International Journal of Pediatrics and Genetics
Vol. 4 No. 11 (2026)
ISSN: 2995-5483

Bronchial Asthma and Factors Influencing the Development of Periodontal Pathology

Atashev Shuhrat Ibragimovich
Urgench Ranch University of Technology

Abstract. Background: Bronchial asthma and periodontitis are common chronic inflammatory diseases, and a growing body of evidence suggests that they are associated. Aim: To summarise current evidence on the general somatic and local factors that influence the development of periodontal pathology in patients with bronchial asthma, with particular attention to the oral microbiota, immune-inflammatory mechanisms and microcirculatory disorders of the periodontium. Methods: A narrative review of international and national publications indexed in PubMed, Scopus, Google Scholar and eLIBRARY was carried out; the evidence was grouped into thematic blocks and summarised in tables and schematic figures. Results: Systematic reviews report higher plaque, gingival and bleeding indices in patients with asthma, and a meta-analysis of pulmonary diseases reported a pooled odds ratio of 3.03 for the association between asthma and periodontitis. Plausible mechanisms include the effects of inhaled corticosteroids and β2-agonists on salivary flow, mouth breathing, shared Th2/Th17-mediated inflammation, aspiration of periodontal pathogens and disorders of gingival microcirculation that can be assessed by laser Doppler flowmetry. Conclusion: Patients with bronchial asthma should be regarded as a risk group for inflammatory periodontal diseases; regular periodontal examination, hygiene education and assessment of gingival microcirculation are recommended, and prospective studies are needed to establish causality.

Keywords: bronchial asthma, periodontitis, risk factors, oral microbiota, inhaled corticosteroids, microcirculation, laser Doppler flowmetry.

INTRODUCTION

Periodontitis is one of the most prevalent chronic inflammatory diseases of humans: in 2010 severe periodontitis affected about 11.2% of the world's adult population [1], and in the United States 47.2% of adults aged 30 years and older had some form of periodontitis [2]. According to the current classification, periodontitis is a chronic multifactorial inflammatory disease associated with dysbiotic plaque biofilms and characterised by progressive destruction of the tooth-supporting apparatus [3].

Modern medicine has accumulated considerable clinical evidence of pathogenetic relationships between diseases of internal organs and inflammatory periodontal lesions. Diabetes mellitus and smoking are the best established general risk factors, and stress, hormonal changes during puberty, pregnancy and menopause, functional insufficiency of neutrophils and age are also discussed [4]. Associations of periodontitis with cardiovascular, endocrine, digestive and respiratory diseases have been described [5], and national studies emphasise the combined effect of somatic pathology, poor oral hygiene and dental anomalies on the development of chronic generalised periodontitis [6]. These data support the concept of inflammatory periodontal diseases as a local manifestation of disturbances in the whole organism [7].

Bronchial asthma is a heterogeneous disease characterised by chronic airway inflammation, variable expiratory airflow limitation and respiratory symptoms such as wheeze, shortness of breath and cough [8]. It affected an estimated 262 million people worldwide in 2019 [9]. A systematic review of 21 studies found that patients with asthma had significantly higher gingival bleeding, plaque and gingival

Vol. 4 No. 11 (2026)
ISSN: 2995-5483

indices than people without asthma [10], and a later meta-analysis of pulmonary diseases reported a pooled odds ratio of 3.03 for the association between asthma and periodontitis [11]. Nevertheless, the mechanisms of this association, in particular the role of the oral microbiota and periodontal microcirculation, remain insufficiently systematised.

The aim of this article is to summarise current evidence on the general somatic and local factors that influence the development of periodontal pathology in patients with bronchial asthma, and to identify the mechanisms that may serve as targets for prevention and early diagnosis.

MATERIALS AND METHODS

A narrative review of the literature was carried out. Publications were identified in the PubMed, Scopus, Google Scholar and eLIBRARY databases using combinations of the keywords “bronchial asthma”, “periodontitis”, “periodontal disease”, “oral microbiota”, “inhaled corticosteroids”, “microcirculation” and “laser Doppler flowmetry”. Systematic reviews and meta-analyses, epidemiological studies, consensus reports, experimental and clinical studies and monographs published in English and Russian were included. The diagnosis of periodontitis in the reviewed studies was interpreted in accordance with the current staging and grading framework [12].

The selected publications were grouped into four thematic blocks: general and local risk factors of periodontal pathology; the composition of the oral and subgingival microbiota; immune-inflammatory and asthma-specific mechanisms; and microcirculatory disorders of the periodontium and methods for their assessment. For each block the main conclusions and, where available, quantitative results were extracted from the original publications without modification. Because the studies differed in design, population and outcome measures, the evidence was synthesised descriptively, and no pooled statistical analysis was performed.

RESULTS

General and local risk factors. The development and course of periodontal pathology depend on the interaction of general and local factors. Among local factors, the quality of oral hygiene, the presence of bacterial plaque and supra- and subgingival calculus, anomalies of the soft tissues of the oral cavity, the type of dentoalveolar deformity, food retention due to caries or tooth loss and the quality of dentures and orthodontic appliances are of greatest importance [13]. The oral cavity provides favourable conditions for the reproduction of microorganisms: a slightly alkaline environment, food residues, optimal humidity and temperature, and numerous folds and niches of the dentition.

Oral microbiota. In healthy people the oral cavity is colonised by a diverse but stable microbial community dominated by streptococci and other commensal species [14]. In the gingival sulcus, aerobic bacteria of the genera Neisseria, Corynebacterium and Streptococcus and anaerobic bacteria of the genera Peptostreptococcus and Veillonella predominate. Local tolerance to these microorganisms is maintained by innate immune cells of the periodontal tissues, which constantly produce small amounts of pro-inflammatory cytokines and eliminate invading bacteria as long as their number does not exceed a certain threshold. Subgingival bacteria form stable complexes, and the “red complex” of Porphyromonas gingivalis, Tannerella forsythia and Treponema denticola is most closely associated with deep pockets and bleeding on probing [15] (Table 1).

Vol. 4 No. 11 (2026)
ISSN: 2995-5483

Table 1. Main microbial complexes of subgingival plaque and their clinical significance

Complex	Representative species	Clinical significance
Red	Porphyromonas gingivalis, Tannerella forsythia, Treponema denticola	Closely associated with pocket depth, bleeding on probing and progression of periodontitis
Orange	Fusobacterium nucleatum, Prevotella intermedia, Prevotella nigrescens, Parvimonas micra, Campylobacter spp.	Bridge species; precede and support colonisation by the red complex
Yellow	Streptococcus spp. (S. sanguinis, S. oralis, S. mitis, S. gordonii)	Early colonisers; predominate at healthy sites
Green	Capnocytophaga spp., Eikenella corrodens, Aggregatibacter actinomycetemcomitans serotype a	Early colonisers; compatible with periodontal health
Purple	Veillonella parvula, Actinomyces odontolyticus	Early colonisers; compatible with periodontal health

Note: compiled by the author on the basis of the classification of subgingival microbial complexes by S. S. Socransky et al.

An increase in microbial load and the reproduction of flora atypical for a healthy oral cavity lead to dysbiosis, in which a polymicrobial community disrupts host homeostasis and keystone pathogens such as P. gingivalis can subvert the immune response [16]. Studies in national populations have shown that the transition from health to inflammatory periodontal disease is accompanied by an increase in anaerobic bacteroids, fusobacteria, eubacteria and spirochaetes [17]. In severe chronic generalised periodontitis, the microflora of the periodontal pocket also contains higher proportions of Staphylococcus aureus, Streptococcus pyogenes, enterococci and Candida albicans, and the detection of A. actinomycetemcomitans, P. gingivalis, P. intermedia, T. forsythia and T. denticola correlates with the severity of the disease [18]. Strains isolated from patients with severe periodontitis express more pathogenicity and persistence factors, and translocation of microorganisms from the periodontal pocket into the bloodstream has been demonstrated [19].

Immune-inflammatory mechanisms. Periodontitis rarely develops evenly; more often attachment loss occurs in cycles, with exacerbations around individual teeth. During exacerbations the number of gram-negative, anaerobic, motile bacteria increases, direct microbial invasion of the tissues occurs, and areas of micronecrosis or abscesses form. Dysbiotic periodontal inflammation is not confined to the oral cavity: periodontal bacteria, their products and inflammatory mediators enter the circulation and contribute to systemic inflammation [20]. Patients with periodontitis have higher serum levels of C-reactive protein, interleukin-6 and neutrophil counts than periodontally healthy people [21]. Chronic periodontal infection therefore constantly diverts the body's immunological resources and may aggravate the course of systemic diseases, including diseases of the respiratory tract.

Asthma-specific factors. Several factors specific to bronchial asthma may increase the risk of periodontal pathology. Inhaled corticosteroids and β2-agonists reduce salivary flow and the concentration of salivary immunoglobulin A, which weakens the protective properties of saliva, and the mouth breathing typical of patients with nasal obstruction dries the gingiva and promotes plaque accumulation [22]. Asthma and periodontitis also share inflammatory pathways, including activation of neutrophils and production of interleukin-1β, interleukin-6 and tumour necrosis factor-α, while aspiration of periodontal pathogens may in turn contribute to inflammation of the lower airways. These mechanisms are summarised in Figure 1.

Vol. 4 No. 11 (2026)
ISSN: 2995-5483

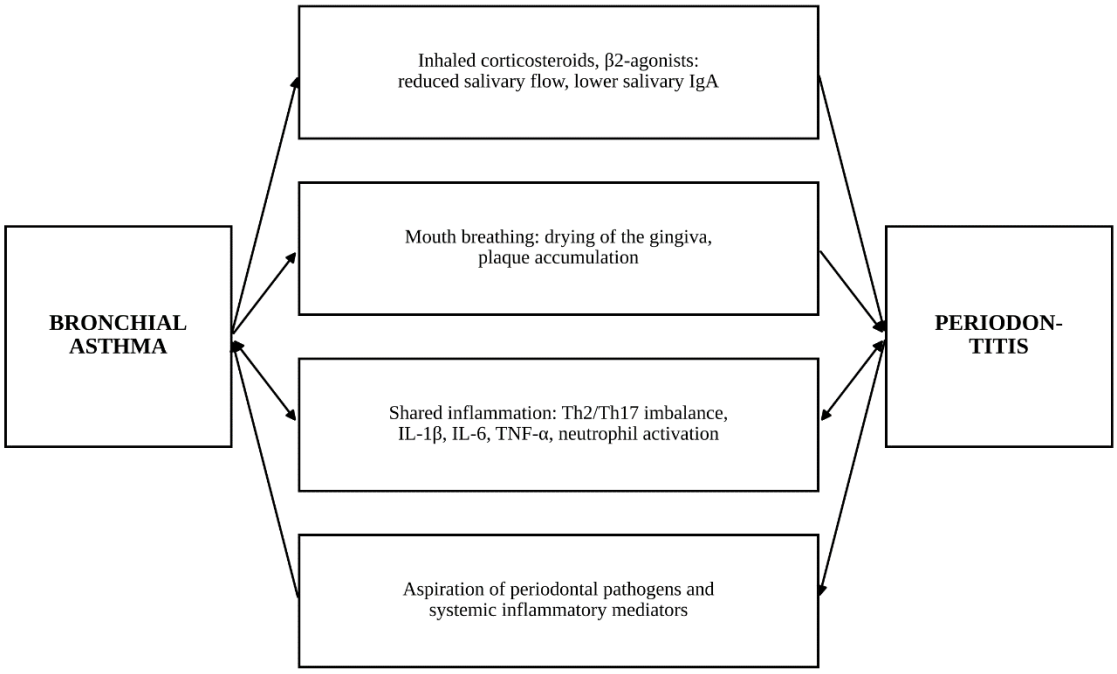


Figure 1. Mechanisms linking bronchial asthma and periodontitis

Microcirculatory disorders of the periodontium. Circulatory disorders play an important role in the pathogenesis of periodontal diseases, both in the periodontal tissues and in the general vascular network. An early sign of microcirculatory disorders in periodontitis is a local spasm of arterioles, congestion in the venular segment and a decrease in nutritive blood flow; impaired capillary blood flow, ending in capillary stasis, can be regarded as the central link of microcirculatory disorders. Laser Doppler flowmetry, based on the Doppler shift of laser light scattered by moving erythrocytes [23], allows gingival perfusion to be recorded non-invasively and in real time, and differences in local gingival blood flow between healthy and inflamed sites have been demonstrated with this method [24]. Analysis of the oscillatory components of the flowmetry signal makes it possible to distinguish the endothelial, neurogenic and myogenic mechanisms of microvascular tone and to calculate indices of neurogenic and myogenic tone and the microcirculation index (Figure 2).

Vol. 4 No. 11 (2026)
ISSN: 2995-5483

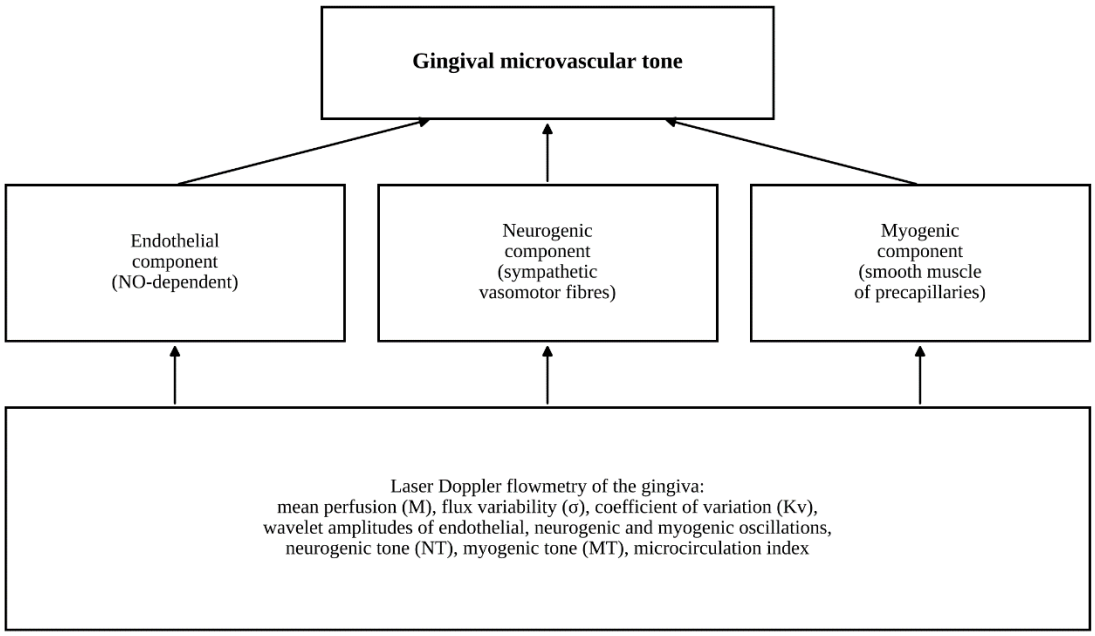


Figure 2. Components of gingival microvascular tone and indices assessed by laser Doppler flowmetry

DISCUSSION

The reviewed evidence indicates that bronchial asthma and periodontitis are associated, and that this association is probably bidirectional. Quantitative studies of the association are summarised in Table 2. The joint workshop of the European Federation of Periodontology and the European arm of the World Organization of Family Doctors examined the evidence linking periodontal diseases with respiratory diseases and called for closer cooperation between physicians and dentists [25]. The systematic review prepared for that workshop also showed that most available studies are cross-sectional or case–control, which limits conclusions about causality [26].

Table 2. Selected quantitative evidence on periodontitis and its association with bronchial asthma

Study	Design and population	Main result
Eke et al., 2012	NHANES 2009–2010; 3,742 adults aged ≥30 years, USA	47.2% of adults had periodontitis
Kassebaum et al., 2014	Systematic review and meta-regression, 1990–2010	Age-standardised global prevalence of severe periodontitis 11.2% in 2010
Moraschini et al., 2018	Systematic review and meta-analysis; 21 studies, 1979–2017	Higher gingival bleeding, plaque and gingival indices in patients with asthma ($p < 0.001$)
Wu et al., 2022	Meta-analysis of pulmonary diseases and periodontal health	Pooled OR for asthma and periodontitis 3.03; adjusted OR for pulmonary diseases overall 1.93 (95% CI 1.60–2.33)

Note: OR – odds ratio; CI – confidence interval. Compiled by the author from the cited publications.

The mechanisms described above suggest that the periodontal status of patients with asthma depends not only on oral hygiene but also on the treatment of the underlying disease, the pattern of breathing and the state of the microcirculation. From a clinical point of view, structural and functional changes in

periodontal microvessels detected by laser Doppler flowmetry may serve as an early diagnostic criterion of trophic disorders in the periodontium. Microcirculatory disorders in gingivitis and in mild and moderate periodontitis are focal, whereas with increasing severity they become generalised; the combination of periodontitis and asthma may therefore be associated with more pronounced changes in neurogenic and myogenic tone than either disease alone. This hypothesis, however, requires confirmation in controlled studies with standardised flowmetry protocols. The main limitations of the present review are its narrative design, the heterogeneity of the definitions of periodontitis and asthma in the original studies and the predominance of cross-sectional data.

CONCLUSION

Bronchial asthma and inflammatory periodontal diseases are linked by common risk factors and by several interacting mechanisms: changes in salivary secretion and immunity caused by inhaled medication, mouth breathing, dysbiosis of the subgingival microbiota with predominance of periodontopathogenic species, systemic inflammation and disorders of periodontal microcirculation. Patients with asthma should therefore be regarded as a risk group for periodontitis; it is advisable to include regular periodontal examination, individual oral hygiene training and rinsing of the mouth after inhalation in their management, and to use laser Doppler flowmetry to detect early microcirculatory disorders of the periodontium. Prospective studies are needed to establish whether periodontal treatment improves the control of bronchial asthma.

REFERENCES

[1] N. J. Kassebaum, E. Bernabé, M. Dahiya, B. Bhandari, C. J. L. Murray, and W. Marcenes, “Global burden of severe periodontitis in 1990–2010: A systematic review and meta-regression,” *J. Dent. Res.*, vol. 93, no. 11, pp. 1045–1053, 2014, doi: 10.1177/0022034514552491.

[2] P. I. Eke, B. A. Dye, L. Wei, G. O. Thornton-Evans, and R. J. Genco, “Prevalence of periodontitis in adults in the United States: 2009 and 2010,” *J. Dent. Res.*, vol. 91, no. 10, pp. 914–920, 2012, doi: 10.1177/0022034512457373.

[3] P. N. Papapanou *et al.*, “Periodontitis: Consensus report of workgroup 2 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions,” *J. Periodontol.*, vol. 89, no. S1, pp. S173–S182, 2018, doi: 10.1002/JPER.17-0721.

[4] R. J. Genco and W. S. Borgnakke, “Risk factors for periodontal disease,” *Periodontol. 2000*, vol. 62, no. 1, pp. 59–94, 2013, doi: 10.1111/j.1600-0757.2012.00457.x.

[5] G. J. Linden, A. Lyons, and F. A. Scannapieco, “Periodontal systemic associations: Review of the evidence,” *J. Clin. Periodontol.*, vol. 40, no. S14, pp. S8–S19, 2013, doi: 10.1111/jcpe.12064.

[6] L. M. Tebloeva and K. G. Gurevich, “Risk factors for the development of chronic generalized periodontitis,” *Institut Stomatologii*, no. 2, pp. 54–56, 2014 (in Russian).

[7] A. S. Grigoryan, A. I. Grudyanov, and N. A. Rabukhina, *Periodontal Diseases: Pathogenesis, Diagnosis, Treatment*. Moscow, Russia: MIA, 2004 (in Russian).

[8] Global Initiative for Asthma, *Global Strategy for Asthma Management and Prevention*, 2024 update. [Online]. Available: https://ginasthma.org

[9] World Health Organization, “Asthma,” Fact sheet, 2024. [Online]. Available: https://www.who.int/news-room/fact-sheets/detail/asthma

[10] V. Moraschini, J. A. Calasans-Maia, and M. D. Calasans-Maia, “Association between asthma and periodontal disease: A systematic review and meta-analysis,” *J. Periodontol.*, vol. 89, no. 4, pp. 440–455, 2018, doi: 10.1902/jop.2017.170363.

[11] Z. Wu *et al.*, “Pulmonary disease and periodontal health: A meta-analysis,” *Sleep Breath.*, vol. 26, pp. 1857–1868, 2022, doi: 10.1007/s11325-022-02577-3.

[12] M. S. Tonetti, H. Greenwell, and K. S. Kornman, "Staging and grading of periodontitis: Framework and proposal of a new classification and case definition," *J. Periodontol.*, vol. 89, no. S1, pp. S159–S172, 2018, doi: 10.1002/JPER.18-0006.

[13] A. I. Grudyanov and E. V. Fomenko, *Etiology and Pathogenesis of Inflammatory Periodontal Diseases*. Moscow, Russia: MIA, 2010 (in Russian).

[14] J. A. Aas, B. J. Paster, L. N. Stokes, I. Olsen, and F. E. Dewhirst, "Defining the normal bacterial flora of the oral cavity," *J. Clin. Microbiol.*, vol. 43, no. 11, pp. 5721–5732, 2005, doi: 10.1128/JCM.43.11.5721-5732.2005.

[15] S. S. Socransky, A. D. Haffajee, M. A. Cugini, C. Smith, and R. L. Kent Jr., "Microbial complexes in subgingival plaque," *J. Clin. Periodontol.*, vol. 25, no. 2, pp. 134–144, 1998, doi: 10.1111/j.1600-051X.1998.tb02419.x.

[16] R. P. Darveau, "Periodontitis: A polymicrobial disruption of host homeostasis," *Nat. Rev. Microbiol.*, vol. 8, no. 7, pp. 481–490, 2010, doi: 10.1038/nrmicro2337.

[17] O. A. Zorina, A. A. Kulakov, and A. I. Grudyanov, "Oral microbiocenosis in health and in inflammatory periodontal diseases," *Stomatologiya*, no. 1, pp. 73–78, 2011 (in Russian).

[18] L. A. Dmitrieva and A. G. Krainova, "Current concepts of the role of microflora in the pathogenesis of periodontal diseases," *Parodontologiya*, no. 1 (30), pp. 8–15, 2004 (in Russian).

[19] V. V. Labis, E. A. Bazikyan, and I. G. Kozlov, "Bacterial factor as a participant in the infectious-inflammatory process in the oral cavity," *Rossiiskii Stomatologicheskii Zhurnal*, no. 4, p. 19, 2013 (in Russian).

[20] G. Hajishengallis, "Periodontitis: From microbial immune subversion to systemic inflammation," *Nat. Rev. Immunol.*, vol. 15, no. 1, pp. 30–44, 2015, doi: 10.1038/nri3785.

[21] B. G. Loos, "Systemic markers of inflammation in periodontitis," *J. Periodontol.*, vol. 76, no. 11S, pp. 2106–2115, 2005, doi: 10.1902/jop.2005.76.11-S.2106.

[22] M. S. Thomas, A. Parolia, M. Kundabala, and M. Vikram, "Asthma and oral health: A review," *Aust. Dent. J.*, vol. 55, no. 2, pp. 128–133, 2010, doi: 10.1111/j.1834-7819.2010.01226.x.

[23] M. D. Stern, "In vivo evaluation of microcirculation by coherent light scattering," *Nature*, vol. 254, no. 5495, pp. 56–58, 1975, doi: 10.1038/254056a0.

[24] C. Gleissner, O. Kempski, S. Peylo, J. H. Glatzel, and B. Willershausen, "Local gingival blood flow at healthy and inflamed sites measured by laser Doppler flowmetry," *J. Periodontol.*, vol. 77, no. 10, pp. 1762–1771, 2006, doi: 10.1902/jop.2006.050194.

[25] D. Herrera *et al.*, "Association between periodontal diseases and cardiovascular diseases, diabetes and respiratory diseases: Consensus report of the Joint Workshop by the European Federation of Periodontology (EFP) and the European arm of the World Organization of Family Doctors (WONCA Europe)," *J. Clin. Periodontol.*, vol. 50, no. 6, pp. 819–841, 2023, doi: 10.1111/jcpe.13807.

[26] A. Molina *et al.*, "The association between respiratory diseases and periodontitis: A systematic review and meta-analysis," *J. Clin. Periodontol.*, vol. 50, no. 6, pp. 842–887, 2023, doi: 10.1111/jcpe.13767.