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©O. A. Bedenyuk, T. I. Dzetsiukh, O. S. Bedenyuk, A. B. Vorobets

I. Horbachevsky Ternopil National Medical University
e-mail: bedenyukoa@tdmu.edu.ua

Histological changes in the structural components of the gums in lipopolysaccharide periodontitis

ІНФОРМАЦІЯ

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Key words: periodontitis; atrophic gastritis; morphological changes.

АНОТАЦІЯ

Summary. Inflammatory diseases of periodontal tissues are one of the most important problems in dentistry. There is a strong connection between periodontal pathology and inflammatory diseases of the stomach.
The aim of the study – to investigate pathomorphological changes in rat periodontal tissues with generalized lipopolysaccharide periodontitis associated with chronic atrophic gastritis.
Materials and Methods. The experiments were performed on white outbred male rats weighing 160–180 g. 30 animals were used during the work. The tested animals were divided into the following groups: I – intact rats (control); II – animals with a model of generalized lipopolysaccharide periodontitis. For a morphological study of the periodontium, a fragment of the upper jaw was isolated. Histological preparations were studied using an SEOSCAN light microscope. Such classical research methods make it possible to study the structure of tissues, as well as the nature and depth of morphological changes, the sequence of development of destructive and regenerative processes.
Results and Discussion. It has been established that with generalized lipopolysaccharide periodontitis, a reorganization of all structural components of the gum tissue occurs. It is manifested by changes in the epithelium of the mucous membrane of the gums, an increase in the stratum corneum, a decrease in the spinous layer, expansion of intercellular spaces, growth of lymphocytic infiltration, swelling of the connective tissue of the mucous membrane, expansion, blood filling of veins and hemocapillaries, cells of the walls of fibrous structures.
Conclusions. Concomitant chronic atrophic gastritis significantly aggravates the pathomorphological picture of periodontitis induced in animals by the endotoxin of gram-negative microflora lipopolysaccharide.

Introduction. Inflammatory diseases of periodontal tissues today are perhaps the most important problem in dentistry, which has not only medical, but also enormous social significance, which is due to the widespread prevalence of periodontitis, damage to young people, the possibility of periodontitis influencing the occurrence of somatic pathology, and the lack of effective methods of diagnosis and prevention and treatment [1, 2, 3]. The prevalence of periodontitis in different countries of the world is 50–90 %; Ukraine is also one of the countries with a significant prevalence of periodontal diseases – depending on the region and age of those examined, it reaches 85–95 % [4, 5].

Since the periodontium is considered as an integral component of the whole organism, an important factor determining the severity and prognosis of generalized periodontitis is the presence of con-

comitant somatic pathology. Epidemiological studies clearly confirm that diabetes mellitus, hypertension, septic endocarditis, urolithiasis, hepatitis and a number of other diseases are combined with periodontal damage with absolute regularity [6, 7]. A strong connection has been noted between periodontal pathology and inflammatory diseases of the stomach and intestines. Among patients with diseases of the digestive system, pathological changes in periodontal tissue occur in more than 90 % of cases [8, 9]. The features of the molecular mechanisms underlying the influence of gastric diseases on the pathogenesis of periodontitis are currently insufficiently illuminated and require more detailed study. The remaining pathomorphological mechanisms of the development of periodontitis associated with chronic atrophic gastritis induced by endotoxin of gram-negative microflora lipopolysaccharide have not been studied [10, 11].

The aim of the study – to investigate pathomorphological changes in rat periodontal tissues with generalized lipopolysaccharide periodontitis associated with chronic atrophic gastritis.

Materials and Methods. The experiments were performed on white outbred male rats weighing 160–180 g. 30 animals were used during the work. The animals were kept on a standard vivarium diet in accordance with sanitary and hygienic standards. All stages of the experiments were carried out in accordance with the International Requirements for the Humane Treatment of Animals in accordance with the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986).

The tested animals were divided into the following groups: I – intact rats (control); II – animals with a model of generalized lipopolysaccharide periodontitis. Rats in this group were injected into the gum tissue with E. Coli lipopolysaccharide (LPS) (Sigma-Aldrich, USA). LPS was diluted in sterile saline and administered using an induction syringe at a dose of 40 microliters (1 mg/ml) every other day for 2 weeks; III – rats with lipopolysaccharide periodontitis against the background of chronic atrophic gastritis. Atrophic gastritis was induced in animals of this group by intragastric administration of 2 % sodium salicylate for 6 weeks. Drinking water was replaced with 20 mM sodium deoxycholate. The development of atrophic gastritis was confirmed by histological studies. Starting from the 43rd day after the administration of sodium salicylate, animals were injected with LPS into the gum tissue for 2 weeks according to the method described above.

The rats were decapitated under thiopental anesthesia the day after the last injection of LPS. For a morphological study of the periodontium, a fragment of the upper jaw was isolated. Pieces of periodontal tissue fragments were fixed in a 10 % solution of buffered neutral formalin for two weeks with three changes in the fixing solution. Formalin solution was prepared immediately before use. After fixation in formaldehyde, decalcification of the jaw tissue was performed. Then the material was washed in running water, dehydrated in ethyl alcohol solutions of increasing concentrations and compacted with paraffin. The preparations were impregnated with paraffin at a temperature of 56°C for 2 hours. The production of serial paraffin sections with a thickness of 4–6 microns was carried out on an MS-1 sled microtome. The preparations were stained with hematoxylin and eosin. Histological preparations were studied using an SEOSCAN light microscope. Such classical research methods make it possible to study the structure of tissues, as well as the nature and depth of morphological changes, the sequence of development of destructive and regenerative processes.

Results and Discussion. Microscopic studies of rat gums with periodontitis have revealed pronounced changes in all its structural components. There was a significant thickening of the stratum corneum of the epithelial plate, especially in the free area of the gums. Existing areas of contact disruption between horny scales. This indicates the phenomena of hyperkeratosis in the epithelium of the free part of the rat gums in response to the action of a damaging factor.

There is also a decrease in the thickness of the spinous layer of this area of the gums. In many epithelial cells, the karyoplasm of the nuclei is optically transparent, so they appear vacuole-shaped. In the epithelial cells of the granular layer, there are many basophilic keratohyalin granules in the cytoplasm.

Also microscopically, a decrease in the thickness of the epithelial plate of the gum mucosa in the areas of the groove and attachment is observed. Unlike rats of the intact group, in the epithelial lamina, the furrow nuclei of the cells of the spinous layer are small in size and have basophilic karyoplasm, which indicates their pyknotic changes. Lymphocytes, single macrophages and neutrophilic granulocytes are observed in the expanded intercellular spaces. Mitotic division of epithelial cells of the basal layer occurs rarely.

In the lamina propria of the gum mucosa, disorders of the connective tissue structure and vascu-

lar disorders are observed. Existing swelling of the amorphous component of the intercellular substance, destructive changes in fibers, manifested by disorganization and thinning of collagen fibers. The mesh layer of the connective tissue of the gums is made up of compacted bundles of collagen fibers, which have an intense oxyphilic color, and between the bundles there are light, irregularly shaped areas of an amorphous component, which indicates edema. Damage to fibroblasts and the presence of fibrocytes are noted. Leukocyte infiltration increases, especially in areas of connective tissue and the attachment of the groove.

Significant changes are revealed in the components of the microvasculature of the gingival lamina propria. Arterioles are characterized by a significant reduction in lumen areas, wall thickening, and perivascular edema. The lumens of the venules and capillaries increase, they are filled with blood, their walls are graceful. Our results are confirmed by the work of scientists [8, 9, 11]

Small focal infiltrates are found in the perivascular spaces. Near the wall of the hemocapillaries, tissue basophils are observed - mast cells with signs of degranulation. In the epithelium of the free part of the gums and gingival sulcus, an increase in the number of intraepithelial lymphocytes is noted.

Conclusions. Histological studies of the gums of animals with experimental periodontitis established the reorganization of all its structural components in comparison with animals of the control group. It is manifested by changes in the epithelium of the gum mucosa, both free and areas of the groove and attachment, an increase in the stratum corneum, a decrease in the spinous layer, expansion of the intercellular spaces, an increase in lymphocytic infiltration, and swelling of the connective tissue of the mucous membrane. The reaction of the vascular bed to simulated periodontitis is manifested by expansion, blood filling of veins and hemocapillaries and narrowing and thickening of the arterial wall. The connective tissue of the mucosa is characterized by swelling of the amorphous substance and damage to the fibrous structures.

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Тернопільський національний медичний університет імені І. Я. Горбачевського МОЗ України

Гістологічні зміни структурних компонентів ясен при ліпополісахаридному пародонтиті

Резюме. Запальні захворювання тканин пародонта є однією з найважливіших проблем у стоматології. Існує стійкий зв'язок між патологією пародонта і запальними захворюваннями шлунка.
Мета дослідження – вивчити патоморфологічні зміни у тканинах пародонта щурів із генералізованим ліпополісахаридним пародонтитом, асоційованим з хронічним атрофічним гастритом.
Матеріали і методи. Досліди виконані на білих безпородних щурах-самцях масою тіла 160 г. У процесі роботи використано 30 тварин. Дослідних тварин поділили на групи: перша – інтактні щури (контроль); друга – тварини з моделлю генералізованого ліпополісахаридного пародонтиту. Для морфологічного дослідження пародонта виділяли фрагмент верхньої щелепи. Гістологічні препарати вивчали за допомогою світлового мікроскопа SEOSCAN та фотодокументували за допомогою відеокамери Vision CCD Camera. Такі класичні методи досліджень дають можливість вивчити структуру тканин, а також характер і глибину морфологічних змін, послідовність розвитку деструктивних та регенераторних процесів.
Результати досліджень та їх обговорення. Встановлено, що при генералізованому ліпополісахаридному пародонтиті відбувається реорганізація усіх структурних компонентів тканин ясен. Вона проявляється змінами епітелію в слизовій оболонці ясен, збільшенням зроговілого шару, зменшенням остистого шару, розширенням міжклітинних просторів, зростанням лімфоцитарної інфільтрації, набряком сполучної тканини слизової оболонки, розширенням, кровонаповненням вен і гемокапілярів, потовщенням стінки артерій, набряком аморфної речовини сполучної тканини й ушкодженням її волокнистих структур.
Висновки. Супутній хронічний атрофічний гастрит суттєво погіршує патоморфологічну картину пародонтиту, індукованого у тварин ендотоксином грамнегативної мікрофлори ліпополісахаридом.

Ключові слова: пародонтит; атрофічний гастрит; морфологічні зміни.

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