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ПАТОМОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ В ПАРОДОНТАЛЬНОМ КОМПЛЕКСЕ ПРИ ПАРОДОНТИТЕ ЛИПОПОЛИСАХАРИДНОГО ГЕНЕЗА

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Резюме. Целью нашего исследования было выяснение патоморфологических особенностей поражения в пародонтальном комплексе у крыс липополисахаридным эндотоксином грамотрицательной микрофлоры. Эксперименты проведены на белых крысах-самцах. Животным для моделирования пародонтита вводили липополисахарид E. Coli, готовили гистологические срезы, которые после предварительно проведенной декальцинации окрашивали гематоксилин-эозином. Забор материала для электронно-микроскопических исследований проводили за общепринятой методикой. Ультратонкие срезы, изготовленные на ультрамикротоме LKB-3, окрашивали 1% водным раствором уранилацетата, контрастировали цитратом свинца по методу Рейнольдса и изучали в электронном микроскопе. Проведенные гистологические исследования обнаружили выраженные изменения воспалительного характера всех составляющих компонентов. Микроскопически в эпителиальной пластинке обнаруживались признаки гиперкератоза десны, базальный слой содержал преимущественно пикнотические клетки, редко оказывались фигуры митоза. Уменьшалась толщина остистого слоя. Отмечалось истончение эпителиальной пластинки в участках борозды и прикрепления. Нарушение эпителиального прикрепления и волокон циркулярной связи, привело к образованию пародонтального кармана, в которой оказывались детритные массы, десквамированные эпителиоциты и лейкоциты. Выявлялось нарушение минерализации зубной альвеолы, очагово определялась лакунарная резорбция. Надкостница была гиперплазированна с пролиферацией молодых остеобластов. Субмикроскопические изучения структурных компонентов периодонта зуба животных при экспериментальном пародонтите также выявило нарастание деструктивных изменений. Определялись зоны расслоения коллагеновых волокон, отек аморфного компонента рыхлой соединительной ткани. Вокруг фиброцитов локализовались фрагментированные пучки коллагеновых волокон и аморфное межклеточное вещество соединительной ткани.

Ключевые слова: пародонт, липополисахарид, воспаление, периодонтит, эпителий.

Abstract.

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PATHOMORPHOLOGICAL CHANGES IN THE PERIODONTAL COMPLEX WITH PERIODONTITIS OF LIPOPOLYSACCHARIDE GENESIS

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The purpose of our study was to elucidate the pathomorphological features of lesions in the rat's periodontal complex with lipopolysaccharide endotoxin of gram-negative microflora. The experiments were carried out on male white rats. To simulate periodontitis, the animals were injected with E. Coli lipopolysaccharide, prepared histological sections, which, after preliminary decalcification, were stained with hematoxylin-eosin. The sampling of material for electron microscopic studies was carried out according to the generally accepted technique. Ultrathin sections made on an LKB-3 ultramicrotome were stained with a 1% aqueous solution of uranyl acetate, contrasted with lead citrate according to the Reynolds method, and studied under an electron microscope. The histological studies carried out revealed pronounced changes in the

inflammatory nature of all constituent components. Microscopically, signs of gingival hyperkeratosis were found in the epithelial lamina, the basal layer contained predominantly pyknotic cells, and rarely mitotic figures. The thickness of the spinous layer decreased. Thinning of the epithelial lamina was noted in the areas of the furrow and attachment. Disruption of the epithelial attachment and the fibers of the circular connection led to the formation of a periodontal pocket, in which detrital masses, desquamated epithelial cells and leukocytes were found. There was a violation of the mineralization of the dental alveoli, focal lacunar resorption was determined. The periosteum was hyperplastic with proliferation of young osteoblasts. Submicroscopic studies of the structural components of the periodontal complex of animals with experimental periodontitis also revealed an increase in destructive changes. The zones of collagen fiber stratification, edema of the amorphous component of loose connective tissue were determined. Fragmented bundles of collagen fibers and amorphous intercellular substance of connective tissue were localized around the fibrocytes.
Keywords: periodontium, lipopolysaccharide, inflammation, periodontitis, epithelium.

Relevance. In Ukraine, the prevalence of periodontal disease among the population ranges from 92 to 98%, the most common are chronic catarrhal gingivitis and generalized periodontitis [1]. There is evidence that even 15-18-year-old adolescents in 55-89% of cases suffer from gingivitis or the initial stage of generalized periodontitis [2]. In addition, a feature of paradontopathogenic microflora is high virulence and toxicity – the outer wall of one gram-negative bacterium can contain up to 3.5 million molecules of lipopolysaccharide endotoxin. After cell death, the endotoxins of these bacteria remain biologically active molecules and, in combination with tissue damage, provoke the activation of mononuclear phagocytes and the release of a huge amount of biologically active substances, increasing the inflammatory response in the periodontium [3, 4]. However, despite numerous studies, the features of pathomorphological and ultrastructural changes in the development of generalized inflammation and destruction in the periodontal complex have not yet been sufficiently studied [5]. Thus, the research focuses on the study of the pathogenesis of generalized periodontitis and the establishment of the characteristics of his current relevant and promising for a successful therapeutic strategy in patients with this disease [6, 7].

Purpose of the study. To elucidate the pathomorphological features of lesions in the rat's periodontal complex with lipopolysaccharide endotoxin of gram-negative microflora.

Material and research methods. The experiments were carried out on male white rats. To simulate periodontitis, the animals were injected into the gum for 2 weeks every other day, 40 microliters (1 mg / ml) of lipopolysaccharide (LPS) E. Coli («Sigma-Aldrich», USA»). The sampling of material for microscopic and electron microscopic studies was carried out according to the generally accepted technique [8]. The organocomplex (frontal group of teeth of both jaws: alveolar processes with mucous membrane) was removed, fixed in 10% formalin solution, histological sections were prepared after preliminary decalcification, stained with hematoxylin-eosin. Ultrathin sections made on an LKB-3 ultramicrotome were stained with a 1% aqueous solution of uranyl acetate, contrasted with lead citrate according to the Reynolds method, and studied under an electron microscope.

Results and their discussion. Histological studies of periodontal components in rats with lipopolysaccharide periodontal inflammation revealed pronounced changes in the

inflammatory nature of all components. Microscopically, signs of hyperkeratosis of the free part of the gums were found in the epithelial lamina; the thick stratum corneum often contained light gaps between the stratum corneum, where intercellular contacts were disrupted. The basal layer contained predominantly pyknotic cells; mitotic figures were rarely found. The thickness of the spinous layer decreased. In most epithelial cells, the karyoplasm of the nuclei was clear, which led to their vacuolization. Intra-epithelial lymphocytes appeared in the thickness of the epithelial layer. Experimental damage to the soft tissues of the periodontium in rats also resulted in damage to the sulcular and ligamentous epithelium with zones of erosion and ulceration. Thinning of the epithelial lamina was noted in the areas of the furrow and attachment. Desmosomal contacts between epithelial cells were disrupted with the formation of wide intercellular spaces, infiltrated leukocytes and macrophages appeared. Disruption of the epithelial attachment and the fibers of the circular connection led to the formation of a periodontal pocket, in which detrital masses, desquamated epithelial cells and leukocytes were found. In addition, damage to the hard periodontal tissues was observed. There was a violation of the mineralization of the dental alveoli, the Havers canals were dilated, in the wall of which there were numerous osteoclasts, and lacunar resorption was focal. The periosteum was hyperplastic with the proliferation of young osteoblasts. There was unevenly mineralized cement in the apex and thinning of the primary cement.

Ultrastructural studies of the rat gingival mucosa revealed changes in the vessels of the microvasculature. Most of the hemocapillaries were with dilated lumens, blood supply. Endothelial cells contained electron-dense nuclei with a predominance of heterochromatin. The intussusception of the karyolema was significant, and the expansion of the perinuclear space was determined. The cytoplasm of the cells contained damaged organelles, swollen mitochondria with fragments of cristae. In the peripheral cytoplasmic areas of endothelial cells, there are few micropinocytic vesicles and caveolaes. The basement membrane was edematous, loosened, and indistinct. Submicroscopic studies of the structural components of the periodontal teeth of animals with experimental periodontitis also revealed an increase in destructive changes. The zones of collagen fiber stratification, edema of the amorphous component of loose connective tissue were determined. In fibroblasts, changes were also found on the part of the nuclei. They had an irregular shape, invagination of the karyolem. The karyoplasm contained a breast of heterochromatin, which was located mainly under the karyolem. The cytoplasm also looked electron-dense, homogeneous, the density of organelles was low. The fibrocytes contained altered nuclei, there were significant invaginations of the karyolem, and heterochromatin predominated in the karyoplasm. Fragmented bundles of collagen fibers and amorphous intercellular substance of connective tissue were localized around the fibrocytes. Violation of vascular-tissue relations was characterized by the presence of tissue basophils containing numerous electron-dense granules in the areas of damage to the periodontium, and degranulated cells were also found.

Conclusions. Microscopic studies of the periodontal components of experimental rats with lipopolysaccharide inflammation established the reorganization of all its structural

components, changes in the epithelium of the gingival mucosa, both free and in the areas of the groove and attachment, an increase in the stratum corneum with thinning of the spinous were observed. Submicroscopic studies of periodontal components have established that blood filling, sludge, and wall damage are determined in the microvessels of the gums and periodontium. Violation of microcirculation led to edema of the main substance of the gums and periodontium, changes in the ultrastructure of cellular elements and damage to fibrous structures were revealed.

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