Modern concepts of etiology and pathogenesis of inflammatory periodontal diseases

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Abstract
This article presents recent evidence of early regeneration processes in the course of periodontal diseases with immunological products and their significance in the development and progression of childhood systemic diseases, pathology, internal organs, indicating the relationship of the latter with gingivitis and periodontitis. The important role of preventing bacterial infection mouth from a position of the cytokine theory of periodontal disease.

Keywords: Microorganisms, periodontal disease, dental plaque, regeneration

1. Introduction
Analysis of domestic and foreign literature shows that the main clinical forms of periodontal disease is gingivitis, periodontitis and periodontal disease. In the development of periodontal disease we can underline three main directions, based on the pathological process in periodontal tissues.
The first direction is based on the fact that various clinical manifestations is a single pathological process – dystrophy of periodontal tissues, which gradually produce to resorption of the alveolar process, the formation of clinical pockets, their purulence, and finally the loss of teeth [1, 6].
The second direction is based on two different pathological processes such as: symptomatic lesions of periodontium as a result of internal and body systems (periodontal syndrome) and real (initial) periodontitis in healthy people.
The third direction is based on identification the presence in periodontium different pathological processes the processes which are accompanied by inflammatory, dystrophic and tumor modifications (changes). It includes all diseases that occur both separate periodontal tissues and functional tissues complex, no matter if it develops under the influence of local or general causes [8].

2. The purpose of the study was to conduct a modern review of the etiology and pathogenesis of infiltration periodontal diseases.

3. Results and their discussion. In modern periodontology we can count several dozen classifications of periodontal diseases, some are explained not only by diversity of periodontal pathology, but mainly on the principles and character of its systematization [5].
Most foreign scholars [1, 4, 5] believe that the local factors are principal in the etiology and pathogenesis of periodontal diseases, connecting its development with the effect of dental plaque. According to the concept that the oral cavity is seen as a balanced biological system, and periodontal disease in mostly the result of imbalance between bacterial symbiosis and the oral cavity tissues. According to these facts, the key of Etiological Studying of periodontal tissue diseases is that there are three groups of factors such as: the state and products of metabolism in dental plaque and calculus; oral factors that can strengthen or weaken the potential of pathogenic microorganisms and products of their metabolism; general factors that regulate the metabolism of oral tissues which depend on the response to pathogenic influences.
Long-term contact between the microorganisms of dental plaque and periodontal tissues causes of autoimmune processes that can cause the chain reaction that is accompanied by progressive changes in periodontal tissues. Due to the well-studied microbe flora of periodontal pockets,
the possibility of microbe allergization of the body and the development of autoimmune reactions.

Dental plaque is a complex of proteins, which include almost all representatives of the microbial flora of the mouth. In oral fluid there are from 4mln to 5 billion. Ml., and in Ig. plaque – there are from 10 to 100 billion microorganisms.

According to scientist’s learning, there are 500 species of microorganisms, all of which can be a source of infection. However, the results of recent studies prove that the clinical picture of periodontitis is not always correlated with the degree of infection and composition of bacterial plaque.

Initially, the anaerobic microflora prevails. This stage of the disease is characterized by a protective response systems - increased proliferation and desquamation of the epithelium, the influx of leukocytes to inflammation and active salivation.

Later, as the author mentioned, a long course of inflammation will reduce protection mechanisms at the level of the mucous membrane, accompanied by a change in the microbial landscape towards conditional - pathogenic streptococci. The presence of associative flora contributes to chronic inflammatory diseases with periodic exacerbations [3, 7].

Bacteria entering the cell, secrete toxins, enzymes that destroy intracellular structure. These enzymes belong phosphatas, aminopeptidaza, phospholipase, protease, glycosides, hyaluronidase, hondoitysulfataza, fibrinolyzin. In addition, microorganisms secrete cytotoxic metabolic products: ammonium sulfate, hydrogen, indole, butyrate, propionate, fat, toxic acid. The number of works that demonstrate the leading role of microflora in the development of inflammation in periodont is growing up sharply. However, some researchers have doubts about the role of microbial factors in periodontal destruction [2]. Also there are discussions that gingivitis can occur in the absence of dental plaque. In other works tartar plays secondary role and was proposed to study the dependence of hemodynamic microflora in periodontal pockets.

Assessing the role of plaque, many researchers, especially foreigners, believe that all the other local and general factors only exacerbate its effects on periodont but have no great influence on it. Therefore, prevention of periodontal disease depends on the culture and efficiency of patient oral hygiene.

While the comparative analysis of the treatment of periodontal tissue in 211 patients, which were observed for 22 years, according to the authors research demonstrated that therapy of patients cannot be replaced by a briefing on oral hygiene. Other researchers also did not notice a significant dependence on good oral hygiene status prevalence of gingivitis. However, this trend (especially abroad), is very popular and practical results of numerous studies reflected in the large arsenal of drug and non-drug inhibition of microbial plaque and the prevention or treatment of inflammatory changes. But it remains paradoxical fact that scientifically based and effective in controlling long-term experimental and clinical studies prevention methods do not give practical effect, confirmed by epidemiological study periodontal status in different countries.

Many researchers [2, 4, 7] argue that the basics for the development of inflammatory periodontal tissue diseases are genetic and functional factors and lower overall non-specific immunological reactivity and resistance of periodontal tissues. Thus, the leading juvenile periodontal etiological factor is heredity. Based on examination they concluded that this disease can be transmitted autosomal recessive way. As the result of the genetic causes a tendency to produce immune to certain groups of bacteria or the formation of pathological periodontal tissues. Hereditary predisposition to certain periodontal disease manifests itself dominant type when symptoms occur in every generation. Some scientists say the possibility of genetic susceptibility to periodontal pathology, it does not affirming. The main role is played by hereditary differences metabolism and tissue differentiation, as evidenced by biochemical research at the molecular level. Actually it is probably related to the pathology of bite, which has an important role in the development of periodontal disease. During the examining of 80 twins, the scientists have established rigid genetic determination of deep incisive overlap in times of temporary and permanent occlusion, and during AC - prevailing influence of environmental factors. Hereditary predisposition to the development of periodontal disease should be subject to different study, as well as clarify the place of genetic factors in the pathogenesis, open new opportunities in early diagnosis, prevention and treatment.

Generalized periodontitis is a polyetiological disease, associated with the pathology of internal organs. Analyzing the role of transferred and related diseases, hormonal disorders, we should notice that they reduce local resistance barrier periodontal autosensibilization contribute to the development of mechanisms and immunopathological process of bone resorption of alveolar processes of jaws [3]. The changes of reactivity and the decrease of resistance periodontal tissue and the appearance of catarhal gingivitis may happen in some cardio - vascular and gastro - intestinal diseases, hormonal disorders, infectious diseases, radiation damage, collagenesis, dysfunction of the hypophys, thyroid and sexual glands and others.

The other authors state the close relationship between the various pathologies of internal organs and the state of periodontal tissue in 97% of patients with generalized periodontitis [5].

Relating periodontitis with neurogenic nature, the violation of periodontal trophic is explained like vasomotor functions and it is trophic disorders. Periodontal tissues are characterized by a high sensitivity to stress, manifested as destruction of gum, violation of hemocirculation, increased alveolar bone resorption, structural infringement of cells and intercellular substance. It was found that during stress it increases infectious as a result of immunological conflict in periodontal tissues.

In 42% of patients with destructive inflammatory periodontal pathology have problems with gastro - intestinal tract. In studying the prevalence of periodontal disease in patients with peptic ulcer and chronic gastritis, authors determined that gingivitis and periodontitis appear more often (87.6%) than in healthy people (49.2%). More often gingivitis and periodontitis were observed in patients with hypo and anatsyd gastritis (92.5%). According to the anamnesis, it takes (84.8%), of patients, gastrointestinal disease has a prior signs of periodontal pathology. However, according to some authors’ theories, the disease of liver, stomach, may affect the periodontal tissues, but not be causal factors. According to that theories, the destruction of internal organs affecting the phylogenetically younger periodontal tissue complex on humoral and visceral ways [2, 6].

Some patients determine the inverse relationship between diseases of the mouth and whole body. Many authors wrote a lot articles about the role of focal pathology of teeth-jaw system in causing diseases of internal organs. Some scientists believe that the constant presence of
plaque Helicobacter pilori, can cause peptic ulcer as stomach cancer. The authors have established a high incidence of chronic catarrhal gingivitis, the tendency to generalization of the inflammatory process in children 5 - 14 years with gastroduodenal pathology associated with Helicobacter pilori. A large group of researchers [1, 2, 4, 8] undertook a study of the relationship between periodontal diseases and endocrine diseases that are closely associated with hypoo and hyperfunction of thyroid, sexual and parathyroid glands. Pathological changes in periodontal tissues of people who suffered from diabetes occur very often, according to different authors there are 45 - 87% in children and 75 - 93% in adults. With increasing duration of diabetes, increases the frequency and severity of periodontal lesions.

At the same time, the researchers 5, 6 attaching great importance to hypoxia, which leads to a reduction of ascorbic acid in the blood and tissues. Based on the results reoparodontography, definition and partial pressure of carbon dioxide in the peripheral blood of patients with gum cardio - vascular pathology, write about fairly rapid development in their periodontal tissue and circular signs of hypoxia, which causes more rapid progression of periodontal tissue destruction.

Inflammatory - destructive periodontal changes associated with dysfunction of microcirculation. The impact of atherosclerotic vascular lesions within the vascular concept formulated by AI Evdokimov from 1939 to 1940. Based on a large number (10 930 people) clinical and radiological examination of the population aged from 15 to 60 years with various types of arteriosclerosis and cardio - vascular diseases, and in combination with diabetes hypothyroid diseases, obesity and inflammatory periodontal disease was found in 73.8%.

The high percentage of periodontal tissue diseases under the occupational hazard, indicate a number of authors, noting not only their degree, but the duration of action. You may argue that the approach to the study of periodontal disease in terms of medical ecology and identifying links with environmental factors.

The increase of frequency of periodontal disease connected with increasing of age, explaining the age characteristics of immune and skeletal systems, leading the main role to aging tissues and collagen vascular changes, the amount of which decreases sharply.

It is worth drawing attention to parafunctions of teeth-jaw system (without consciously and not intentional aging teeth gnashing, biting lips and nails), which cause degenerative changes in the periodontal tissues. Underlying parafunction, in their opinion, are psychological phenomena and psychological character, breach of occlusion, discomfort in the presence of prostheses. The diversity of the clinical picture of parafunction masticatory muscles depends on local factors and general (background) changes in the body. According to that fact the disease does not begin with gingivitis and changes of bone alveolar processes under the influence of traumatic or endogenous factors. From classification of parafunction most are focuses on violations of the act of swallowing, which may be the only reason for the nomination forward front teeth, especially lower teeth, that leading to disruption of articulation, congestion and development of periodontal disease process. Among the various pathologies of bite based on author’s facts, occupies a special place overbite, which fluttering pathological changes in periodontal front teeth of the upper and lower jaws.

The factors that cause periodontal disease include functional chewing intact teeth. Functional problems is a consequence of modern civilization. Scientists believe that chewing load reduction occurs in ontogeny at artificial feeding and pathogenesis sufficiently studied with hypokinesia and physical inactivity due to Space medicine. Insufficient of chewing function reduces periodontal tissue resistance to external factors (microbes injury) and facilitates deferral of tartar, and therefore the development of inflammatory destructive phenomenon.

Without denying the role of disorder function of the nervous system, hormonal and immune systems, genetic predisposition it should be underlined the importance of local mechanical factors. The last one, according to the authors, there is at higher threshold "mechanical immunity" acquired in the evolution of tissues that amortize chewing effort. Overloading periodontal tissue is always accompanied by changes in hydrostatic pressure, blood - and lymph flow and consequently, the emergence of hemo and lymphostasis, violation microanatomy permeability barrier, perivascular edema, diapedesis of formed elements cause tooth mobility and the pain when person bites down.

Conclusion
Epidemiological studies show a steadily high prevalence of periodontal disease among young people. According to the researches of most authors, gingivitis is the most common form of pathology of periodontal tissues in adolescents and children. But later, the frequency of more severe pathological processes such a periodontitis and the number of removed teeth significantly increases. In the processing of numerous works about the leading role of local factors in the development of periodontal tissue diseases can note their conflict and the increasing need for an explanation for certain provisions of endogenous factors. During learning the pathogenic mechanisms of inflammatory periodontal diseases, many hypotheses were formed, the most important of all are immunologic, neurogenic and vascular.

There is only one drawback in the study of periodontal disease. Many researchers try to explain the origin of the pathological process by one reason. However, the pathogenesis and diagnostics of periodontal diseases should have a systematic approach, the content of which is that each component of the biological system of the body could not be interpreted as a separate entity.

References
