

Periodontal Status in Patients with Pulmonary Tuberculosis: Modern Concepts of Pathogenesis and Clinical Manifestations

Nodirkhon Akhrorkhodjaev

Fakulty Therapeutic Dentistry Department, PhD, Associate Professor, Tashkent State Medical University, Tashkent, Uzbekistan

Nargiza Parpiyeva

Director of the Republican Scientific and Practical Medical Center for Phthysiology and Pulmonology, Chief Phthysiatrician of the Republic, MD, Professor. Head of the Department of Phthysiology, Tashkent State Medical University, Tashkent, Uzbekistan

Aliya Kadirbaeva

Hospital Therapeutic Dentistry Department, MD, Associate Professor, Tashkent State Medical University, Tashkent, Uzbekistan

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Abstract

Pulmonary tuberculosis remains one of the leading infectious diseases worldwide and is accompanied by profound immunological, metabolic, and inflammatory alterations that significantly affect the condition of oral tissues. Numerous investigations indicate that patients with pulmonary tuberculosis demonstrate a high prevalence of inflammatory periodontal diseases, including chronic generalized gingivitis and generalized periodontitis. Immune dysregulation, chronic intoxication, oxidative stress, nutritional deficiencies, smoking, poor oral hygiene, and long-term anti-tuberculosis therapy contribute to periodontal tissue destruction in this category of patients. The present review summarizes contemporary scientific data regarding periodontal status in patients with pulmonary tuberculosis and analyzes the principal pathogenic mechanisms involved in periodontal alterations associated with tuberculosis infection.

Keywords: Pulmonary tuberculosis, periodontal diseases, periodontitis, oral health, gingivitis, periodontal tissues.

Introduction

Pulmonary tuberculosis is a chronic infectious disease caused by *Mycobacterium tuberculosis* and continues to represent a serious global medical and social problem. According to the World Health Organization, tuberculosis remains among the major causes of mortality from infectious diseases worldwide, particularly in developing countries and regions with unfavorable socioeconomic conditions.

In addition to pulmonary involvement, tuberculosis exerts systemic effects on multiple organs and tissues, including the oral cavity. Chronic tuberculosis infection is associated with prolonged inflammatory reactions, immune dysfunction, endogenous intoxication, oxidative stress,

and nutritional disturbances, all of which may negatively influence the condition of periodontal tissues [1].

The periodontium represents a complex structural and functional system consisting of gingiva, periodontal ligament, cementum, and alveolar bone. The integrity of periodontal tissues depends on balanced microbial flora, adequate immune response, proper vascularization, and satisfactory oral hygiene. Any disturbance in these mechanisms may contribute to the development and progression of inflammatory periodontal diseases [2].

Recent studies indicate that patients with pulmonary tuberculosis frequently present with gingivitis, chronic generalized periodontitis, increased periodontal pocket

depth, gingival bleeding, alveolar bone resorption, and unsatisfactory oral hygiene indices. Furthermore, periodontal inflammation may itself contribute to systemic inflammatory burden and negatively influence the course of respiratory diseases [3].

The purpose of this review is to summarize modern scientific literature concerning periodontal status in patients with pulmonary tuberculosis and to analyze the principal pathogenic and clinical aspects of periodontal alterations associated with tuberculosis infection.

Epidemiology of Periodontal Diseases in Patients with Pulmonary Tuberculosis

Numerous epidemiological investigations demonstrate increased prevalence and severity of periodontal diseases among patients with pulmonary tuberculosis compared with healthy individuals. Clinical studies performed in tuberculosis hospitals and pulmonary departments have revealed high frequency of chronic generalized gingivitis and generalized periodontitis in this patient population [4].

According to contemporary clinical observations, the severity of periodontal destruction often correlates with the duration and clinical severity of pulmonary tuberculosis. Patients with prolonged disease course, extensive pulmonary involvement, chronic intoxication syndrome, and immunosuppression tend to demonstrate more pronounced periodontal tissue destruction.

Several investigators reported significantly increased periodontal pocket depth, clinical attachment loss, gingival bleeding, and plaque accumulation among tuberculosis patients. Moreover, the prevalence of periodontitis appears to be particularly high among middle-aged and elderly male patients suffering from chronic pulmonary tuberculosis [5,6].

Socioeconomic factors also play an important role in periodontal deterioration among tuberculosis patients. Poor living conditions, limited access to dental care, malnutrition, tobacco smoking, alcohol consumption, and inadequate oral hygiene contribute substantially to progression of inflammatory periodontal diseases in this category of patients.

Pathogenetic Mechanisms of Periodontal Alterations in Tuberculosis

Pulmonary tuberculosis is accompanied by profound alterations in both cellular and humoral immunity. Chronic activation of the immune system results in increased production of pro-inflammatory cytokines including tumor necrosis factor-alpha (TNF- α), interleukin-1 β (IL-1 β), interleukin-6 (IL-6), and interferon-gamma (IFN- γ). These mediators play a critical role in tissue destruction and bone resorption within periodontal tissues [7].

Persistent immune activation and dysregulation of macrophage and T-cell function impair local protective mechanisms within the oral cavity, thereby increasing susceptibility to chronic periodontal inflammation and bacterial colonization. Immune imbalance may additionally reduce regenerative capacity of periodontal tissues and contribute to progression of attachment loss

[8].

Chronic Inflammation and Oxidative Stress

Both pulmonary tuberculosis and periodontal diseases are characterized by chronic inflammatory processes accompanied by increased oxidative stress. Excessive generation of reactive oxygen species leads to cellular damage, collagen degradation, epithelial injury, and microvascular dysfunction [9,10].

Oxidative stress contributes significantly to destruction of periodontal connective tissue and alveolar bone. Persistent inflammatory stimulation may further aggravate periodontal breakdown and impair tissue healing processes in patients with tuberculosis.

Nutritional Deficiencies

Patients with pulmonary tuberculosis frequently suffer from protein-energy malnutrition and deficiencies of vitamins. Deficiency of vitamins A, C, D, and B-complex negatively affects collagen synthesis, epithelial regeneration, immune response, and bone metabolism.

Nutritional insufficiency contributes to gingival bleeding, delayed wound healing, increased vascular permeability, and progression of periodontal destruction. Reduced resistance of oral tissues additionally predisposes patients to secondary infections and chronic inflammatory lesions [11-12].

Influence of Anti-Tuberculosis Therapy

Long-term administration of anti-tuberculosis medications may also negatively influence oral and periodontal health. Anti-tuberculosis drugs are capable of causing xerostomia, dysbiosis of oral microflora, mucosal irritation, opportunistic fungal infections, and alterations in salivary composition.

Reduced salivary secretion impairs the natural cleansing and antimicrobial functions of saliva, thereby facilitating plaque accumulation and progression of periodontal inflammation. Long-term pharmacotherapy may also influence oral microbial ecology and contribute to opportunistic infections within periodontal tissues [13].

Clinical Manifestations of Periodontal Diseases in Tuberculosis Patients

Patients with pulmonary tuberculosis commonly present with signs of chronic gingival inflammation. Clinical manifestations include gingival hyperemia, edema, cyanotic discoloration, bleeding on probing, and gingival tenderness. Chronic catarrhal gingivitis is considered one of the earliest and most frequent periodontal manifestations observed in tuberculosis patients [14].

In some cases, gingival tissues may demonstrate hypertrophic or granulomatous alterations resembling nonspecific inflammatory lesions. Gingival enlargement and spontaneous bleeding may occur in severe inflammatory forms associated with immune dysfunction and chronic infection.

Progression of inflammatory periodontal destruction leads to formation of periodontal pockets and clinical attachment loss. Deep periodontal pockets create favorable conditions for retention of pathogenic anaerobic microorganisms and chronic suppurative inflammation.

Clinical studies demonstrate significantly increased probing pocket depth and attachment loss among patients with pulmonary tuberculosis compared with healthy control groups. In advanced cases, purulent exudation, tooth mobility, and periodontal abscess formation may develop [15-16].

Radiographic investigations reveal generalized horizontal alveolar bone resorption in many tuberculosis patients. Bone destruction tends to progress more rapidly in individuals with severe pulmonary involvement, chronic intoxication syndrome, smoking habits, and nutritional deficiencies.

Chronic inflammatory activation and cytokine-mediated osteoclastic activity contribute significantly to alveolar bone loss and progression of generalized periodontitis in tuberculosis patients.

Most clinical investigations report unsatisfactory oral hygiene among patients with pulmonary tuberculosis. Significant accumulation of dental plaque and calculus contributes to persistence and progression of chronic periodontal inflammation.

Poor oral hygiene in tuberculosis patients is associated with multiple factors including severe general condition, prolonged hospitalization, chronic weakness, low socioeconomic status, limited access to dental care, and reduced patient motivation. Tobacco smoking additionally aggravates oral hygiene deterioration and periodontal tissue destruction.

The periodontal pockets of tuberculosis patients contain complex polymicrobial biofilms similar to those observed in chronic generalized periodontitis. Increased colonization by anaerobic periodontal pathogens such as *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia* has been reported [17].

Certain studies additionally detected *Mycobacterium tuberculosis* DNA within saliva and dental plaque samples of tuberculosis patients suffering from periodontitis, suggesting possible interaction between tuberculosis infection and oral microbial ecology.

Diagnosis of periodontal diseases in patients with pulmonary tuberculosis requires comprehensive clinical and radiological examination. Assessment includes evaluation of oral hygiene indices, gingival inflammation, periodontal pocket depth, clinical attachment loss, tooth mobility, and radiographic analysis of alveolar bone condition.

Microbiological and immunological investigations may additionally be performed in complicated or atypical cases. Prior to invasive dental procedures, consultation with a phthisiatrician is recommended in order to assess the patient's systemic condition and infection control

requirements [18].

Regular dental examinations and preventive periodontal care are essential components of medical rehabilitation in patients with pulmonary tuberculosis. Early diagnosis and timely periodontal treatment may reduce inflammatory burden and improve overall quality of life [19-20].

Conclusion

Pulmonary tuberculosis exerts significant adverse effects on periodontal tissues through complex mechanisms involving chronic inflammation, immune dysregulation, oxidative stress, malnutrition, microbial imbalance, and prolonged pharmacotherapy. Patients with pulmonary tuberculosis demonstrate increased prevalence and severity of gingivitis and generalized periodontitis compared with healthy individuals.

Periodontal diseases in tuberculosis patients are characterized by gingival inflammation, periodontal pocket formation, attachment loss, alveolar bone resorption, and poor oral hygiene status. These pathological changes may further aggravate systemic inflammatory burden and negatively affect general health condition.

Comprehensive preventive dental care, early diagnosis, and multidisciplinary management are essential for maintaining periodontal health in patients with pulmonary tuberculosis. Further clinical and molecular investigations are necessary to clarify the precise pathogenic relationship between tuberculosis infection and periodontal diseases and to optimize preventive and therapeutic strategies for this category of patients.

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