GOSPODARKA I INNOWACJE

LABORATORIUM WIEDZY Artur Borcuch

Volume: 24 | 2022

ISSN: 2545-0573

CURRENT LOOK AT CHRONIC GENERALIZED CATARRHAL GINGIVITIS

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ARTICLEINFO.

Key words:
Inflammation, pathogenesis, chronic generalized catarrhal gingivitis, chronic generalized periodontitis.

Annotation

Classification of periodontal diseases 1983-2001 [11], there are such inflammatory forms of periodontal diseases as gingivitis (gum inflammation caused by adverse effects of local and general factors, which occurs without violating the integrity of the gingival attachment and manifestations of destructive processes in other parts of the periodontium) and periodontitis (inflammation of periodontal tissues characterized by progressive destruction of the periodontium and alveolar bone). The most important feature of the oral cavity is that the various physiological and pathological processes occurring in it are carried out in the presence of microbes. A normal (healthy) periodontal condition is maintained due to the homeostasis of microorganisms that form a biofilm and cells that provide anti-infective immunity. Violation of this homeostasis leads to disruption of the mechanisms of immunological tolerance and, as a consequence, to a weakening of local immunoreactivity [9]. The effect of bacterial exposure under these conditions depends on reactive processes in the body, which can both limit and promote destructive processes in periodontal tissues. Apparently, this is due to the reactions of the body's defenses associated with the most complex system of immunogenesis and inflammation [17].

It is believed that inflammatory periodontal diseases also occur with the participation of representatives of the permanent microflora of the body, which are weakly pathogenic or complete saprophytes. The presence of more than 500-700 species of microbial strains in the oral cavity, the possibility of their combined action, and the variability of the saprophytic microflora composition present significant difficulties in assessing the role of bacteria as etiological and pathogenetic factors in the development and further progression of inflammatory-destructive periodontal diseases [5]. Many provisions of the "infectious origin" of inflammatory periodontal diseases are unsubstantiated and so far only accepted "on faith". As you know, "etiology answers the question of why, pathogenesis-the question of how the process develops" [10]. Currently, a vast amount of material has been accumulated on the pathogenesis of many diseases [8]. The aim of the study was to analyze the etiological and pathogenetic factors of

development and progression of chronic inflammatory generalized periodontal diseases: chronic catarrhal gingivitis and chronic generalized periodontitis. The causes, conditions and mechanisms of transformation of gingival inflammation (gingivitis) into periodontal tissue inflammation characterized by progressive destruction of periodontal and alveolar bone (periodontitis) are considered.

Thus, a convincing concept of etiology and pathogenesis has not been developed (the pathogenesis at the molecular and genetic level has not been deciphered) of the main generalized inflammatory periodontal diseases, especially in relation to their initial stages (gingivitis, transition /transformation/ of the latter into mild periodontitis). At the same time, it is not entirely clear how and why the microflora of the oral cavity (plaque, biofilm) (if it is considered the "root cause" of gingivitis) at the initial stage of the disease development overcomes the natural mechanical, chemical and biological barriers of the oral mucosa, gingival sulcus and periodontal as a whole. The factors that prevent (counteract) the maturation of granulation tissue in the periodontal pocket (which, as a rule, occurs in other localizations of this typical pathological process) with the formation of a scar have not been established. Mechanisms that inhibit the reparative regeneration of components of the periodontal complex (in particular, periodontal and bone tissue) lost during this pathological process has not been studied. We believe that the pathogenesis of chronic generalized periodontitis (CGP) is determined by a complex of events that develop sequentially and/or occur simultaneously (in parallel) in the periodontium, and consist in the destruction of a part of the tissue of one or several of its components by a variety of periodontal pathogens that"move" in the apical direction. Thus, an active course (and especially an exacerbation) CGP is a discrete (i.e. intermittent, consisting of several parts) event against the background of complex relationships between the human body and "its microflora". It seems to us that the nature of these events is due to a number of circumstances: first, the peculiarities of the anatomical structure and functions of the periodontal complex and its constituent tissues; second, the peculiarity of the oral cavity infection and especially the "dental plaque" (biofilm); third, the state of immune biological reactivity and resistance of the macro organism in general and periodontal in particular fourth, the development of autoimmune processes that may be a leading factor in the chronization of periodontal inflammation. Recently, two points of view regarding the mechanism of development (i.e. , pathogenesis) of chronic generalized periodontitis are becoming more and more clearly formed: 1) there are certain microbes that cause destructive damage to periodontal tissues; 2) a failure in the functioning of the body's defense mechanisms leads to the development of periodontitis. If we follow only the microbial etiology of periodontitis ("bacteriological etiology"), as some authors point out [14], then it is obvious that at least 5 conditions must occur and interact for the development of this disease: 1) the presence of periodontal pathogenic bacteria in an amount sufficient to start the process; 2) living conditions in the niche (oral cavity) should promote the growth and reproduction of bacteria; 3) periodontal tissues should be free of microbes-antagonists of periodontal pathogenic bacteria; 4) the microbe should be spatially localized so that it and its waste products could act on target cells of periodontal cancer. 5) the human body must be sensitive to microbes and their waste products. According to modern concepts, most periodontal diseases are based on inflammation - initially acute in the form of a short – term reaction, and as immune reactions are included in the pathogenesis and generalization of this process, the transition to the chronic phase occurs. Unfortunately, the current stage of the study of inflammation is characterized by a clearly insufficient knowledge of the nature of such a chronic pathological process, especially in periodontitis. With the further progression of gum inflammation, the destruction of the gingival junction, the formation of periodontal pockets, and resorptive changes in the alveolar bone occur. Thus, the disease acquires typical clinical and morphological features of chronic periodontitis of varying severity. The main difference between gingivitis and periodontitis, according to the authors [5], is the topic, severity and severity of inflammation, and the degree of involvement of various periodontal structures in it. Acute forms of periodontal inflammation (acute gingivitis, pericoronitis) are rarely observed by clinicians. When the reparative phase of acute inflammation is inadequate, it becomes chronic, which is a manifestation of dysregulation. A necessary prerequisite for chronic inflammation is the inability to complete acute inflammation with regeneration, which is due to the peculiarities of the

macroorganism's reaction and the specifics of pathogenic factors. With such chronic forms (chronic catarrhal generalized gingivitis and periodontitis) of inflammation, dentists often meet in their practice. In the light of the above, we can speak not only and not so much about the etiological role of oral microflora in the occurrence of generalized inflammatory periodontal diseases, but rather about the "pathogenetic involvement" of individual periodontal pathogens in the implementation of specific stages of development (i.e., pathogenesis) of periodontal inflammation. Many dentists are still forced to admit that the etiology of periodontal diseases is unclear. Often, risk factors (RF) are indicated instead of the etiology. In some cases, elements (links, aspects) of pathogenesis are listed. Evidence of the etiology is found only in acute chemical, physical, mechanical injuries, some iatrogenic and" man-made", usually localized, gum lesions. With regard to the pathogenesis of inflammatory periodontal diseases, there are still conflicting opinions with consistent judgments that the development of these diseases occurs only when the force of pathogenic factors exceeds the protective and adaptive capabilities of periodontal tissues and the human macro organism [18, 26].

Multifactorial nature of CVD development In the light of modern knowledge, chronic generalized periodontitis is considered as a multifactorial disease, thereby emphasizing the absence of a specific etiological factor. "Dental" plaque is considered a necessary component for the development of periodontal diseases, but its absolute role as the root cause of periodontal inflammation can be questioned [31]. And in the pathogenesis of periodontitis, immune disorders [18, 21,26], active participation of periodontal pathogenic microflora in the inflammatory process[26], and microcirculation disorders in the periodontium solve a lot. Neuroendocrine, metabolic, and immune mechanisms are also involved in the pathogenesis of CGP; however, endogenous and exogenous factors, along with immune defects, may play a role in the formation and nature of the course of the pathological process in periodontitis. Inflammatory periodontal diseases are interrelated, usually chronic forms of pathology (gingivitis and periodontitis). The presence of bacteria is a necessary, but not the only condition for the development of pathology(from gingivitis to periodontitis). The probability of developing periodontitis and its clinical course (type and severity) are significantly affected by the reactions of the macro organism and additional risk factors (smoking, stress, comorbid conditions, etc.), which may have the same significance in the pathogenesis of these multifactorial periodontal diseases as bacteria [3]. Currently, most authors [3, 5] recognize that inflammatory periodontal diseases, as a rule, begin with an inflammatory process in the gum. At the same time, it is emphasized that against the background of clinically defined chronic catarrhal gingivitis, a morphological picture characteristic of chronic ostitis is observed. The inflammation that develops in the gum eventually takes on the character of a chronic one, eventually acquiring features typical of periodontitis, with its steadily progressive and undulating destructive involvement in the process of periodontal tissue structures and the appearance of resorptive changes in bone tissue. The gradual disappearance of the bone substance of the alveolar process (part) of the jaw is the most typical and dramatic event in terms of consequences for the maxillary system and human health. The deep connection and mutual interweaving of the symptom complexes of these two nosological forms of periodontal pathology (gingivitis and periodontitis) determine the uncertainty of the bound ariesseparating these diseases [4, 5]. Obviously, with this "uncertaintyof borders", the transformation (transition or so-called borderline states) of acute periodontal tissue inflammation into chronic inflammation may deserve special attention. The concept of "borderline states" as a condition of occupational risks in the diagnosis and treatment of such dental diseases as caries and its complications was proposed by V. T. Shestakov [19,28]. We also considered it in one of our previous works in relation to inflammatory periodontal diseases [26]. The guide for physicians [7] notes that " ...generalized periodontitis should be considered as a disease, and ... catarrhal gingivitis in the acute phase, which has arisen due to violations of oral hygiene and proceeds as an acute inflammatory reaction limited to the marginal gum as a localized pathological process." The main features in the differential diagnosis of generalized catarrhal gingivitis and generalized periodontitis, according to A. S.Grigoryan [6], are: 1) destruction of the gingival joint with the formation of a periodontal pocket and

the development of granulation tissue under its bottom, which plays an important role as a destructive factor and is one of the leading links in the pathogenesis of this form of pathology; 2) destruction of the periodontal ligament; 3) bone resorption. Unfortunately, the initial phases of periodontitis are difficult to access for clinical differential diagnosis, and are evaluated by clinicians as generalized gingivitis. Dynamics of the inflammatory process in periodontal tissues, according to A. I.Grudyanova and E. V. Fomenko [7, 8], traced on the basis of comparison of morphological data and assessment of the clinical state of periodontitis, can be divided into four stages: 1) initial (preclinical) - corresponding to acute vasculitis; 2) early damage – the appearance of dense small-cell infiltrates with a predominance of lymphocytes; 3) established (progressive process) – with signs of both chronic and acute inflammation; 4) developed inflammation (with pronounced chronization of the process, damage to periodontal vessels, destruction of collagen tissue elements and active bone resorption).

Conclusion: Based on the above ideas about the pathogenesis of inflammatory periodontal diseases, "borderline states" in periodontal pathology, arguments also become logical in favor of the fact that the treatment of patients with chronic generalized gingivitis and periodontitis, being by definition strictly individualized, should nevertheless include several mandatory, consistently, adequately and timely stages of treatment, prevention and maintenance interventions that provide a comprehensive nature of therapy for these types of pathology.

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