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Etiopathogenesis of periodontal pocket (Literature review)

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Abstract: The leading factor for improving the management of the pathological process that occurs in periodontal diseases is the understanding of the pathogenesis of the disease. Pathogenesis implies processes that gradually lead to the development of the disease, violating the structure and function of the periodontal. (1) Periodontal diseases tend to develop under the combined influence of general and undoubtedly local factors against the background of reactivity of the body. (3) A pathognomonic sign of periodontitis is the presence of a periodontal pocket, which occurs as a result of atrophy of the gingival pocket and the alveolar process. This article presents an overview of the data of domestic and foreign literature, etiological factors leading to the formation of periodontal pocket, the mechanism of its development from the point of view of modern dentistry.

Keywords: periodontology, etiology, pathogenesis, periodontitis, periodontal pocket, microorganisms.

No matter how quickly modern dentistry develops, periodontal pathology remains one of the most urgent and complex problem. The pathognomonic symptom of periodontitis is the loss of attachment of periodontal tissues, loss of alveolar bone and the formation of pockets.

Microorganisms and environmental factors as the main etiological factors of the development of periodontal tissue disease.

One of the conducted experiments showed a causal relationship between the accumulation of bacterial deposits in the gingival sulcus and gingival inflammation (12). Young volunteers with healthy periodontium were instructed not to brush their teeth for a certain period of time. As soon as signs of inflammation of the gum and surrounding tissues began to appear in the oral cavity, patients were given detailed instructions on oral hygiene methods using a brush and wooden massage sticks. As a result of the lack of proper oral hygiene, bacteria multiplied and their waste products, the remains of accumulated food led to the formation of macroscopically visible deposits on the teeth. Within 9-21 days, clinical signs of gingivitis appeared. After carrying out professional oral hygiene, removing bacterial deposits and resuming brushing teeth, gingivitis subsided.

The composition of the microbial deposits was assessed microscopically(13): initially they consisted mainly of Gram-positive cocci and bacilli; later they also contained spindle and filamentous organisms; and a few days later spirochaetes were also detected.

Undoubtedly, in the formation of an inflammatory focus in periodontal tissues, the main role is played by such microorganisms as Actinobacillus, Actinomycetem

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comitans, Porphyromonas gingivalis, Bacteroidas forsythus, Campylobacter rectus, Eikenella corrodens, Streptococcus intermedius, Spirochetes, etc. (4,5,6) With classical bacterial infections, the diversity of the microbiota actually decreases as the disease develops, and therefore the probable pathogen is easily recognized, for example, Staphylococcus aureus or Pseudomonas aeruginosa with purulent infection. However, in most cases of periodontitis, the diversity of flora increases with the development of the disease.(15)

The local manifestation of signs of gum inflammation also depends on factors occurring in the host body that modulate the inflammatory response to microbial colonization. In addition, environmental factors influence inflammatory responses to microbial colonization. The amounts of interleukin-1beta, interleukin-4 and interleukin-8 were assessed in the gingival sulcus fluid of smokers and non-smokers with experimental gingivitis (14). Although there was no difference in plaque accumulation on day 10, clinical signs of gingivitis (gingival index and bleeding on probing) were significantly less pronounced in smokers than in non-smokers. Throughout the experiment, non-smokers showed a higher total amount of interleukin-4, but a lower amount of interleukin-8 than smokers. The total amount of interleukin-1beta and interleukin-8 increased significantly during plaque accumulation in both groups. The amount of interleukin-4 did not change in the group of smokers, but decreased in the group of non-smokers. In conclusion, it can be concluded that microorganisms, external factors and the state of the body as a whole play one of the main roles of the etiology of periodontal disease.

Etiopathogenesis of periodontal destruction leading to the formation of a periodontal pocket.

Proteolytic and hydrolytic enzymes of dental plaque have a destructive effect on periodontal tissues. The collagenase enzyme hydrolyzes the collagen of the periodontal ligament and bone tissue of the alveolar process. (4) Destruction of collagen and cement matrix fibers is also associated with direct phagocytosis of collagen fibers. (9)

In turn, the hyaluronidase produced by bacteria breaks down hyaluronic acid and, against the background of local hypoxia, fibroblasts, connective tissue epithelium are destroyed, blood vessel permeability increases, and leukocyte migration increases. (4,7) An increase in vascular permeability leads to an increase in the amount of gingival fluid and, as an immune response of the body, increases the flow of neutrophils and lymphocytes into the gingival sulcus to absorb bacteria. The basal cells of the epithelial tissue begin to divide to protect the intact barrier from bacteria and their toxins, as a result of which the epithelium begins to grow in areas where there is a lack of collagen. (8) The mechanisms of periodontal tissue destruction are also related to cytokines produced by normal cells of non-inflamed tissue.

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Clinically, as a result of edema, the gums look swollen, the gingival sulcus deepens slightly and becomes a favorable environment for the growth and development of the subgingival biofilm with its further apical proliferation. (8) During the loss of collagen, the apical epithelial cells begin to grow along the root and stretch out in the form of finger-like protrusions by 2-3 cells in thickness. (8)

A pocket is called a gingival sulcus, the depth of which has increased as a result of periodontal disease. (10) In turn, in the formation of a pathological pocket, an important role is given not only to the degeneration of the connective epithelium (since this will only worsen, and not stimulate the formation of a pocket), but also to the presence of healthy epithelial cells that stimulate the spread of connective tissue along the root, i.e. approaching the alveolar bone. (8) This, in turn, affects the hemostasis of bone tissue by activating the resorptive process.

The most severe degenerative changes in the periodontal pocket occur along the lateral wall, due to bulbous enlargements resulting from the accumulation of epithelial cells and their spread from the lateral wall of the pocket to the adjacent inflamed connective tissue. The severity of degenerative changes is not related to the depth of the pocket. There may be both deep pockets in which the lateral epithelium has a mild degree of degeneration, and shallow periodontal pockets with ulceration of the lateral wall.

The relationship of clinical and histopathological signs of periodontal pocket:

- the color change occurs due to stagnation and slowing of blood flow in the vessels - bluish-red;
- weak elasticity due to damage to the gingival fibers and surrounding tissues;
- swelling – as a result of infiltration of connective tissue by inflammatory infiltrate;
- shiny surface - due to edema and atrophy of the epithelium;
- bleeding - due to increased vascularization, thinning and degeneration of the epithelium, as well as the location of vessels near the surface;
- soreness of the inner part of the pocket during probing occurs as a result of ulceration of the inner surface of the pocket wall;
- discharge of pus with finger pressure can occur with purulent inflammation of the inner wall. (9,10)

Periodontal pockets, being the result of a chronic process, are constantly in the process of healing. But due to the presence and negative effects of bacterial flora, there is a constant degeneration of new tissue elements, which in turn are formed due to continuous recovery. (9)

Depending on whether destructive or constructive processes prevail in the tissues of the pocket, it is possible to determine the state of the soft tissues of the periodontium. Clinically, this will be expressed by edematous and fibrous forms, which in turn are determined by the color, consistency and relief of the pocket wall. The two forms presented above can constantly undergo transformations, depending

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on which processes will prevail in them. The edematous form is characterized by the presence of cellular exudate and inflammatory fluid, cyanotic-red color of the pocket wall, smooth, porous and shiny surface. The predominance of newly formed connective tissue cells and fibers, the manifestation of a pinker color of the pocket wall, the consistency of which is more solid, clinically this form is called fibrous. It should also be remembered that these two forms represent two extremes of the same disease. There may also be cases when the pocket looks pink and dense on the outside, but inside there is inflammation and ulceration.

The contents of the periodontal pocket are represented by microorganisms and their toxins and enzymes, food debris, saliva components, exfoliated epithelial cells, leukocytes. Over 180 strains of representatives of microbial flora are isolated and identified, the species composition is mainly represented by 9 species: the genus Streptococcus, the genus Staphilococcus, Corynebacteria and yeast-like fungi. (11)

Among streptococci, differences were found in the number of strains. Most of all were streptococci of S. mitis (31 strains — 17.22%) and S. mutans (30 strains — 16.67%), followed by S. agalactis (26 strains — 14.44%) and S. salivarius (23 strains - 12.78%), the least were representatives of S. sanguis (17 strains — 9.44 %). The genus Staphilococcus included 21 (11.67%) strains. Identification of their species was not carried out, they were limited only to the determination of the ability to coagulate the citrate plasma of a rabbit (coagulase-positive). This ability was possessed by 11 (6.11%) strains (coagulase-positive), 10 (5.56%) strains did not possess (coagulase-negative). Yeast-like fungi included 21 (11.67%) strains, Corynebacteria (gram-positive rods) - 11 (6.11%) strains.

In conclusion, it can also be added that the presence of periodontal pockets seeded with microorganisms that actively produce endotoxins that destroy nearby tissues and advance apically can cause atrophic and inflammatory diseases of the tooth pulp. Pocket infection can penetrate directly through the apical opening of the pocket, or through the lateral tubules of the tooth.

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