Morphofunctional changes in the uterine tissues of laboratory animals in chlamydial infection

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Abstract. In diseases caused by Chl psittaci, a hematogenic type of infection is often observed, accompanied by damage to the CNS and internal organs: liver, spleen, lung. Lymphogenic spread of chlamydia (Chl trachomatis, venereal lymphogranulomatosis) is possible. The hematogenic pathway of infection is most frequently observed in Chl trachomatis diseases (serovars D and K). Different biological variants (subspecies) of chlamydia cause lesions of different organs and systems. Chl trachomatis causes conjunctivitis, locally affects the mucous eye and urogenital tract.

1. Introduction
When examining the internal organs of animals in experimental chlamydiosis, it is possible to study reliable changes in organs and systems during the progressive course of the disease. In adults, diseases caused by chlamydia tend to be asymptomatic. Under certain conditions, namely, the decoupling of immunological functions, the disease can transition to deep systemic lesions of many organs and tissues, as well as provoke autoimmune reactions when the antigen becomes components of cellular structures. In adults, diseases caused by chlamydia tend to be asymptomatic. Under certain conditions, namely, decoupling of immunological functions, the disease can transition to deep systemic lesions of many organs and tissues, as well as provoke autoimmune reactions when the antigen becomes cellular systems of the body. This infection can progress into a chronic stage with preferential pelvic organ damage. Chronic inflammation of organs of small pelvis leads to tube occlusion with further development of infertility, which is especially important in selection of particularly valuable animal breeds. The biovar of venereal lymphogranulomatosis Chl trachomatis infects both macrophages and epithelial cells and spreads through the lymphatic system. Chlamydia pneumoniae causes pneumonia, bronchitis and pharyngitis, moreover, Chl pneumoniae is considered to be associated with a wide range of conditions such as chronic bronchitis and pneumonia with transition to respiratory failure, diseases of the coronary arteries. Chl psittaci is an agent that causes mainly respiratory tract diseases known as psittacosis. Chlamydia cause diseases with polyorganicity of damage due to endotheliotropism and epitheliotropy, they can provoke oncological diseases. The stages of the disease developing after infection with chlamydia are mediated by an immune response. The animal experiment shows the importance of an immune response for release from infection. Chronic chlamydia is known to render immune system protective trigger switches ineffective. The data on the clinical and morphological picture of various chlamydiosis species are quite diverse and extensive. Chlamydia destroy endothelial cells, causing a trigger immune response with damage not
only to the agent, but also to the cells within which it is located. Local lymphocytes and macrophages pulling to the site of infection may be continuing a cycle of destruction and repair. Broken, chlamydia-infected arterial endothelial cells cause increased release of tissue factors of procoagulant activity that cause thrombosis and platelet adhesion. Chronic, chlamydia-induced, endothelial destruction, inflammation, platelet adhesion, thrombosis, endothelial desquamation, and smooth muscle tissue proliferation lead to the formation of chronic vascular insufficiency of many organs and systems at the level of histogematic barriers, which increases their permeability with further development of changes of general pathological and specific nature, often irreversible. This effect may result from direct colonization of vascular walls during infection. This local infection directly affects the vessel wall, resulting in vascular failure, but may indirectly activate endothelial autoimmune processes. Consequently, the primary link in the development and progression of chlamydial infection is the damage of the vascular channel, in particular the arteries, causing further pathological changes in organs.

2. Equipment and devices used in studies

The material for research was pathogenic microorganisms (chlamydia). Experiments were carried out in rats. Chl Psittaci, a strain of "Laurie" isolated from parrot in 1957, was used to infect rats; A description of the microorganism is given in the "Strain Catalogue," item 4, 1962. For the experiments, 30 female unborn sex-aged rats were taken, and the average weight of females was 250g. Two weeks 2 the experiment, the animals were quarantined. The infectious material was administered to the animals intraperitoneally as a 10% suspension of differential centrifugation purified ovoculture Chl psittaci strain "Laurie." The infectious titer of the inoculum was 10-7 LD50/0.5 ml for chicken embryos. The animals were divided into 2 groups, the first experimental, the second control, in each group there were 15 individuals. All animals of the first group were infected with the agent, and the second group served as a control, they were injected with saline intraperitoneally. The animals were sacrificed after 30 days by overdose with ether, after which the test material was fixed in 10% formalin. The next day, pieces of fabric were cut, followed by wiring on alcohols of increasing strength, and the material was poured into paraffin. Histological sections were stained with hematoxylin and eosin, hematoxylin stains in blue-violet tones the shell of cell nuclei, chromatin. Eosin dyes in pink-red-orange tones of the cytoplasm and some structures (fibers) (review technique), and by Van Gison to determine the degree of expression of scleroplastic processes. Sections up to 5 microns thick were made from the finished blocks on the slant microtome. The obtained preparations were studied with Zeiss microscope (Axioskop40) with ocular magnification x10, with lenses x 4, x10.

3. The results of the study and their discussion

In our opinion, morphological changes in the organs of the reproductive system deserve the most attention, because persisting the agent in them can serve as a source of intrauterine infection of the fetus with the development of irreversible changes, often leading to death of the organism [1, 2]. In these organs under normal conditions the degree of cellular renewal is increased, metabolic processes are at a high level, which requires adequate access to oxygen and nutrients, which is provided by vascular circulation. Pathological processes in uterus are characterized by development of dyscirculatory, dystrophic and inflammatory changes [3]. The most pronounced morphological changes are observed, as mentioned above, in the vascular channel, especially in the arteries of the endometrium and myometrium (figure 1).

Arterial and venous vessels are full blood, edema is pronounced in the walls. Arteries are thick-walled, endothelial cells of their inner layer have enlarged nuclear-containing parts projecting into vascular lumen [4]. In individual fields of vision, the endothelial cells flow into the lumen of the vessels, exposing the basal membrane (figure 2).
This process is dangerous by subsequent adhesion of platelets, which trigger the process of blood coagulation followed by thrombus formation [5]. The muscle layer of the arterial wall is underlined. Fiber on the side of serous layer contains arterial vessels with similar changes described above, thin-walled full-blood veins. Muscle shell of arterial walls with signs of myocyte hypertrophy, cytoplasm of cells is clearly evacuated [6]. In some places, the passage of the muscle fibers is interrupted by the interconnecting fibers located there between. Around vessels there are visible layers of fibrous tissue according to the type of perivascular sclerosis, in which cells of lymphomacrophagal row are not uniformly located, small clusters of plasma cells (figure 3).

The mucous membrane of the uterine cavity forms true nipples coated with one layer of cylindrical or flattened cells (figure 4).
In the endometrial stroma there are single small glands formed by cells of cylindrical or cubic shape with uneven lumen. Stroma cells are arranged in thick or sparse fields [7]. Blood vessels are few, characterized by uneven blood filling. The muscle shell of the uterus in the form of ordered muscle layers, cell nuclei-myocytes trace quite well, large, often hyperchromic with distinct nuclei. In mother horns, the changes are similar to those described above. Epithelium of tufts is flattened, mucous membrane forms true nipples of different size, in the basis of which fibrous tissue is located [8]. The muscle shell is thin, double-layered. Blood vessels are few, weak blood filling (figure 5).
4. Conclusion
Chlamydia cause diseases with polyorganicity of lesion due to endotheliotropicity and epitheliotropy. They can provoke cancer. The stages of the disease developing after infection with chlamydia are mediated by an immune response. The animal experiment shows the importance of an immune response to release from infection. Chronic chlamydia is known to render immune system protective trigger switches ineffective. The data on the clinical and morphological picture of various chlamydiosis species are quite diverse and extensive. Chlamydia are known to destroy endothelial cells, causing a trigger immune response with damage not only to the agent, but also to the cells with which it is located. Local lymphocytes and macrophages migrating to the site of infection may be continuing a cycle of destruction and repair. Broken, chlamydia-infected arterial endothelial cells cause increased release of tissue factors of procoagulant activity that cause thrombosis and platelet adhesion. Chronic, chlamydia-induced, endothelial destruction, inflammation, platelet adhesion, thrombosis, endothelial desquamation, and smooth muscle cell proliferation result in chronic vascular failure of many organs and systems at histogemative barrier levels, which increases their permeability with further changes in pathological and specific character, often irreversible. This effect may result from direct colonization of vascular walls during infection. This local infection directly affects the vessel wall, resulting in vascular failure, but may indirectly activate endothelial autoimmune processes. Consequently, the primary link in the development and progression of chlamydial infection is the damage of the vascular channel, in particular the arteries, causing further pathological changes in organs. Morphological changes in the reproductive system deserve the most attention, as persisting the agent in them can cause intrauterine infection of the fetus with the development of irreversible changes, often leading to death of the organism. In these organs under normal conditions the degree of cellular renewal is increased, metabolic processes are at a high level, which requires adequate access to oxygen and nutrients, which is provided by vascular circulation.

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