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Morphological Characteristics of The Lung Tissue in The Reproduction of a New Experimental Model of Acute Lung Abscess Against the Background of Diabetic Microangiopathy

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Abstract

A morphological study of lung tissue in animals with an experimental model of acute purulent-destructive lung disease on the background of diabetic angiopathy was carried out. It has been established that in the initial phase of modeling purulent inflammation against the background of diabetes mellitus, diffuse plethora of blood vessels, disorganization of their walls, development of edema and perivascular hemorrhage, leukocyte infiltration of the interalveolar septa and the appearance of foci of confluent inflammation and distelectasis of the lung tissue are noted. On the 7th day after modeling the pathological process, there is an increase in dyscirculatory, destructive and inflammatory processes in the form of paralytic expansion of the vascular wall due to fibrinoid swelling, increased migration of leukocytes into the lung tissue, the development of destruction, the formation of microabscesses, compaction of the lung tissue due to interstitial inflammation and distelectasis . Diabetic angiopathy leads to the development of mild dystrophic and destructive changes in the tissue structures of the lung, which, when modeling a purulent process, are combined with destructive and inflammatory processes, leading to massive damage to the lung tissue in the form of a complete structureless destruction of the lung tissue, the formation of diffuse and focal purulent inflammation, in particular abscesses. In the later stages after the modeling of a purulent process in the lung, the formation of a limited round and oval shape of abscess foci is noted against the background of the preservation of dystrophic and sclerotic changes in the lung tissue due to diabetes mellitus. At the same time, foci of abscess formed between the lung segments, in the area of localization of large vessels and bronchi.

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INTRODUCTION

Nosocomial pneumonia is one of the most common diseases in humans and is one of the leading causes of death from infectious diseases [1,7].

From a practical standpoint, it is advisable to single out groups of patients with nosocomial pneumonia, taking into account comorbidities (chronic obstructive pulmonary disease, diabetes mellitus, chronic heart failure, cerebrovascular diseases, diffuse diseases of the liver, kidneys, with impaired functions, chronic alcoholism, etc.) [2, 3].

The main reasons that contribute to the development of nosocomial pneumonia in patients with diabetes mellitus are: decreased immunity and general weakening of the body; an increase in the likelihood of infection entering the respiratory tract, including by aspiration; hyperglycemia, which not only contributes to the development of pneumonia, but also leads to a more severe course of the disease than in patients with normal blood sugar levels; pathological changes in the vessels of the lungs

(microangiopathy), which, according to the literature, occur in patients with diabetes mellitus, twice as often as in healthy people; other comorbidities.

All these factors, as well as poor control of blood sugar levels, create favorable conditions in the human body for damage to the respiratory tract, including pneumonia. And an infection penetrating into the lungs becomes the destabilizing factor that aggravates the situation of a weakened organism [3,4,5].

A general decrease in immunity not only increases the likelihood of pneumonia, but can also lead to a severe course of the disease and various complications [4,6,8].

All this dictates the need for more in-depth research into the mechanisms of development of these pathological conditions. Of course, the best way to conduct such studies is experimental. However, difficulties arise not only in terms of modeling a lung disease, but also a combination of the process with another, chronic pathological process, in this case, diabetes mellitus and its complication in the form of angiopathy.

Our team has experience in experimental modeling of acute and chronic surgical diseases. However, the approach in choosing a combination of two complex processes, we carried out in the first place.

The aim of our study: to evaluate the morphological picture of the lung tissue in the simulation of acute lung abscess against the background of diabetic angiopathy.

MATERIAL AND METHODS

In the experiment, outbred rabbits of both sexes weighing 1900-2200 g were used, which were on the usual laboratory diet. By the beginning of the experiment, each group consisted of 10-12 rabbits. A total of 246 animals were used.

Diabetic microangiopathy in rabbits was modeled according to the original method developed by us ("Method for modeling diabetic angiopathy"). Modeling was carried out as follows: rabbits weighing 1900-2200 grams on an empty stomach under ether anesthesia were intraperitoneal injected with 100-110 mg/kg of the drug doxorubicin in a 0.9% sodium chloride solution, and 48 hours after doxorubicin administration, once daily for 3 days, intraperitoneal administered 0.2-0.4 ml of 70% sorbitol solution. Starting from the 20th day, the animals began to develop the clinical picture of diabetic angiopathy.

At the second stage, in the same animal, against the background of formed diabetic angiopathy, an acute purulent-destructive lung disease was modeled according to the original method developed by us ("Method for modeling acute purulent-destructive lung disease").

Modeling was carried out as follows: in order to increase the reactivity of the macroorganism, 0.3-0.5 ml of 10% ammonia solution was first injected into the subcutaneous tissue of the lumbar region of the animal. After 48 hours, the development of a focus of necrosis was noted at the injection site, and on the same day, under surface ether anesthesia, 0.5-0.7 ml of 5% suspension of autocal per 200 g of animal weight was injected into the IV intercostal space on the right midaxillary line.

In dynamics, starting from the third day after the injection of autocal suspension, the development of acute purulent-destructive lung disease was observed. Over the next 28 days, the rabbits developed a clinical picture of acute purulent-destructive lung disease.

For histological examination, pieces of lung tissue were fixed in neutral formalin, Carnoy's fluid and embedded in paraffin. Sections were stained with hematoxylin-eosin according to Van Gieson. Light microscopy and morphometry were carried out on a trinocular microscope sample XSZ-20 (China) with a direct digital electronic attachment.

RESULTS

The results of a morphological study of the modeling of acute purulent-destructive lung disease against the background of diabetic angiopathy in the experiment showed that on the 3th day of the experiment, expansion and plethora of blood vessels were noted in the lung tissue, a violation of the rheological properties of blood in the form of separation of blood elements from the plasma component and the development of edema of the interalveolar septum and lumen of some alveoli. There is a diffuse infiltration of the interalveolar septum with segmented leukocytes, an increase in the proliferative activity of local histiocytic cells in the walls of the alveoli (figure 1).

In places in the lung tissue, inflammatory infiltration becomes more pronounced and infiltrates both the interalveolar septa and the lumen of the alveoli with the development of confluent inflammation and the appearance of foci of distelectasis. In the foci of inflammation, the vessels are full-blooded, in their wall the development of mucoid and fibrinoid swelling is noted with an increase in permeability and the appearance of foci of perivascular hemorrhage.

Examination of the lung tissue under a large microscope object showed that the venous vessels were sharply dilated, plethoric with hemol-

ysis of erythrocytes and the appearance of hemoglobinogenic pigments both in the lumen of the vessels and in the foci of hemorrhage around the vessels. At the same time, the bronchioles are narrowed, the wall is corrugated due to the tortuosity of the integumentary epithelium and edema, plasmorrhagia of the muscle layer (figure 2). The interalveolar septa are thickened and deformed due to severe infiltration by segmented leukocytes, hemorrhage, and edema of the intestinal tissue.

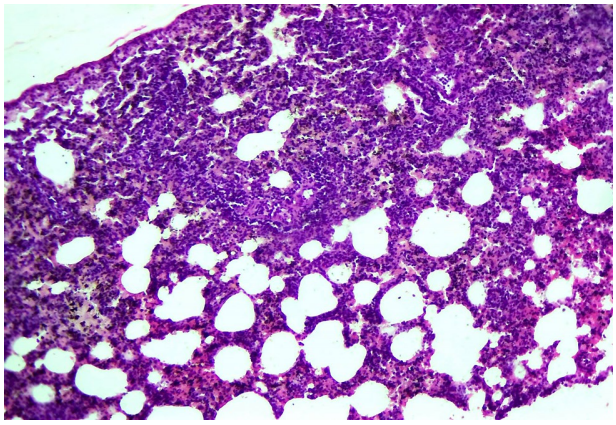

Figure 1. 3th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Diffuse segmented cellular infiltration. Stained with hemotoxin-eosin. Magnification: 10x10.

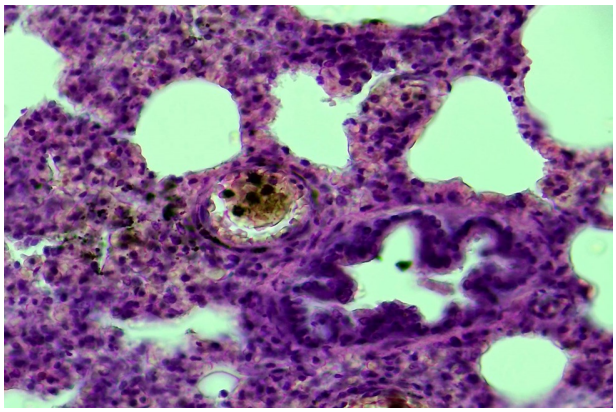

Figure 2. 3th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Narrowing of the bronchioles, plethora of blood vessels, the appearance of hemoglobinogenic pigments. Stained with hemotoxin-eosin. Magnification: 10x40.

During this period of the experiment, thrombosis of large venous vessels and a pronounced plethora of capillaries of the interalveolar septa were also noted. At the same time, in the lumen of a large venous vessel of the lung tissue, the formation of a homogeneous protein mass from plasma proteins, the sedimentation of blood elements from both red and leukocyte clones is determined (figure 3). The interalveolar septa are somewhat thickened, deformed due to the plethora of capillaries and diapedetic hemorrhage.

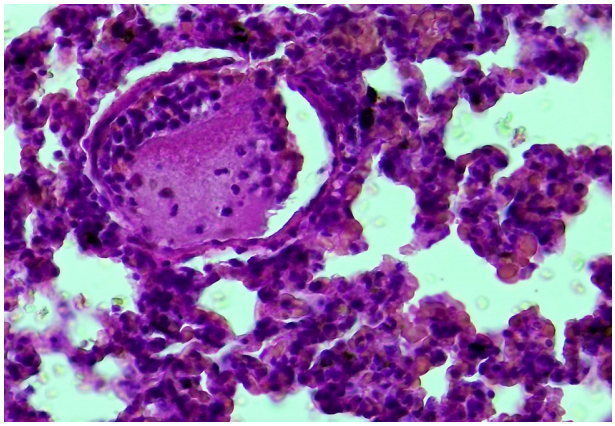

Figure 3. 3th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Thrombosis of venous vessels, hemorrhages in the interalveolar septa. Stained with hemotoxin-eosin. Magnification: 10x40.

Examination of lung tissue under a large microscope objective 3th day modeling of purulent inflammation in the lung tissue by introducing autocal marked expansion of microvessels of the interalveolar septum, parietal localization of neutrophilic leukocytes. At the same time, the wall of the microvessels of the interalveolar septum is loosened, edematous in the form of defibration of fibrous structures, vacuolization of the intercellular substance, an increase in the proliferative activity of both endothelial and pericytic cells of the vascular wall. Deformed erythrocytes, marginal location of segmented leukocytes (figure 4), disorientation of monocytic and lymphoid cells are determined in the lumen of the vessel.

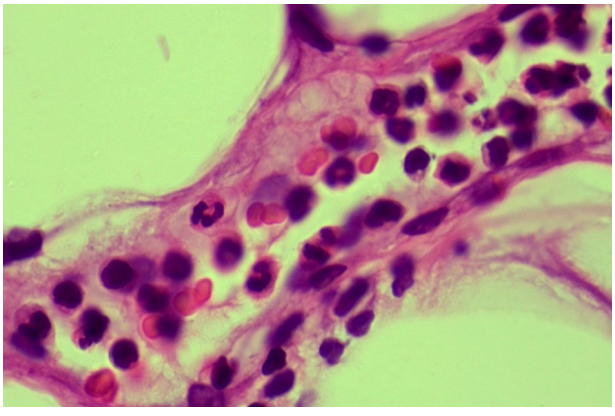

Figure 4. 3th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Expansion of microvessels of the lung, marginal location of segmented leukocytes. Stained with hemotoxin-eosin. Magnification: 10x100.

Thus, in the initial phase of modeling purulent inflammation against the background of diabetes mellitus, diffuse plethora of blood vessels, disorganization of their walls, develop-

ment of edema and perivascular hemorrhage, leukocyte infiltration of the interalveolar septa and the appearance of foci of confluent inflammation and dystelectasis of the lung tissue are noted.

On the 7th day after the modeling of acute purulent-destructive lung disease against the background of diabetic angiopathy, there is an increase in dyscirculatory and inflammatory processes. All types of blood vessels are in a state of paralytic expansion with diffuse hemorrhage into the perivascular space and alveolar tissue. The interstitial tissue around the vessels and bronchi, the interalveolar tissue and the lumen of the alveoli are diffusely infiltrated by erythrocytes with their decay and the formation of hemoglobinogenic pigments. The walls of the vessels are thickened due to mucoid and fibrinoid swelling, as well as plasmorrhagia, where endothelial and pericytic cells are in a state of proliferative activity (figure 5).

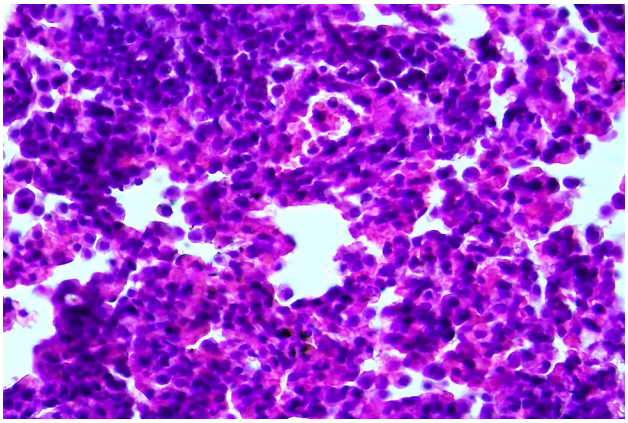

Figure 5. 7th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Strengthening of discirculatory and inflammatory phenomena in lung tissue. Stained with hemotoxin-eosin. Magnification: 10x40.

The interalveolar septa are deformed and destroyed due to severe infiltration of both segmented and mononuclear leukocytes. Among the inflammatory infiltration, the appearance of monocytic cells and macrophages is determined, in the cytoplasm of which the appearance of phagosomes and pigment particles is noted.

The progression of dyscirculatory and inflammatory phenomena on the 7th day of the experiment leads to the formation of foci of microabscesses and zonal accumulation of segmented leukocytes. At the same time, characteristic pathomorphological changes occur in the microvessels of the lung tissue in the form of an expansion of the lumen of microvessels, an increase in permeability and an increase in the development of chemotaxis, which lead to the migration of polynuclear leukocytes into the surrounding tissues of the lung (figure 6).

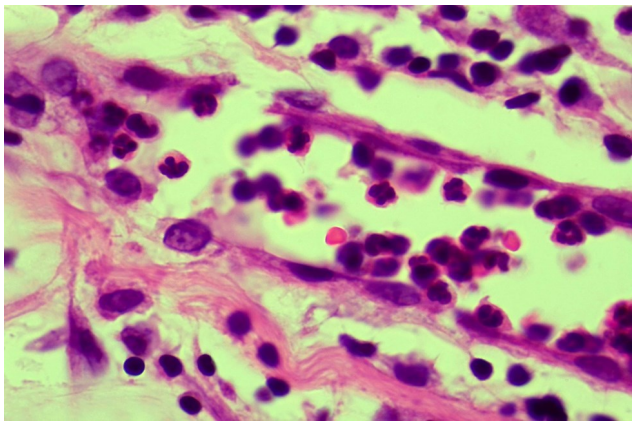

Figure 6. 7th day of modeling acute purulent-destructive lung disease against the background of diabetic angiopathy. An increase in the number of neutrophilic leukocytes in the lumen of the microvessels, their migration to the interalveolar septa. Stained with hemotoxin-eosin. Magnification: 10x100.

At the same time, the fact is determined that enlarged pores appear in the wall of the microvessel, the migration of neutrophilic leukocytes through the interendothelial spaces and transendothelial migration. A large number of neutrophilic leukocytes is determined in the lumen of the vessel and in the surrounding tissue.

During the formation of purulent inflammation in the lung tissue, there is an increased migration of neutrophilic leukocytes through both damaged and undamaged walls of microvessels. Histological examination of the lung tissue shows a thickening of the interalveolar septa due to neutrophilic infiltration and the formation of microabscesses (figure 7).

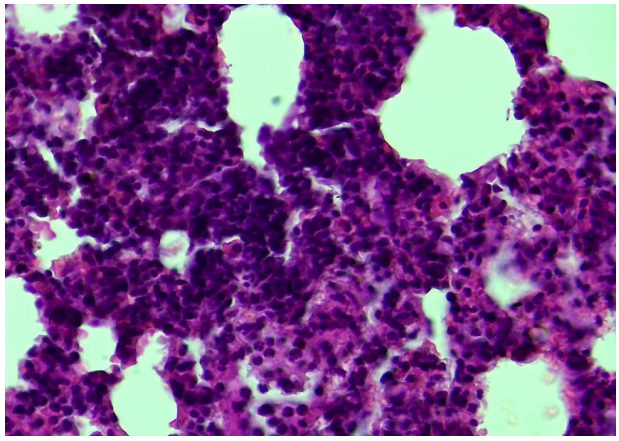

Figure 7. 7th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. The appearance of foci of microabscesses in the lung tissue. Stained with hemotoxin-eosin. Magnification: 10x40.

Thus, on the 7th day after modeling an acute purulent-destructive lung disease against the

background of diabetic angiopathy, there is an increase in dyscirculatory, destructive and inflammatory processes in the form of paralytic expansion of the vessel wall due to fibrinoid swelling, increased migration of leukocytes into the lung tissue, development of destruction, formation microabscesses, compaction of the lung tissue due to interstitial inflammation and distelectasis.

On the 14th day of reproduction of an acute purulent destructive lung disease against the background of diabetic angiopathy, the development of dystrophic, destructive changes in the walls of the lung vessels characteristic of diabetes mellitus, which manifested as mucoid, fibrinoid swelling, plasmorrhagia of the walls of medium-sized vessels, is noted. In the wall of small vessels, swelling and desquamation of endothelial cells, edema and loosening of the basement membrane with increased permeability and the release of both erythrocytes and leukocytes into the interstitial tissue of the lung are determined. Due to these changes on the part of the vessels in the lung tissue, the appearance of foci of perivascular and massive hemorrhage (figure 8), the formation of hemoglobinogenic pigments, massive infiltration by polynuclear leukocytes with the formation of microabscesses are determined. Active macrophages and hypertrophied histiocytic cells appear among the leukocyte infiltration.

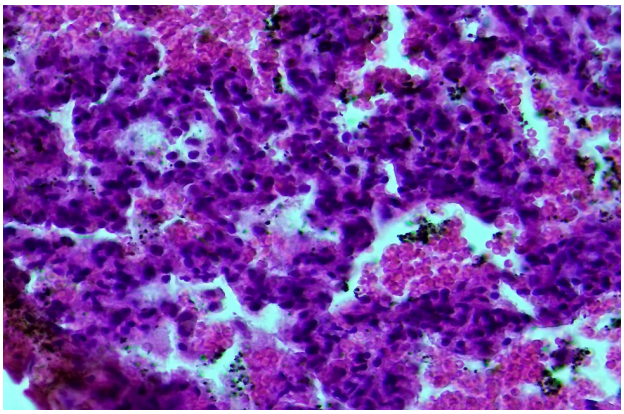

Figure 8. 14th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Massive foci of hemorrhage, dystelectasis and the formation of microabscesses. Stained with hemotoxin-eosin. Magnification: 10x40.

Degenerative and destructive changes in the lung tissue in diabetic angiopathy are combined with destructive and inflammatory processes after modeling purulent inflammation. At the same time, pronounced edema, loosening, complete destruction of the alveolar tissue with the loss of the respiratory and alveolar structures characteristic of lung tissue are morphologically noted. Interalveolar septa are not determined due to the collapse and destruction of tissue elements (figure 9). Such unstructured areas are diffusely and densely infiltrated with polynuclear and mononuclear hematogenous cells.

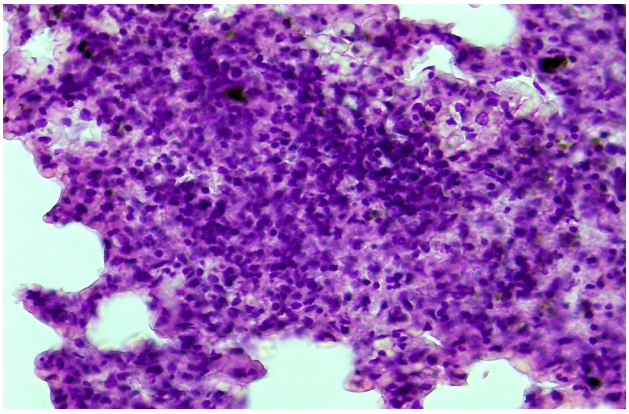

Figure 9. 14th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. Decay, destruction of lung tissue and the formation of an abscess focus. Stained with hemotoxin-eosin. Magnification: 10x40.

The accumulation of blood leukocytes in the focus of destruction, in particular lung tissue, is assessed as the formation of a focus of purulent inflammation or abscess. In this case, destructive changes in the blood elements, as well as local histiocytic cellular and tissue structures, predominate in the focus of the abscess.

The study of lung tissue after modeling purulent inflammation on a large microscope lens allows you to qualitatively determine all types of cellular composition of the abscess.

The figure shows an experimental lung abscess, which is represented mainly by neutrophilic leukocytes, macrophages, single lymphoid cells, destructive fragments of various cells (figure 10). Also, dystrophic and destructive changes in the background lung tissue are determined in the form of decay of connective tissue cells and fibrous structures that developed due to diabetes mellitus.

Thus, the modeling of diabetes mellitus is accompanied by the development of mild dystrophic and destructive changes in the tissue structures of the lung, which, when modeling a purulent process, are combined with destructive and inflammatory processes, lead to a massive lesion of the lung tissue in the form of a complete structureless destruction of the lung tissue, the formation of diffuse and focal purulent inflammation, in specific abscesses.

At the later stages of the experiment (21st and 28th days) in the lung tissue, the formation of a limited abscess is noted. At the same time, stabilization of dystrophic and destructive

changes characteristic of diabetes mellitus is noted in the lung tissue. The walls of the vessels are somewhat thickened due to sclerosis and proliferation of histiocytic cells. In the circumference of the vessels, the presence of edema and loosening of the adventitial tissue is determined. On the part of the alveolar tissue, dyscirculatory, degenerative changes are observed in the form of vascular plethora, diffuse and focal hemorrhage with the development of distelectasis and pigmentation. The development of distelectasis around large vessels and bronchioles is especially noted. In the wall of the bronchi and bronchioles and in their circumference, the development of proliferative inflammation and sclerosis is noted in the form of proliferation of cellular and fibrous connective tissue. In the lung tissue, especially in the area between the lung segments, around large vessels, the presence of a limited accumulation of neutrophilic leukocytes in the form of abscess formation is determined. An abscess is morphologically manifested as a round or oval dense accumulation of predominantly segmented leukocytes with an admixture of macrophages and single lymphoid cells (figure 11). The leukocyte mass of the abscess was histologically stained with hematoxylin, hyperchromic blue.

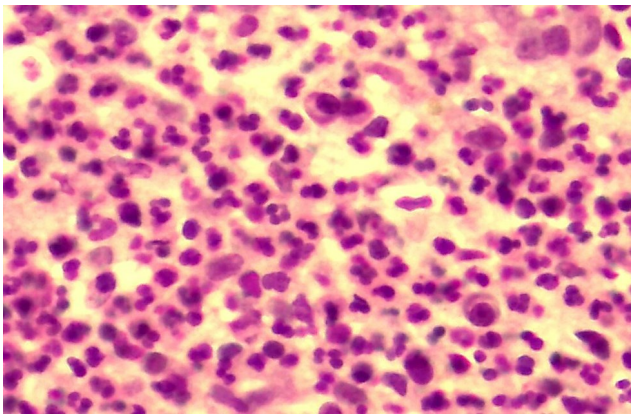

Figure 10. 14th day of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. The composition of the lung abscess consists of neutrophilic leukocytes, macrophages, single lymphocytes and destructive cells. Stained with hemotoxin-eosin. Magnification: 10x100.

When studying the abscess focus under a large microscope lens, a dense accumulation of predominantly polynuclear leukocytes is noted, among which there are macrophages, lymphoid cells and fragments of the destruction of various cellular and tissue elements (figure 12). In contrast to the previous terms, the presence of group arrangements of neutrophilic leukocytes around macrophages and histiocytic cells is determined in the composition of cellular infiltration. The presence of decay of both neutrophilic and macrophage cells is also noted. The connective tissue basis of the abscess is represented by proliferatively active histiocytic and monocytic cells, a mellow homogenized fibrous structure.

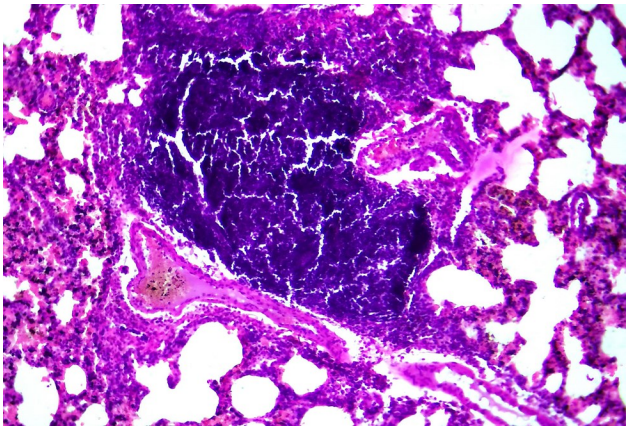

Figure 11. Day 21 of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. The formation of a large focus of abscess in the lung. Stained with hemotoxin-eosin. Magnification: 10x10.

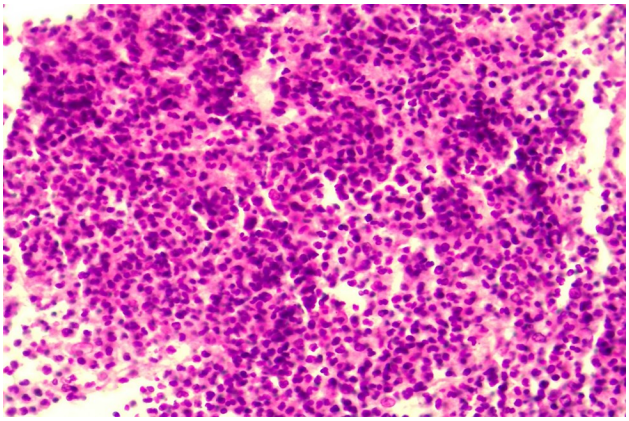

Figure 12. Day 28 of modeling acute purulent-destructive lung disease on the background of diabetic angiopathy. A dense accumulation of leukocytes, macrophages and lymphoid cells in the abscess. Stained with hemotoxin-eosin. Magnification: 10x10.

Thus, in the later stages after the modeling of a purulent process in the lung, the formation of a limited round and oval shape of abscess foci is noted against the background of the preservation of dystrophic and sclerotic changes in the lung tissue due to diabetes mellitus. At the same time, foci of abscess formed between the lung segments, in the area of localization of large vessels and bronchi.

Conflict of Interest - The author declares no conflict of interest.

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Compliance with the principles of bioethics - all studies were carried out with the permission of the bioethical committee.

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