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STRUCTURE AND FUNCTIONS OF PERIODONTAL TISSUES. CLASSIFICATION OF PERIODONTAL DISEASES

Jambilov Ravshanbek Sotvoldiyevich
Assistant of the Department of Therapeutic Dentistry
Andijan State Medical Institute

Annotation: The scientific data concerning a role of various factors of the oral cavity in formation of gingivitis and parodontitis are submitted. The results of a research devoted to studying of pathogenetic mechanisms of development of inflammatory pathology of the parodont are given. Etiopathogenetic aspects of the development of periodontal diseases can be used in the complex treatment of these types of pathology.

Keywords: Etiology, pathogenesis, gingivitis, parodontitis, dentistry, treatment.

Periodontal disease is a disease, or more likely a series of diseases, of the periodontal tissues that results in loss of attachment and destruction of alveolar bone. The natural history of periodontal disease results in tooth loss in some, but not all, patients.1 Periodontal disease, however, includes a spectrum of diseases broader than just periodontitis, and recognition of these diseases requires diagnosis.

In a healthy state, the periodontium performs a number of functions assigned to it:

• Support. The main function is due to which the tooth is held between bone plates.
• Shock-absorbing function. Correctly distributes pressure over the entire dentition.
• Trophic. A function responsible for nutrition and ensuring metabolism of the tissue complex.
• A protective function that helps create a barrier against the effects of bacteria.
• Reflex – affects the correct distribution of the chewing load.
• The plastic function is responsible for the elasticity of periodontal tissues.

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All tissues of the oral cavity, both teeth and gums, are interconnected and naturally have a complex structure. Gums, just like teeth, require careful and thorough care. Periodontics is a distinct branch of dentistry that deals with the study of gum disease. Inflammation from the teeth can spread to periodontal tissue, and vice versa. Therefore, it is so important to maintain careful hygiene of the entire oral cavity and promptly treat any dental diseases [1].

Periodontal disease has been known for at least 5,000 years. Clinicians have long recognized that there are distinct differences in the presentation of periodontal disease, and attempts have been made to classify these diseases. The established classification systems allowed doctors to structure information, allowing them to identify diseases by etiology, pathogenesis and treatment. This gives us the opportunity to organize effective treatment of our patients' diseases. Once the disease is diagnosed and classified, the etiology and appropriate evidence-based treatment are proposed to the clinician. A unified classification also allows healthcare professionals to communicate effectively using a common language. The first attempts at classification were based on the clinical signs of diseases and theories of their etiology. These attempts were not supported by any evidence. As scientific knowledge expanded, traditional pathology formed the basis of classification [2]. Later, classifications followed based on our knowledge of various periodontal infections and the body's response to them. However, the classification of periodontal diseases remained problematic. For much of the last century, physicians and researchers have faced challenges and have met from time to time to review and develop classifications of various forms of periodontal disease as research has expanded our knowledge of these diseases. This led to frequent revisions and changes [1, 2, 3].

Modern epidemiological data indicate that pathological changes occur as a result of poor oral hygiene, poor-quality dentures and fillings, dentoalveolar deformations, occlusal trauma, structural disorders of the tissues of the oral vestibule, medications used, and previous and concomitant diseases. Many researchers note a high level of prevalence of periodontal diseases among the population; with age, the frequency of lesions increases, along with this, the severity of the pathological process in periodontal tissues increases. Numerous theories of the etiology and pathogenesis of periodontal diseases can be divided into several groups: the inflammatory-dystrophic theory puts the toxic effect of dental plaque in the first place; metabolic-alimentary - nutritional and metabolic disorders; endocrine - hormonal disorders of regulatory processes in pathologies of the thyroid, parathyroid, pancreas and gonads; immunological - processes of autoallergy and autoimmunization, disorders of humoral and cellular immunity. For example, it has been established that immunological changes and autoallergization processes develop as destructive changes in periodontal tissues worsen. With developed forms of periodontitis, the bactericidal properties of blood serum and saliva decrease, and antibody titers to altered gum antigens increase. The level of these antibodies fluctuates depending on the severity of the disease [1, 2].

Currently, periodontal diseases represent one of the most common and complex pathologies in modern dentistry Approximately 50% of the population in various regions of the world aged 17-60 years has various forms of periodontal disease. Moreover, almost 90% of the population in developed countries have symptoms of gingivitis, 50% are diagnosed with moderate generalized periodontitis (GP), and 3% are diagnosed with severe. According to WHO (2005), functional disorders of the dental system caused by tooth loss from periodontal diseases develop 5 times more often than with complications of caries, and occupy the second place in prevalence among all dental diseases[1].

With developed forms of periodontitis, the bactericidal properties of blood serum and saliva decrease, and antibody titers to altered gum antigens increase. The level of these antibodies fluctuates depending on the severity of the disease. According to other authors, the allergic state of the body, including in periodontal tissues, is determined not by local causes, but by systemic disorders [2]. Researchers have established such disorders of the immune system in periodontitis as neutropenia, inhibition of the functional activity of T-lymphocytes, and an increase in their spontaneous blast transformation. Marked primary damage to polymorphonuclear leukocytes and lymphocytes during periodontitis [3, 4, 5, 6].

The current level of knowledge about the etiology of inflammatory periodontal diseases often identifies subgingival microflora as the dominant causative factor. For the occurrence of periodontitis, the most important factor is supra- and subgingival plaque. The latter is especially important, localized in the space of the periodontal sulcus and on the surface of the connective epithelium. The microbial flora in periodontitis is diverse and depends on the severity and phase of the disease. Despite the diversity of the microbial landscape, some types of microorganisms are recognized as specific periodontal pathogens. Combinations of these bacteria are found in areas of greatest destruction of periodontal tissue. These include:

- gram-negative anaerobic microorganisms of the bacteroid group, to a lesser extent anaerobiospirillum, spirochetes, fusobacteria;
- gram-positive anaerobic bacteria of the actinomycete group, to a lesser extent peptostreptococci [10, 11, 12, 13, 14, 15].

The high pathogenicity of the above bacteria is due to their virulence and characteristics of energy metabolism. Microorganisms living in the gingival sulcus and periodontal pockets have acquired highly developed defense mechanisms. Most periodontal pathogens have a high ability to attach and penetrate tissue. Their mechanism of invasion is similar to the mechanism of

invasion of enteropathogenic bacteria. This property is a significant factor in the etiopathogenesis of periodontal diseases. The ability of these microorganisms to adhere to the basement membrane is also a predisposing condition for the development of the inflammatory process [1].

It has been established that in case of ulcerative necrotizing gingivitis, juvenile periodontitis, and some adult periodontitis with electron microscopic examination, In a scientific study, a large number of anaerobic bacteria are found in periodontal tissues, although in most cases, plaque bacteria are not able to penetrate the epithelium and connective tissue. The development of the clinical picture of periodontitis is also associated with a progressive decrease in the content of neutral glycoprotein in the epithelium - one of the main components of the tissue barrier. The process of local loss of neutral glycoprotein in the surface layer is considered especially important, which can also be considered as a condition promoting the migration of oral microflora into the epithelial layer. The immunofluorescence method revealed that in acute and chronic periodontitis, focal alteration of the gingival basement membrane occurs, facilitating the penetration of microflora into deeper tissues. As a result, the epithelium of the gingival groove is destroyed, and collagenase and elastase produced by bacteria destroy connective tissue fibers, which leads to the development of an inflammatory-destructive process in periodontal tissues [7, 8, 9, 10].

Thus, therapeutic measures in the treatment of periodontitis should be aimed primarily at eliminating the microbial factor and reducing secondary tissue alteration.

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