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CHRONIC GENERALIZED CATARRHAL GINGIVITIS AND CHRONIC GENERALIZED PERIODONTITIS: COMMON ORIGINS, SEQUENTIAL TRANSITION (DISCUSSION)

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✓ ***Resume***

This article discusses the causes, conditions and mechanisms of transformation of gum inflammation (gingivitis) into inflammation of periodontal tissue, characterized by progressive destruction of the periodontium and alveolar bone (periodontitis)

Key words: inflammation, pathogenesis, chronic generalized catarrhal gingivitis, chronic generalized periodontitis

SURUNKALI UMUMIY KATARAL GINGIVIT VA SURUNKALI UMUMIY PARODONTIT: UMUMIY KELIB CHIQISHI, BOSQICHMA-BOSQICH O'TISHI

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✓ ***Rezyume***

Ushbu maqolada milk yallig'lanishining (gingivit) parodont to'qimalarning yallig'lanishiga aylanishining sabablari, shartlari va mexanizmlari ko'rib chiqiladi, bu periodont va alveolyar suyakning (parodontit) progressiv destruksiya bo'lishi bilan tavsiflanadi.

Kalit so'zlar: yallig'lanish, patogenez, surunkali umumiy kataral gingivit, surunkali umumiy parodontit

ХРОНИЧЕСКИЙ ГЕНЕРАЛИЗОВАННЫЙ КАТАРАЛЬНЫЙ ГИНГИВИТ И ХРОНИЧЕСКИЙ ГЕНЕРАЛИЗОВАННЫЙ ПАРОДОНТИТ: ПРОИСХОЖДЕНИЕ, ПОСТЕПЕННЫЙ ПЕРЕХОД

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✓ ***Резюме***

В этой статье рассмотрены причины, условия и механизмы трансформации воспаления десны (гингивита) в воспаление тканей пародонта, характеризующееся прогрессирующей деструкцией периодонта и альвеолярной кости (пародонтит)

Ключевые слова: воспаление, патогенез, хронический генерализованный катаральный гингивит, хронический генерализованный пародонтит.

Relevance

According to domestic classifications of periodontal diseases from 1983 to 2001, two inflammatory forms of periodontal diseases are identified: gingivitis, which is the inflammation of the gums caused by unfavorable local and systemic factors, occurring without disruption of the attachment of the tooth to the gum and without destructive processes in other areas of the periodontium; and periodontitis,

characterized by inflammation of the periodontal tissues, which features progressive destruction of the periodontium and the alveolar bone. A crucial aspect of the oral cavity is that the various physiological and pathological processes occurring within it take place in the presence of microbes. The normal (healthy) state of the periodontium is maintained through the homeostasis of microorganisms forming a biofilm and cells that provide anti-infective immunity.

Disruption of this homeostasis leads to a breakdown of the mechanisms of immunological tolerance and, consequently, a weakening of local immune reactivity. The effect of bacterial influence under these conditions depends on the reactive processes within the body, which can either limit or promote destructive processes in the periodontal tissues.

Causes, Conditions, and Mechanisms of Transformation from Gingival Inflammation (Gingivitis) to Periodontal Tissue Inflammation Characterized by Progressive Destruction of the Periodontium and Alveolar Bone (Periodontitis)

Understanding the Mechanisms of Biofilm Formation in Chronic Inflammatory Periodontal Diseases an analysis of literature data and the observations of the staff from the Department of Therapeutic Dentistry at BSMI over the past decades regretfully reveals that even at the beginning of the 21st century, several unresolved issues persist in Uzbek periodontology. Notably, a convincing concept of the etiology and pathogenesis of major generalized inflammatory periodontal diseases, particularly concerning their initial stages (gingivitis and its transition to mild periodontitis), has not been established.

There is no comprehensive explanation of pathogenesis at the molecular-genetic level. Moreover, it remains unclear how and why oral microflora (dental plaque and biofilm), considered the "primary cause" of gingivitis, overcomes the natural mechanical, chemical, and biological barriers of the oral mucosa, gingival sulcus, and periodontium as a whole at the initial stage of disease development.

Factors that inhibit (counteract) the maturation of granulation tissue in the periodontal pocket—an outcome typically observed in other localizations of this pathological process—resulting in scar formation have also not been identified.

Unstudied Mechanisms Inhibiting Reparative Regeneration of Periodontal Complex Components Lost During the Pathological Process

The mechanisms that hinder the reparative regeneration of components of the periodontal complex lost during the described pathological process (particularly the periodontium and bone tissue) remain unexplored.

We believe that the pathogenesis of chronic generalized periodontitis (CGP) is determined by a complex of sequentially developing and/or simultaneously occurring (parallel) events within the periodontium. These events lead to the destruction of tissue components of one or more parts of the periodontium by various periodontal pathogens advancing in an apical direction. Thus, the active progression (and particularly exacerbation) of CGP is a discrete (i.e., intermittent, consisting of several phases) process amidst complex interactions between the human body and its "microflora."

In our view, the nature of these events is influenced by several factors:

1. The anatomical structure and functional characteristics of the periodontal complex and its constituent tissues;
2. The unique features of oral infections, particularly those associated with dental plaque (biofilm).
3. Immune Reactivity, Resistance, and Autoimmune Processes in the Pathogenesis of Chronic Generalized Periodontitis
4. Thirdly, the condition of immunobiological reactivity and resistance of the host organism as a whole, and of the periodontium in particular, plays a crucial role. Fourthly, the development of autoimmune processes, which may become a leading factor in the chronic inflammation of the periodontium, must also be considered.
5. In recent years, two increasingly distinct perspectives have emerged regarding the mechanism of development (i.e., pathogenesis) of chronic generalized periodontitis:
6. The existence of specific microbes that cause destructive damage to periodontal tissues;
7. The failure of the body’s protective mechanisms, leading to the development of periodontitis.
8. If one adheres solely to the microbial etiology of periodontitis (the "bacteriological etiologism"), as some authors suggest [14], it becomes clear that at least five conditions must be present and interact for this disease to develop:

9. The presence of periodontal pathogens in a quantity sufficient to initiate the process;
10. Environmental conditions in the niche (oral cavity) must favor the growth and reproduction of these bacteria;
11. Antagonistic microbes that counteract periodontal pathogens must be absent in the periodontal tissues;
12. The microbes must be spatially localized in such a way that they, and the products of their metabolism, can act on the target cells of the periodontal complex;
13. The human organism must be susceptible to the microbes and their metabolic products.

Modern Understanding of Periodontal Disease Pathogenesis and the Transition from Gingivitis to Periodontitis

According to current perspectives, most periodontal diseases are rooted in inflammation. Initially, this manifests as an acute, short-term reaction. However, as immune responses become involved and the process generalizes, the inflammation transitions into a chronic phase. Unfortunately, the current state of knowledge about the chronic nature of this pathological process, particularly in the periodontium, is notably insufficient.

As gingival inflammation progresses, the dentogingival junction is destroyed, periodontal pockets form, and resorptive changes occur in the alveolar bone. This leads to the disease acquiring the characteristic clinical and morphological features of chronic periodontitis of varying severity. The primary distinction between gingivitis and periodontitis, as noted by authors [5], lies in the location, severity, and intensity of the inflammation, as well as the extent of involvement of various periodontal structures.

Acute Periodontal Inflammation and Its Transition to ChronicForms
Acute forms of periodontal inflammation, such as acute gingivitis or pericoronitis, are rarely observed by clinicians. When the reparative phase of acute inflammation is inadequate, the condition becomes chronic, manifesting as dysregeneration. A prerequisite for chronic inflammation is the inability of acute inflammation to resolve through regeneration. This failure is influenced by the organism's response characteristics and the specific nature of pathogenic factors.

Dentists most commonly encounter chronic forms of inflammation in their practice, such as chronic generalized catarrhal gingivitis and periodontitis.

Rethinking the Role of Oral Microflora
In light of the above, the role of oral microflora in the development of generalized inflammatory periodontal diseases should not be viewed solely through an etiological lens but rather through the "pathogenetic involvement" of specific periodontal pathogens in the realization of certain stages of the inflammatory process.

Challenges in Etiology and Pathogenesis
Many dentists still acknowledge the unclear etiology of periodontal diseases. Instead of defining etiology, risk factors (RF) are often identified, or elements (links, aspects) of pathogenesis are listed. Etiology becomes evident only in cases of acute chemical, physical, or mechanical trauma, some iatrogenic injuries, and "man-made" lesions, which are typically localized to the gingiva. Regarding the pathogenesis of inflammatory periodontal diseases, opinions remain divided. However, there is consensus that these diseases develop only when the pathogenic factors' impact exceeds the protective and adaptive capabilities of the periodontal tissues and the host organism. The Multifactorial Nature of Chronic Generalized Periodontitis Development. In light of current knowledge, chronic generalized periodontitis (CGP) is considered a multifactorial disease, emphasizing the absence of a single etiological factor. Dental plaque is recognized as a necessary component in the development of periodontal diseases; however, its absolute role as the primary cause of periodontal inflammation can be questioned. Immune dysfunctions, active involvement of periodontal-pathogenic microflora, and microcirculatory disorders in the periodontium play significant roles in the pathogenesis of periodontitis.

The pathogenesis of CGP also involves neuroendocrine, metabolic, and immune mechanisms. Endogenous and exogenous factors, alongside immune defects, may significantly influence the formation and progression of the pathological process in the periodontium.

Interconnected Chronic Forms of Periodontal Pathology
Inflammatory periodontal diseases, such as gingivitis and periodontitis, are interconnected and typically chronic forms of pathology. The presence of bacteria is a necessary but not sufficient condition for the

progression from gingivitis to periodontitis. The likelihood of developing periodontitis and its clinical presentation (type and severity) are greatly influenced by host responses and additional risk factors such as smoking, stress, and comorbid conditions. These factors may be as important in the pathogenesis of these multifactorial diseases as bacteria themselves

From Gingivitis to Periodontitis: A Gradual Transition
Most authors agree that periodontal inflammatory diseases generally begin with gingival inflammation. It is noted that, in the context of clinically apparent chronic catarrhal gingivitis, morphological changes characteristic of chronic osteitis are observed. Over time, gingival inflammation evolves into a chronic condition, eventually taking on the hallmarks of periodontitis. Periodontitis is characterized by steadily progressive and wave-like destructive involvement of periodontal tissue structures, leading to resorptive changes in the bone. The gradual loss of alveolar bone tissue, a crucial component of the jaw, is the most typical and dramatic consequence for the dentoalveolar system and overall health.

Gingivitis and Periodontitis: Separate Nosological Units or Stages of a Unified Pathological Process?

On one hand, gingivitis is considered by some authors [11] to be an independent nosological unit within the inflammatory forms of periodontal disease. It is characterized by a localized pathological process confined to gingival tissues—this applies only when the gingiva is part of the periodontal structure. In the case of an edentulous ridge, the gingiva is viewed merely as a component of the oral cavity's soft tissue structures, and its damage in such cases is not associated with the periodontal tissue complex.

On the other hand, as summarized in one of the chapters of the manual by A.S. Grigoryan and O.A. Frolova (2004), it is stated that: "... contrary to the entrenched views in periodontology, despite the clinical 'heterogeneity' of gingivitis and periodontitis, we are, in fact, dealing with a single inflammatory periodontal disease, and each of these 'nosological' units represents only sequential stages of a unified pathological process." The deep interconnection and intertwining of the symptom complexes of these two nosological forms of periodontal pathology (gingivitis and periodontitis) lead to uncertainty regarding the boundaries that separate these diseases [4, 5].

Uncer tainty of Boundaries and Transitional States
This "uncertainty of boundaries" raises significant interest in the issues surrounding the transformation (transition or so-called borderline states) of acute inflammation of periodontal tissues into chronic forms. The concept of "borderline states," as a condition for professional risks in the diagnosis and treatment of dental diseases such as caries and its complications, was proposed by V.T. Shestakov [29]. We have previously examined this concept in the context of inflammatory periodontal diseases.

The understanding of these transitional states is critical for improving diagnostic accuracy and treatment strategies for periodontal pathologies, particularly in distinguishing between gingivitis and early stages of periodontitis.

Generalized Periodontitis and Catarrhal Gingivitis: Differences in Diagnosis and Stages of Periodontal Inflammation In the clinical guide for physicians , it is noted that:"... generalized periodontitis should be considered a disease, whereas catarrhal gingivitis in its acute phase, arising from poor oral hygiene and presenting as an acute, localized inflammatory reaction in the marginal gingiva, is a localized pathological process."

Key Features for Differential Diagnosis
According to A.S. Grigoryan [6], the fundamental criteria for differentiating generalized catarrhal gingivitis from generalized periodontitis include:

1. Destruction of the dentogingival junction with the formation of a periodontal pocket and the development of granulation tissue beneath its base, which plays a critical role as a destructive factor and represents one of the key elements in the pathogenesis of this form of pathology.
2. Destruction of the periodontal ligament.
3. Bone resorption**.**

Unfortunately, the early stages of periodontitis are difficult to clinically differentiate and are often misdiagnosed as generalized gingivitis by clinicians.

Stages of the Inflammatory Process in Periodontal Tissues
Based on the morphological data and clinical assessment of the periodontal condition, A.I. Grudyanov and E.V. Fomenko [7, 8] identified four stages in the dynamics of the inflammatory process in periodontal tissues:

1. Initial (subclinical) stage – corresponding to acute vasculitis.
2. Early damage stage – characterized by the appearance of dense small-cell infiltrates with a predominance of lymphocytes.
3. Established (progressive) stage – featuring signs of both chronic and acute inflammation.
4. Advanced inflammation stage – marked by pronounced chronicity of the process, vascular damage in the periodontium, destruction of collagenous tissue elements, and active bone resorption.

These stages underscore the progressive nature of periodontal disease and highlight the need for precise diagnostic methods to detect early pathological changes. According to G.F. Wolf et al. [3], the stage of 'pronounced gingivitis' can exist for several years without transforming into periodontitis. for several years, not passing into periodontitis, the transformation of which is due, on the one hand, to a change in the pathogenic potential of the 'dental' plaque (on the one hand on the one hand, changes in the pathogenic potential of the 'dental' plaque (biofilm), and, on the other hand, due to the inadequate response of the macroorganism to infection and exposure to risk factors. Virtually all researchers agree that periodontitis, as a rule, is accompanied by gingivitis, the degree of severity of which can vary widely. Proven pathogenetic comorbidity (commonality of many diseases of internal organs and periodontal disease) requires a unified approach. and periodontal diseases) requires unified interdisciplinary approaches to the treatment of such patients. In medical practice, a multitude of concepts are used to name several diseases occurring simultaneously in one patient simultaneously in one patient, many concepts and terms are used. One of such term is 'polypathology' (multiplicity of diseases). Polymorbidity is the presence of several diseases in one patient, whether related or unrelated to each other genetically or pathogenetically. genetically or pathogenetically. In this case, comorbid diseases are proposed to be considered, diseases that have common pathogenetic links, and co-morbid diseases that develop according to different pathogenetic scenarios pathogenetic scenarios. Multifactorial diseases - another term used to refer to multiple diseases in a single patient. They are caused by complex interactions between the organism at the genetic level and a multitude of unfavourable environmental and systemic factors. The development and ineffectiveness of treatment, especially in chronic generalised periodontitis is associated with the presence of modifiable (irrational nutrition, hypodynamia, smoking, dietary intake, hypodynamia, smoking, etc.).

Nutrition, hypodynamia, smoking, obesity) and non-modifiable (sex, age) risk factors., which, with proper and timely diagnosis and treatment of inflammatory periodontal diseases can increase the volume and quality of periodontal manipulations even in the current model of organising patients' dental care the current model of organising dental care for patients. At the beginning of the 21st century, it was suggested that the overall outcome of successful therapy for periodontal diseases should be the cessation of periodontal destruction periodontal diseases should be the cessation of periodontal destruction.

In our opinion, chronic generalised periodontitis is, firstly, multifactorial disease, arising under the influence of unfavourable cumulative influence of exogenous general and local factors (infectious agents of biofilm, anomalies of the frenulum attachment, filling defects, dentures, anomalies of tooth position, bite disorders) and endogenous influences (gastrointestinal diseases, hormonal disorders, blood diseases), infection-induced immune damage of the periodontal complex with a high probability of genetic predisposition; secondly, chronic generalised periodontitis is a disease resulting from a disturbance of the equilibrium (balance) between aggression factors (periodontopathogens) and defence factors of the macroorganism, mouth and periodontal complex, proceeding with initial lesion of the gingiva (gingivitis) and subsequent (and/or concurrently ongoing) involvement of other periodontal structures in the pathological process (periodontitis); thirdly, chronic generalised periodontitis is a disease characterised by progressive wave-like course (i.e., recurring in time and localization periods of exacerbations /not relapses!/ and remissions) with the outcome in bone resorption of the alveolar process, destruction of the tooth retention apparatus, formation of a periodontal pocket and ending with periodontal pocket and ending (as a rule, without timely and adequate treatment) with loss or extraction of the tooth loss or extraction, periodontal elimination and undoubtedly impaired function of the dento-mandibular system and the maxillary system function of the dento-mandibular system and the organism as a whole. One thing is clear: a wide nosological range, complexity of pathogenesis of the majority of periodontal diseases, in which there is involvement of the periodontal system and the organism as a whole.

The wide nosological range, complexity of the pathogenesis of most periodontal diseases, which involves infectious, immune, inflammatory, metabolic, psychoneuro-endocrine and other environmental

and genetic mechanisms, do not exclude the possibility of heterogeneous nature of diseases the possibility of heterogeneous nature of periodontal diseases (primarily periodontitis), require continuation, expansion and deepening of scientific research, development of complex programmes of prevention, treatment and rehabilitation prevention, treatment and rehabilitation programmes with the participation of specialists from different fields to solve the complex problems of modern periodontology.

Thus, the applied significance of the fundamentals of general human pathology requires concretisation in the due to the utilitarian and not always true nature of the theories (concepts, views) of the etiology and pathogenesis of inflammatory generalised periodontal diseases. Widespread of various infections of the oral cavity in persons without signs of periodontal pathology is a strong argument refuting the thesis about the leading role of bacterial infection in the occurrence and inflammatory generalised periodontal diseases. In patients with chronic generalised periodontitis in the mouth and periodontal pockets are found to have numerous types of microflora. It is known that by the density of microbial contamination of the the oral cavity ranks second after the large intestine. At the same time, it is surprising fact that hypercolonisation of the intestine by P. gingivalis (on average 2-fold) and T. denticola (on average 6-fold) is not associated with the risk of periodontitis. From this fact, these authors draw the following conclusion that the migration of periodontopathogens from the mucosa of one biotope to another does not play a role in the development of periodontitis a role in the development of periodontitis. Therefore, we can only speak about oral dysbacteriosis and its role in the development of inflammatory diseases of oral organs and tissues. And on the question ‘Infectious agents of the oral cavity: friend or foe?’ we can make a judgement that there is a a certain balance between the negative and positive effects of these infectious agents on humans beings. The defence mechanisms of the organism, oral cavity and periodontal complex, ‘designed’ to fight against damaging agents are simultaneously responsible for the disease progression, which leads to the destruction of their own tissues.

Thus, the question posed at the beginning of our article ‘chronic generalised generalised catarrhal gingivitis and chronic periodontal disease’ is answered catarrhal gingivitis and chronic generalised periodontitis: common origins and sequential transition? Can be answered as follows - these diseases have common origins, and in the absence of timely origins, and in the absence of timely adequate therapeutic interventions - and a sequential transition from one phase (phase) to another sequential transition from one phase (state) to another.

Conclusion

Based on the above-mentioned ideas about the pathogenesis of inflammatory periodontal diseases, ‘borderline states’ in periodontal pathology, the arguments in favour of the following become logical that treatment of patients with chronic generalised gingivitis and periodontitis, being by definition strictly individualised, becomes logical by definition strictly individualised, should, nevertheless, include a few mandatory, consistently, adequately and timely conducted stages of therapeutic, prophylactic and supportive interventions, which provide a comprehensive nature of therapy in these types of pathology nature of therapy in these types of pathology.

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