastroesophageal reflux (GER) among individuals with bronchial asthma has attracted particular attention, as approximately 75% of patients with asthma exhibit GER symptoms, whereas its prevalence in the general population is substantially lower. Increase in intra-abdominal pressure, relaxation of the lower oesophageal sphincter against the background of beta-2-agonists, neurogenic mechanisms and microreflux during asthmatic attacks create prerequisites for the ingestion of gastric contents into the oral cavity. Acid exposure of teeth in chronic reflux can cause severe erosions, which is confirmed by numerous studies.

Therefore, dental erosions in bronchial asthma patients have a complex mechanism of occurrence, with reduced levels of saliva secretion, direct contact of acids with inhaler particles in powder form, and gastroesophageal reflux, enhancing acid aggression in the mouth cavity.

Prevention includes educating patients on rinsing the oral cavity with decontaminating solutions after inhaler use. Rinsing with milk, sodium bicarbonate solution, or antacids has been shown to be effective in restoring the acid-base balance in the oral cavity. Also, it is suggested that spacers should be used to minimize

contact of the drug with the teeth and mucosa of the oral cavity. Since patients with bronchial asthma are vulnerable to gastroesophageal reflux, dentists should refer the illness to a gastroenterologist to monitor and institute commitment to the measurement of oesophageal pH daily accompanied by antireflux therapy in case of acid erosion of the teeth.

Early diagnosis of erosions in patients with bronchial asthma, a comprehensive approach to prevention, including normalisation of salivary secretion, neutralisation of the acid effect of inhaled drugs and control of symptoms of gastroesophageal reflux, can reduce the risk of destruction of hard tissues of teeth and preserve the dental health of this group of patients.

#### Asthma & inflammatory (periodontal) disease

Bronchial asthma and periodontal diseases exhibit a complex pathogenetic relationship, which has been demonstrated in several clinical studies. However, the findings reported in the literature remain inconsistent. Some studies have indicated that patients with bronchial asthma present with more pronounced signs of inflammatory periodontal disease compared with healthy individuals. In contrast, other investigations have not identified significant differences in the prevalence of periodontitis between asthmatic patients and control groups[4].

The principal pathogenetic mechanism underlying periodontal deterioration in patients with bronchial asthma is associated both with immune system dysfunction and with the adverse effects of medications used to manage bronchial obstruction. Inflammatory changes in the gingival tissues are attributed not only to immunological imbalance but also to mucosal dehydration resulting from mouth breathing[2]. It is known that constant mouth breathing disturbs periodontal tissues hydration, reduces the level of saliva secretion and suppresses its protective properties, which creates conditions for increased bacterial contamination and chronicity of inflammatory processes.

Another important thing is that people with bronchial asthma have higher levels of immunoglobulin E (IgE) in their periodontal tissues [12]. IgE-mediated activation of mast cells in response to bacterial flora antigens leads to the release of histamine, prostaglandins and other inflammatory mediators, which contributes to the destruction of periodontal structures. These data indicate that allergic hyperactivity of asthmatics can contribute to the aggressive progression of periodontitis.

It has been shown that children with asthma exhibit a higher rate of calculus formation compared with children without respiratory pathology[6]. The elevated deposition of tartar is related to the alterations in the saliva composition: in asthmatics, the level of calcium and phosphate concentration in the parotid and submandibular gland secretions has been observed to increase. This preconditions mineralisation of soft dental plaque and development of hard deposits which is a permanent source of bacterial toxins inducing periodontal inflammation.

Inhaled corticosteroids (ICS) used as a basic therapy for bronchial asthma may pose a risk to periodontal health. Prolonged administration of ICS at moderate to high doses can lead to a systemic decrease in bone mineral density due to suppression of osteoblast activity and stimulation of osteoclastic resorption. A reduction in mandibular bone mass is associated with an increased risk of tooth loss in long-term ICS users, supporting the notion that corticosteroid-induced osteopenic processes may exacerbate alveolar bone destruction in inflammatory periodontal diseases [9].

An additional risk factor is oropharyngeal candidiasis, which may develop in patients who regularly use inhaled glucocorticosteroids. The incidence of candidiasis can be particularly high among patients using high-dose ICS. Inhaled corticosteroids have been shown to reduce the level of secretory IgA in saliva, weakening local immune defense mechanisms of the mucous membranes. Increased glucose content in saliva, along with the presence of lactose in powder inhalers, creates favorable conditions for the adhesion and proliferation of *Candida* species, which can exacerbate inflammatory periodontal diseases and contribute to the development of candidal gingivitis.

For the prevention of candidiasis and destructive periodontal changes in patients with bronchial asthma it is recommended:

- use of spacers to reduce drug deposition on the mucous membrane;
- mandatory rinsing of the oral cavity after inhalation with warm water, milk or alkaline solutions;
- regular follow-up with a dentist to assess the condition of the periodontium;
- prescription of topical antimycotics (nystatin) at the first signs of gingivitis.

#### Current research in the CIS and Uzbekistan

The fact that the CIS countries, including Uzbekistan, suffer much due to their high rates of respiratory disease among the population and the rising number of patients currently taking long-term inhaled therapy using glucocorticosteroids and hematologists warrants the relevance of examining the relationship between bronchial asthma and inflammatory periodontal diseases. Researchers are concerned with investigating the impact of asthma medications on the periodontal state, microbial composition of the mouth cavity, the defense of the gingival tissues and saliva activity.

A study conducted in Kazakhstan demonstrated that patients with bronchial asthma have a higher risk of developing chronic generalized periodontitis compared with healthy individuals. Asthmatic patients were

found to exhibit increased plaque levels (PI), greater gingival bleeding (BI), and more pronounced periodontal pocket depth (PD) [4]. According to the authors, long-term therapy with inhaled corticosteroids leads to suppression of local immunity, decreased saliva secretion and mucosal dehydration, which creates conditions for bacterial invasion into periodontal tissues. Complex treatment with emphasis on the use of local anti-inflammatory agents and remineralising solutions is recommended.

A study conducted in Tashkent investigated the characteristics of periodontal status in patients with bronchial asthma receiving inhaled glucocorticosteroids and bronchodilators. It was found that asthmatic patients exhibit hypertrophic gingivitis, a tendency toward dental deposit formation, and oral dryness due to reduced salivary secretion. Elevated levels of IgE in periodontal tissues indicated the influence of an allergic component on the progression of inflammatory periodontal diseases. Local application of gels containing ketoprofen and diclofenac was suggested as a promising approach to reduce periodontal inflammation without systemic effects [3].

Special attention has been given to the periodontal status of children with bronchial asthma. It has been noted that more than 60% of children receiving inhaled steroids develop catarrhal gingivitis, characterized by gingival bleeding and hyperemia. In children using powder inhalers, enamel demineralisation and the formation of dental erosions have also been observed. These changes are attributed to the systemic effects of corticosteroids and persistent mucosal dryness. The use of spacers to minimize contact of inhaled medications with the oral mucosa, along with the application of remineralising agents, has been recommended [8].

A study conducted in Dagestan confirmed a decline in bone mineral density in patients with bronchial asthma who are long-term users of inhaled corticosteroids. The findings indicated that prolonged use of steroids, exceeding five years, was associated with increased gingival recession and reduced alveolar bone levels, as assessed by radiography. These results are consistent with global evidence on the systemic effects of inhaled corticosteroids on bone metabolism. To prevent such outcomes, regular monitoring of jawbone condition and the administration of calcium supplements along with locally applied NSAIDs have been recommended [1].

The need for a personalized therapeutic approach for patients with bronchial asthma and persistent inflammatory periodontal disease has been emphasized. Reduced salivary secretion, changes in the oral microbial composition, and the systemic effects of inhaled medications necessitate comprehensive treatment, including the use of topical antiseptics, anti-inflammatory gels (ketoprofen, diclofenac), and remineralising pastes containing calcium and fluoride.

## 5 Conclusion

Bronchial asthma and its therapy have a multifactorial influence on the state of periodontal tissues. Disruption of saliva secretion, immunological imbalance, decreased bone mineral density and increased risk of candidiasis create prerequisites for an aggressive course of inflammatory periodontal diseases, which requires the development of individual preventive and therapeutic strategies for this category of patients.

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