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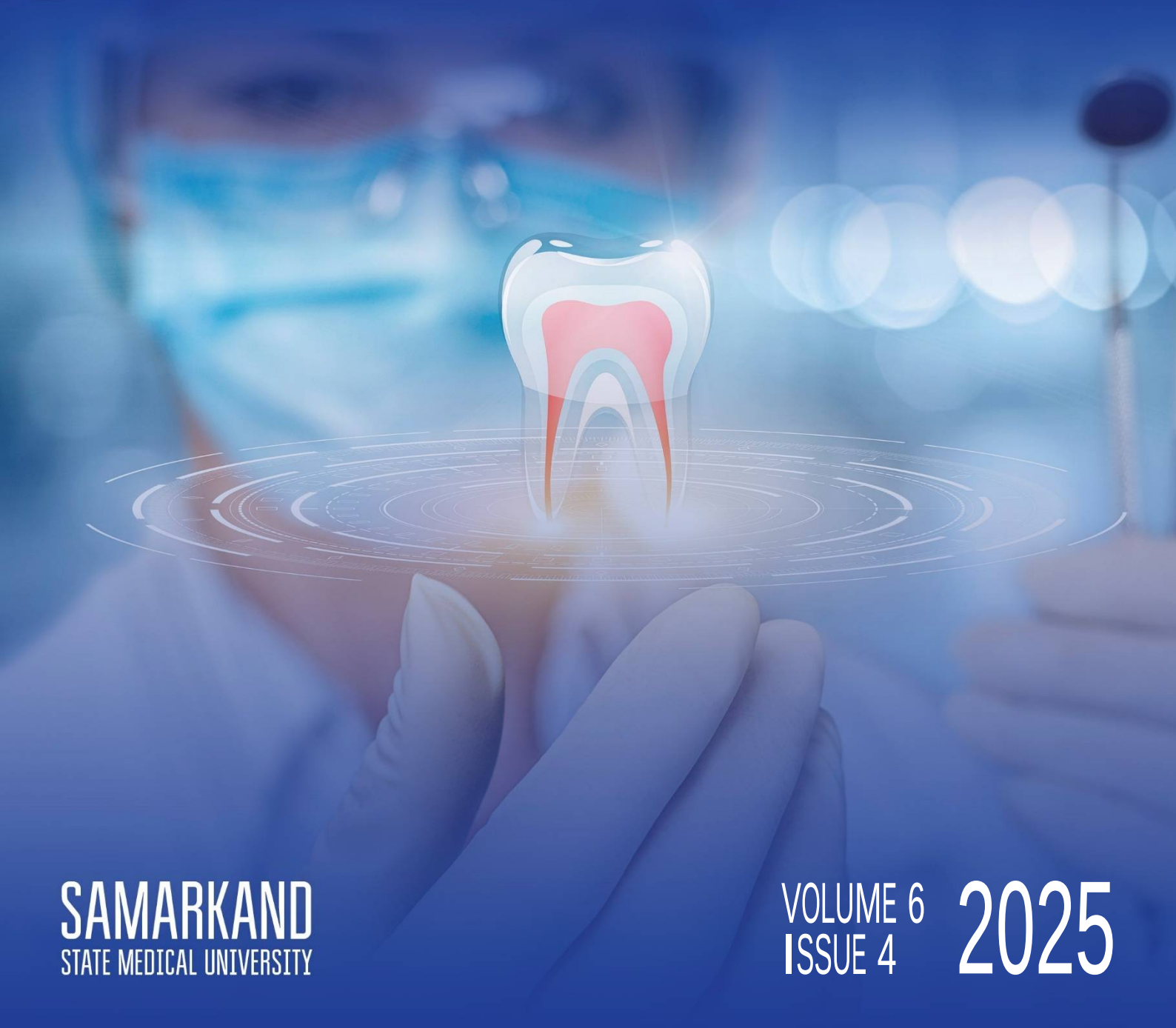

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MODERN CONCEPTS OF ETIOPATHOGENESIS AND CLINICAL CHARACTERISTICS OF CHRONIC GENERALIZED PERIODONTITIS: A LITERATURE REVIEW

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ANNOTATION

This review summarizes contemporary scientific evidence on the etiopathogenesis and clinical features of chronic generalized periodontitis (CGP). Chronic periodontitis is described as a multifactorial immunoinflammatory disorder resulting from the interaction between pathogenic anaerobic biofilms and a dysregulated host response. Key pathogenic mechanisms include microbial dysbiosis, activation of pro-inflammatory cytokines, oxidative stress, microcirculatory impairment, and imbalance of bone remodeling.

Clinical manifestations such as gingival inflammation, periodontal pocket formation, attachment loss, alveolar bone resorption, and tooth mobility reflect progressive structural and functional deterioration. Numerous studies highlight the roles of systemic conditions (diabetes, cardiovascular disease, chronic kidney disease), behavioral habits (smoking, poor oral hygiene, stress), and anatomical factors in modifying disease severity. Understanding these mechanisms is essential for improving diagnostic accuracy and developing comprehensive treatment strategies aimed at halting progression and preserving periodontal stability.

Keywords: chronic generalized periodontitis, etiopathogenesis, biofilm, inflammation, microcirculation, bone resorption, cytokines.

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СОВРЕМЕННЫЕ ПРЕДСТАВЛЕНИЯ ОБ ЭТИОПАТОГЕНЕЗЕ И КЛИНИЧЕСКИХ ОСОБЕННОСТЯХ ХРОНИЧЕСКОГО ГЕНЕРАЛИЗОВАННОГО ПАРОДОНТИТА: ОБЗОР ЛИТЕРАТУРЫ

АННОТАЦИЯ

Обзор обобщает современные научные данные об этиопатогенезе и клинических проявлениях хронического генерализованного пародонтита (ХГП). ХГП рассматривается как многофакторное иммуновоспалительное заболевание, возникающее вследствие взаимодействия патогенной анаэробной микрофлоры и дисрегулированного иммунного ответа организма. К ключевым механизмам разрушения тканей относятся микробный дисбиоз, активация провоспалительных цитокинов, оксидативный стресс, нарушение микроциркуляции и дисбаланс костного ремоделирования.

Клиническая картина характеризуется гингивитом, углублением пародонтальных карманов, потерей прикрепления, резорбцией альвеолярной кости и подвижностью зубов. Литературные данные подчёркивают значимость системных заболеваний (сахарный диабет, сердечно-сосудистые нарушения, хроническая болезнь почек), поведенческих факторов и анатомических особенностей, усиливающих течение ХГП. Понимание данных механизмов необходимо для совершенствования диагностики и комплексного лечения с целью стабилизации процесса и сохранения пародонта.

Ключевые слова: хронический генерализованный пародонтит, этиопатогенез, биоплёнка, воспаление, микроциркуляция, резорбция кости, цитокины.

Yusupova Manzuraxon Qabuljon qizi
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SURUNKALI UMUMLASHGAN PARODONTITNING ETIOPATOGENEZI VA KLINIK XUSUSIYATLARI BO‘YICHA ZAMONAVIY ILMIY QARASHLAR: ADABIYOTLAR SHARHI

ANNOTATSIYA

Ushbu sharh surunkali umumlashgan parodontitning etiopatogenezi va klinik xususiyatlariga oid zamonaviy ilmiy ma'lumotlarni umumlashtiradi. Surunkali umumlashgan parodontit ko'p omilli immun-yallig'lanish kasalligi bo'lib, patogen anaerob biofilm va organizmning disbalanslangan immun javobi o'zaro ta'siri natijasida rivojlanadi. To'qimalarning yemirilishiga olib keluvchi asosiy mexanizmlar qatoriga mikrobiotsenozning buzilishi, yallig'lanish sitokinlarining ortishi, oksidlovchi stress, mikrotsirkulyatsiya buzilishi hamda suyak remodellasiyasining izdan chiqishi kiradi.

Kasallikning klinik manzarasi gingivaning yallig'lanishi, parodontal cho'ntaklar chuqurlashuvi, birikish yo'qolishi, alveolyar suyak rezorbsiyasi va tishlarning harakatchanligi bilan ifodalanadi. Adabiyotlar sharhida diabet, yurak-qon tomir kasalliklari, surunkali buyrak yetishmovchiligi kabi tizimli holatlar, shuningdek xulq-atvor omillari va anatomik xususiyatlarning kasallik og'irligiga ta'siri ta'kidlangan. Ushbu mexanizmlarni chuqur tushunish diagnostika va davolash strategiyalarini takomillashtirish uchun muhimdir.

Kalit so'zlar: surunkali umumlashgan parodontit, etiopatogenez, biofilm, yallig'lanish, mikrotsirkulyatsiya, suyak rezorbsiyasi, sitokinlar.

Introduction. Chronic generalized periodontitis (CGP) represents one of the most widespread inflammatory–destructive diseases of the oral cavity, posing a significant global health burden and remaining a leading cause of tooth loss in adults. Numerous epidemiological surveys have demonstrated that the prevalence of periodontitis varies between 60% and 85% among adults, depending on age, socioeconomic status, oral hygiene, systemic health and environmental factors. Despite advances in diagnostic methods, biomaterials, and therapeutic approaches, chronic periodontitis continues to progress in many patients, leading to irreversible destruction of periodontal tissues, including the gingiva, periodontal ligament, cementum and alveolar bone [1.2].

The chronicity of the disease is attributed to its complex, multifactorial etiopathogenesis, where microbial biofilms interact with the host immune–inflammatory response, triggering a cascade of pathological processes such as cytokine activation, oxidative stress, vascular impairment and bone remodeling disturbances. Modern literature emphasizes that periodontitis is not merely an infectious disease but a host-mediated immunoinflammatory disorder, in which tissue destruction results largely from dysregulated immune mechanisms rather than microbial load alone[3].

Scientific findings over the past decades have shown that chronic generalized periodontitis is strongly influenced by systemic conditions such as diabetes mellitus, chronic kidney disease, cardiovascular disorders, hormonal imbalances and age-related physiological changes. The presence of harmful habits (e.g. smoking), inadequate oral hygiene, genetic predisposition and stress-related factors further exacerbates disease progression[3.5].

The purpose of this review is to summarize and critically analyze modern scientific data on the epidemiology, etiological factors, microbiological aspects, mechanisms of inflammation and immunity, microcirculatory disorders, bone metabolism changes and contemporary therapeutic concepts in chronic generalized periodontitis.

Epidemiology of chronic generalized periodontitis

Epidemiological studies consistently demonstrate that chronic generalized periodontitis is highly prevalent worldwide. The disease typically increases with age and becomes particularly common after 40 years, affecting both men and women. According to global assessments, approximately 15–20% of adults experience severe periodontal destruction, while moderate forms are observed in more than 50% of the population.

Research shows that:

- The prevalence rises sharply between the ages of 35 and 60, where connective tissue turnover decreases, salivary composition changes and immune function becomes less efficient.
- Individuals with systemic diseases—especially chronic kidney disease, diabetes mellitus, cardiovascular and metabolic disorders—exhibit significantly higher rates of periodontitis.
- Socioeconomic and behavioral factors—poor oral hygiene, low frequency of dental visits, high carbohydrate intake, smoking and stress—serve as major contributors.

The epidemiology of CGP reflects a complex interaction between environmental influences, oral microbiota and systemic health. Available data also indicate that untreated periodontitis can lead not only to tooth loss but also to systemic consequences via chronic inflammation, bacteremia and cytokine release [4.5.7].

Etiology and risk factors of chronic generalized periodontitis

The etiopathogenesis of CGP is multifactorial and results from a complex interaction between microbial biofilms, local environmental conditions, systemic health status and host immune responses. The development and progression of periodontitis depend on several interrelated groups of factors: microbial, local anatomical–functional, behavioral, systemic and genetic.

Microbial factors play a central initiating role. The subgingival microbiota in CGP consists of gram-negative anaerobic species capable of producing endotoxins, proteolytic enzymes, hydrogen sulfide, ammonia, short-chain fatty acids and tissue-degrading metabolites. The dominant role of the so-called "red complex" microorganisms is widely recognized: *Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*. These bacteria possess high virulence and induce strong inflammatory and immune reactions. They express lipopolysaccharides (LPS), gingipains, flagellins and hemolysins, which destroy gingival epithelial barriers, activate neutrophils, macrophages and T-lymphocytes, and stimulate the release of interleukins and matrix metalloproteinases[5.8].

Local predisposing factors that accelerate periodontal tissue breakdown include malpositioned teeth and occlusal disharmony, traumatic occlusion and overload, plaque retention areas (crowding, calculus, faulty restorations), thin gingival biotype and insufficient attached gingiva, as well as mouth breathing and reduced salivary flow. These factors contribute to plaque accumulation, impair self-cleaning mechanisms and increase susceptibility to inflammation.

Behavioral and lifestyle factors play an important modifying role. Poor oral hygiene, irregular dental visits, smoking, high-carbohydrate diet, and psychoemotional stress are among the most important risk factors. Smoking is associated with vasoconstriction, impaired neutrophil activity, altered fibroblast

function and a reduced gingival bleeding response, masking disease severity while accelerating tissue destruction. Stress-induced immunosuppression increases vulnerability to chronic inflammation[9.11].

Systemic conditions significantly elevate the risk and severity of CGP. These include diabetes mellitus (microangiopathy, impaired neutrophils, increased advanced glycation end-products), cardiovascular diseases (systemic inflammation, endothelial dysfunction), chronic kidney disease (uremic toxins, secondary immunodeficiency, xerostomia, bone metabolism disturbances), osteoporosis (reduced bone density), hormonal disorders (thyroid dysfunction, menopause-related changes) and metabolic syndrome with obesity (increased pro-inflammatory cytokines). Genetic polymorphisms in cytokine genes (IL-1, IL-6, TNF-α), Toll-like receptors and enzymes involved in oxidative balance also influence individual susceptibility[4.12].

In summary, bacteria initiate the process, host immunity mediates the tissue response, systemic conditions modify the course, behavioral factors accelerate progression, and genetic background determines vulnerability.

Microbiology of chronic generalized periodontitis: biofilm architecture and microbial interactions

The microbiological landscape of chronic generalized periodontitis (CGP) represents one of the central pathogenic components in the development and progression of the disease. CGP is characterized by a complex polymicrobial community dominated by gram-negative anaerobic bacteria organized into a structured biofilm that adheres firmly to the tooth root surface and periodontal pocket epithelium. This biofilm is not a random accumulation of microorganisms but a highly organized and cooperative ecosystem in which microbial synergy, nutrient exchange and collective pathogenicity determine the severity of periodontal destruction[5.10].

The formation of the periodontal biofilm begins with the attachment of early colonizers, primarily *Streptococcus*, *Actinomyces* and *Veillonella* species, which create the initial conditioning layer. These microorganisms pave the way for secondary colonizers by modifying the local environment, consuming oxygen, lowering redox potential and producing extracellular polymeric substances (EPS), thereby transforming the gingival sulcus into an anaerobic niche suitable for the proliferation of pathogenic anaerobes. As the biofilm matures, late colonizers such as *Porphyromonas gingivalis*, *Tannerella forsythia* and *Treponema denticola*—the “red complex”—become predominant.

Microbial interactions within the biofilm are cooperative and mutually reinforcing. *P. gingivalis* produces enzymes that degrade immunoglobulins and complement proteins, weakening the host defense and enabling deeper penetration of spirochetes such as *T. denticola*. *T. forsythia* contributes to epithelial detachment through its proteolytic enzymes and induces high levels of pro-inflammatory cytokines. Other key pathogens include *Prevotella intermedia*, *Fusobacterium nucleatum*, *Campylobacter rectus* and *Aggregatibacter actinomycetemcomitans*, each playing a distinct role in altering tissue homeostasis.

The structure of the periodontal biofilm provides exceptional resistance to mechanical removal, antibiotics and oxidative stress. The extracellular matrix composed of polysaccharides, proteins, DNA fragments and lipids creates a diffusion barrier that inhibits the penetration of antimicrobial agents. Quorum sensing regulates collective behavior such as virulence expression, nutrient sharing and formation of microcolonies. Dysbiosis occurs when pathogenic anaerobes outgrow beneficial microorganisms such as *Streptococcus salivarius* and *Lactobacillus* species, amplifying inflammatory and destructive processes and promoting deeper pocket formation[6.9].

Pathogenesis of chronic generalized periodontitis

The pathogenesis of chronic generalized periodontitis (CGP) is a multifactorial and dynamic process that encompasses microbial colonization, host immune–inflammatory responses, vascular and microcirculatory disturbances, oxidative stress and progressive bone remodeling imbalance. The destructive events seen in CGP are not the direct consequence of bacterial invasion alone, but rather the result of a dysregulated host reaction to a persistent microbial challenge.

The process begins with the accumulation of gram-negative anaerobic bacteria in the subgingival biofilm. Their lipopolysaccharides, proteases and metabolic products stimulate innate immunity. Neutrophils, despite being present in large numbers, fail to eliminate the biofilm due to its structural resistance. Activated neutrophils release reactive oxygen species and proteolytic enzymes, which contribute to collateral tissue damage. Macrophages, dendritic cells and epithelial cells recognize microbial antigens through Toll-like receptors and initiate a signaling cascade that produces large quantities of cytokines such as IL-1β, IL-6, TNF-α and chemokines that amplify inflammatory cell recruitment[14.15].

Persistent activation of these pathways transforms the gingival tissue into a chronic inflammatory environment. Prostaglandin E2 and matrix metalloproteinases degrade collagen of the periodontal ligament and gingival connective tissue. Over time, inflammation disrupts the junctional epithelium, deepening periodontal pockets and fostering further microbial colonization.

Microcirculatory dysfunction plays a key role. Gingival blood vessels undergo structural and functional alterations due to chronic inflammation: endothelial swelling, impaired vasomotor regulation and increased permeability lead to edema and tissue hypoxia. Hypoxic conditions stimulate angiogenesis, inflammatory cytokine release and fibroblast dysfunction. Vasoconstriction combined with stasis of blood flow weakens oxygen and nutrient supply, further impairing tissue resistance. Disturbances in perfusion parameters confirm the major contribution of vascular pathology to sustaining chronic inflammation[17.20].

Bone remodeling becomes unbalanced under the influence of inflammatory mediators. Cytokines released by macrophages and T-cells upregulate RANKL expression in periodontal ligament cells and osteoblasts, promoting osteoclast differentiation and bone resorption. Meanwhile, the expression of osteoprotegerin, a natural inhibitor of RANKL, decreases. The resulting RANKL/OPG imbalance shifts bone metabolism toward destructive resorption. Oxidative stress further damages cells and matrix components, potentiates osteoclast activation and inhibits osteoblast function.

Fibroblast dysfunction, reduced collagen synthesis and increased collagenase activity weaken the structural integrity of the periodontal ligament. As the ligament becomes disorganized, mechanical stability diminishes, and teeth develop pathological mobility. Excessive occlusal load then acts as an additional damaging factor, accelerating periodontal breakdown.

Clinical features of chronic generalized periodontitis

The clinical picture of chronic generalized periodontitis reflects the cumulative result of microbial,

immunoinflammatory, vascular and structural disturbances occurring within the periodontal tissues. Early stages may remain asymptomatic, with patients noticing only occasional bleeding during brushing. One of the earliest clinical signs is gingival inflammation manifested by redness, swelling and bleeding upon probing. As the disease advances, the junctional epithelium migrates apically, forming periodontal pockets that accumulate microbial biofilm and inflammatory exudate.

Gingival morphology changes: loss of stippling, edema, soft consistency and bluish-red discoloration due to venous stasis. Chronic inflammation leads to progressive destruction of the periodontal ligament and increased tooth mobility. Patients frequently report discomfort while chewing, and occlusal trauma exacerbates tissue destruction. Radiographically, generalized alveolar bone loss, vertical and horizontal defects, and widening of the periodontal ligament space are observed.

Halitosis is a frequent complaint and results from volatile sulfur compounds produced by anaerobic bacteria. Subgingival and supragingival calculus serve as persistent plaque-retentive factors. Tooth migration, diastema formation, pathological flaring and occlusal disharmony occur in advanced stages. Gingival recession exposes root surfaces, causing dentinal hypersensitivity and increasing susceptibility to root caries, erosion and abrasion.

Exacerbations present with acute pain, swelling and suppuration from periodontal pockets, corresponding to shifts in microbial composition, immune imbalance or acute trauma. Without treatment, progressive attachment loss ultimately results in tooth loss, impaired mastication and reduced quality of life.

Conclusion. This literature review demonstrates that chronic generalized periodontitis is a complex immunoinflammatory disease driven by microbial dysbiosis, host inflammatory activity, vascular disturbances and bone metabolism imbalance. Microbial biofilms initiate the process, but tissue destruction is mainly mediated by the host's dysregulated response. Systemic diseases, behavioral habits and local anatomical factors significantly modulate disease severity and progression.

Successful management requires comprehensive strategies aimed at controlling the microbial biofilm, modulating host immune responses, improving microcirculation and stabilizing biomechanical forces within the dentition. A thorough understanding of etiopathogenesis and clinical characteristics of CGP forms the basis for early diagnosis, individualized treatment planning and long-term preservation of periodontal stability.

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