Inflammatory process in periodontal tissues. Periodontitis. Etiology and pathogenesis

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Abstract. The scientific article is devoted to a topic relevant to modern dentistry - the inflammatory process that occurs in the periodontal tissues, due to the high prevalence of pathology, the lack of a single concept of etiology and pathogenesis, adherence to prescribed therapy aimed at preventing and treating dental caries. Determination of the etiology and pathogenesis of such an inflammatory process as periodontitis plays a key role not only in the theoretical aspect, but also in the practice of a dentist. Due to the fact that the etiology and pathogenesis of inflammatory periodontal disease are not fully understood, I consider this topic to be significant for dentistry, healthcare and medical science.

Keywords: inflammation, periodontal pathology, periodontitis.

Introduction

Inflammation is a protective and adaptive reaction of the whole organism to the action of a pathogenic factor, the expression of which is tissue damage (alteration) - the initial phase of inflammation, impaired microcirculation with an increase in vascular permeability, exudation and emigration of leukocytes, proliferation (cell multiplication) - aimed at restoring damaged tissue [1].

Inflammation includes external signs and microstructural changes. The first include - systemic or local temperature rise, hyperemia, edema, dysfunction. The second group includes the exudative-vascular reaction and the reaction of the formation of a cellular infiltrate together with migration of leukocytes to the focus of inflammation [10].

Periodontal diseases, increasing the effectiveness of complex treatment is an urgent problem of modern dentistry due to the high prevalence of pathology, the lack of a single concept of etiology

and pathogenesis. Periodontal pathologies are not only a medical problem, but also a social one, since in the absence of modern diagnostics and treatment, it can lead to the emergence of foci of chronic infection in the oral cavity, tooth loss and, as a result, a decrease in working capacity - a violation of the function of chewing, speech and the emergence of aesthetic defect [4,6]. In severe cases, inflammation in the periodontium can spread to the periosteum, and then to the bone marrow of the alveolar process. Periostitis occurs, the development of osteomyelitis of the hole is possible. Regional lymph nodes may be involved. With purulent periodontitis of the teeth of the upper jaw, projected into the maxillary sinus, the inflammatory process can be complicated by purulent sinusitis [1].

Etiology and pathogenesis

The causes of inflammation are extremely varied. The causative agents of inflammation are pathogenic microorganisms, mainly streptococci, among which non-hemolytic streptococcus is 62%, Staphilococcus mitis - 26%, Staphilococcus salivarius - 12%. Causes are also physical factors (trauma), chemicals and adherence to prescribed therapy aimed at preventing and treating dental caries. (According to the WHO, dental caries is one of the six diseases of our time, occupies one of the leading places among somatic diseases, first among chronic diseases) [1,7].

The importance of infection in the development of periodontitis is very high, since it not only itself causes the development of inflammation, but also joins other pathogenic factors [7,8,10].

Depending on the localization of the "entrance gate" for microorganisms, there are odontogenic, stomatogenic, tonsillogenic, rhinogenic, otogenic, dermatogenic acute and chronic inflammatory processes of the maxillofacial region and neck.

In the overwhelming majority of cases, inflammatory processes are odontogenic, i.e. microorganisms enter the tissue through partially or completely necrotic pulp of the tooth, or through periodontal pockets.

The pathogenesis of the disease includes a nonspecific inflammatory response [1]. The inflammatory process begins with the penetration of the contents of the root canal beyond the root apex into the periodontal tissue of lipopolysaccharides, which are products of pathogenic microorganisms. Further development of the process depends on the state of the immune mechanisms. With a pronounced protective reaction, the process is localized at the apex of the root and becomes chronic [5,6,10]. If the defense mechanisms are relaxed, then acute diffuse inflammation develops [4,7].

Lipopolysaccharides carry out polyclonal immunostimulation, cause chemotaxis of leukocytes, release of histamine, serotonin, increase the activity of macrophages and their synthesis of inflammatory mediators, such as prostaglandin E, E2, interleukins (IL-1, IL-6, IL-8), tumor necrosis factor-alpha, which, in turn, contribute to the development of microcirculatory disorders in the periodontal tissues (vascular permeability increases, edema develops), the destruction of collagen and non-collagen proteins under the action of lysosomal hydrolases (collagenase, elastase), periodontal ligaments (under the action of lymphokines), a decrease in the content of proteoglycans (play an important role in the formation of the periodontal attachment), bone resorption of the alveolar processes of the jaws [6].

Conclusion

Thus, inflammation is a reaction developed in the course of phylogenesis, has a protective and adaptive character and carries elements of not only pathology and physiology. The spread of irritating factors from the root canal to the periodontium leads, as a rule, to chronic long-term inflammation. Determination of the etiology and pathogenesis of such an inflammatory process as periodontitis plays a key role not only in the theoretical aspect, but also in the practice of a dentist.

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