DIFFERENTIAL DIAGNOSIS OF MORPHOLOGICAL CHANGES IN THE LUNGS IN CHRONIC RENAL FAILURE

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Resume. The study of lung morphology under the influence of various diseases is of great interest to various areas of medicine and biology. In the experiment, the effect of morphological changes in the respiratory part of the lungs of rats was studied in the background of chronic kidney failure in 12-month-old white rats and compared with other diseases.

Key words: Diabetes, macroangiopathy, microangiopathy, microangiopathy, surfactant, metastatic calcification, interstitial fibrosis

Introduction: Morphological changes of the lungs in diabetes include a number of pathological processes, because this disease affects the whole organism, including lung tissue. Although the effects of diabetes on the lungs have not been widely studied, some morphological changes can be observed:

1. Damage to blood vessels: Diabetes can cause microangiopathy (damage to small blood vessels) and macroangiopathy (damage to large blood vessels). The walls of the capillaries in the lungs thicken and may lose their elasticity. It disrupts the blood circulation of the lungs and affects the gas exchange.

2. Fibrosis in the interstitial tissues of the lungs: Diabetes can accelerate the process of fibrosis in the tissues of the lungs, that is, the formation of connective tissue in place of the tissues increases. This reduces the elasticity of the lungs and impairs the respiratory function.

3. Surfactant deficiency: Surfactant is a liquid that closes the alveoli of the lungs, keeping the lungs open. Diabetes can reduce the production of this substance, predisposing the lungs to collapse.

4. Deformation of the alveoli: Due to diabetes, the size and shape of the alveoli of the lungs (air bubbles) can change, which leads to a violation of gas exchange. At the same time, fluid accumulation in the alveoli (fluid in the lungs) can be observed.

5. Susceptibility to infections: Patients with diabetes have a weakened immune system, which increases the risk of lung infections, such as pneumonia.

In chronic heart failure (CHF), a number of morphological changes occur in the lung tissue, because the inability of the heart to pump blood enough leads to fluid accumulation in the lungs and other pathological processes. These changes develop as a result of impaired blood flow and gas exchange from the heart to the lungs. Below are the main morphological changes observed in the lungs in chronic heart failure:

1. Pulmonary edema: Blood stagnation occurs in the pulmonary capillaries due to the weakening of the activity of the left ventricle of the heart. This causes fluid to flow from the capillaries into the alveoli. Pulmonary edema is morphologically characterized by the accumulation of fluid in the alveoli, their expansion, and a violation of gas exchange in the lungs. In the lungs, the space between the bronchus and the alveoli is also filled with fluid.

2. "nutmeg" color of the lungs: As a result of blood stagnation, pigment (hemosiderin) accumulates in the lungs, and microscopic changes are observed in the lungs. Due to the accumulation of blood elements and hemosiderin in the alveoli, the lung tissue acquires a "nutmeg" color. This change depends on the duration and intensity of the hemostatic process.

3. Pulmonary hypertension: As a result of heart failure, an increase in blood pressure in the blood vessels of the lungs is observed. Pulmonary artery walls thicken, blood vessels narrow, which causes high pressure in the pulmonary vessels (pulmonary

hypertension). Morphologically, it is manifested by fibrosis and thickening of the walls of pulmonary arteries and capillaries.

4. Congestive lung: As a result of SUI, blood stagnation (stasis) is observed in the pulmonary capillaries and veins. This process leads to swelling of the lung tissue and a decrease in their elasticity. Morphologically, congestive lung is associated with excess blood accumulation in the alveoli and blood vessels.

5. Changes in capillary walls: An increase in blood pressure causes capillary walls to thicken and lose elasticity. This reduces the ability of the capillary walls to participate in gas exchange, resulting in a lack of oxygen in the lungs.

6. Interstitial fibrosis: Fibrosis develops in the interstitial tissues of the lungs during chronic heart failure. This is due to the excessive formation of connective tissue between the alveoli and bronchi, which reduces the elasticity of the lungs and reduces the volume of breathing.

These changes are expressed in different degrees, depending on the duration of HF, the general condition of the patient, and the degree of impairment of cardiac function.

The purpose of the study: to compare the morphological changes of the lungs of white rats after chronic renal failure with the morphological changes of the lungs developed against the background of diabetes and chronic heart failure.

Research materials and methods. In the study, 150 5,9,12-month-old male and female laboratory white mice were used, and they were divided into 3 groups (n=50 each) depending on the observation period. Animals were kept in vivarium conditions according to standard ration (with food and water supply). They were injected intramuscularly with 5%-0.8mg/100mg glycerin for one month to induce chronic kidney failure in the experiment. Animals were removed from the experiment according to the plan on the 30th and 60th days of observation for morphological examination. Conducting experiments, using experiments on animals, without leaving the scope of legal regulations and the global convention (on the protection of vertebrate

animals, 1997) was fully followed. The lungs of white mice were isolated and fixed in 10% formalin. Histological sections with a thickness of 3-4 μm were stained with hematoxylin and eosin. Histological preparations were analyzed under a microscope and photographed.

Research results.

In chronic kidney failure (CKD), the structure of the lungs undergoes a number of pathological changes, because when the filtration function of the kidneys is disturbed, fluid, electrolytes and toxins accumulate in the body, not only in the kidneys, but also in other organs, including lung tissue. 'causes changes. The structure of the lungs can be changed in the following ways:

1. Pulmonary edema (fluid accumulation)

Due to SBY, the fluid balance in the body is disturbed, which causes fluid to escape from the pulmonary capillaries into the alveoli. Pulmonary edema is accompanied by swelling of the lung tissue, which fills the air spaces inside the alveoli with fluid, as a result of which the gas exchange in the lungs is disturbed. Morphologically, this is expressed by the expansion of the alveoli and the accumulation of fluid in them.

2. Interstitial fibrosis

In renal failure, the formation of connective tissue (fibrosis) is observed in the lung interstitial tissue. As a result of this process, the connective tissue in the lungs grows, preventing the gas exchange between alveoli and capillaries. Due to fibrosis, the lungs lose their elasticity, which makes breathing difficult.

3. Expansion of capillaries and damage to blood vessels

As a result of chronic kidney failure, the walls of the pulmonary capillaries thicken and the vessels dilate. It is accompanied by stagnation of blood in the lung tissue and microcirculation disorder. Morphological changes are represented by blood stagnation (congestion) and edema around the pulmonary capillaries.

4. Metastatic calcification

As a result of disturbances in calcium and phosphorus metabolism in SBY, deposition of calcium and phosphorus salts can be observed in lung tissues. This condition is called "metastatic calcification" and is morphologically represented by the deposition of salts in the lung tissue and hardening of the connective tissue. This process reduces the flexibility of the lungs and causes breathing difficulties.

5. Uremic lungs

Against the background of kidney failure, there is an increase in the level of toxins in the blood, especially urea (uremia). This leads to a condition called "uraemic lung" characterized by inflammation and swelling of the lung tissue. Lung tissue swells and liquid accumulates in the spaces between the alveoli, which disrupts the process of gas exchange.

Thus, in chronic renal failure, morphological changes such as pulmonary edema, fibrosis, damage to blood vessels and calcification occur in the structure of the lungs, which lead to breathing difficulties and impaired gas exchange in the lungs.

Morphological changes observed in the lungs of white rats during experiments with chronic kidney failure are related to similar processes in the human body, mainly due to the accumulation of fluid and toxins that develop as a result of kidney failure. indicates tissue damage. As a result of experiments on rats, the following main morphological changes were determined:

1. Pulmonary edema (fluid accumulation)

Rats have fluid accumulation in their lung tissue, which is called pulmonary edema. When the kidneys are unable to excrete enough fluid, the fluid escapes into the alveoli of the lungs and fills the air spaces within the alveoli. Morphological changes are represented by fluid-filled alveoli in the lung tissue and the presence of fluid instead of air in the lungs.

2. Expansion and thickening of the walls of the alveoli

During the experiments, thickening of alveolar walls and their expansion were observed in the lungs of rats. It is caused by interstitial inflammation and fibrosis. These changes reduce the elasticity of the lungs, which reduces the volume of breathing.

3. Interstitial fibrosis

An increase in connective tissue (fibrosis) was found in the interstitial tissues of the lungs after chronic renal failure in rats. This process changes the structure of the lungs, the spaces between the alveoli are filled with connective tissue, and it is observed with a decrease in elasticity.

4. Expansion of capillaries and damage to blood vessels

Capillary vessels in the lungs of rats are dilated, thickening and loss of elasticity in their walls. This process is associated with a violation of blood circulation in the pulmonary capillaries and the outflow of fluid from the capillaries.

5. Metastatic calcification

Disruption of calcium and phosphorus metabolism in the body as a result of SBY causes calcification (precipitation of calcium salts) in the lungs of rats. This is accompanied by hardening of the lung tissue and structural damage.

6. Thickening and narrowing of the pulmonary artery

Against the background of chronic kidney failure, blood vessels in the lungs, in particular, the walls of the pulmonary artery, thicken and narrow. This indicates the beginning of the process of pulmonary hypertension and is associated with circulatory disorders.

According to the results of experiments, changes such as pulmonary edema, interstitial fibrosis, capillary expansion, fluid accumulation and calcification occurred in the lungs of white rats due to chronic kidney failure. These changes have led to a violation of gas exchange in the lungs and breathing difficulties. The results of these experiments help to understand how the lungs are affected in chronic kidney failure.

In chronic kidney failure (CKD), a number of morphological changes are also observed in the lung tissues, because when the activity of the kidneys decreases, fluid

and toxins accumulate in the body, which directly affects the lungs and respiratory system. Lung changes may also be associated with a condition known as "uraemic lung." Below are the morphological changes observed in the lungs in chronic renal failure:

1.Fluid accumulation in the lung tissue (pulmonary edema): When the kidneys are unable to remove enough fluid, excess fluid accumulates in the body and enters the lungs. Lung tissues are filled with fluid, and as a result of increased fluid in the alveoli, gas exchange becomes difficult. This condition is morphologically expressed by swelling of alveoli and loss of their elasticity.

2. Pulmonary fibrosis processes: Uremia develops against the background of chronic kidney failure, which can cause inflammatory processes in lung tissue. As a result of inflammation, fibrosis develops in the interstitial tissues of the lungs, and connective tissue increases. This reduces the elasticity of the lungs and reduces the volume of breathing.

3. Deposition of calcium and phosphorus salts in the lungs: Against the background of kidney failure, the calcium-phosphorus balance in the body is disturbed. As a result, the accumulation of calcium and phosphorus salts in the lungs (metastatic calcification) can be observed. This leads to hardening of the lung tissues and a decrease in their function.

4. Pleural effusion: In SBY, fluid may collect in the pleural layer surrounding the lungs, and pleural effusion (pleural fluid) may develop. This condition is morphologically manifested by the accumulation of fluid in the pleural cavity.

5. Pulmonary hypertension: in SBY, excessive fluid and sodium accumulation in the body leads to an increase in pressure in the heart and blood vessels. Pulmonary hypertension (increased blood pressure in the pulmonary artery) develops as a result of a violation of blood circulation in the lungs and thickening of the walls of blood vessels in the lungs. Morphologically, it is associated with thickening and narrowing of pulmonary vessels.

6.Lung infections: Patients with SBY are at increased risk of lung infections, such as pneumonia, due to a weakened immune system. Morphologically, inflammatory processes in the lung tissue, inflammation of the alveoli and purulent collections in the alveoli can be observed.

Thus, the morphological changes observed in the lungs in chronic renal failure are mainly associated with fluid accumulation, fibrosis, calcification, and inflammatory processes.

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