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TABLE OF CONTENTS

AGRICULTURAL SCIENCES

VETERINARY SCIENCES

BIOLOGICAL SCIENCES

MEDICAL SCIENCES

EMPHYSEMA OF THE LUNGS AS A MODERN PROBLEM

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Introductions.

Pulmonary emphysema is a disease of the respiratory tract, which is caused by pathological expansion of the air spaces distal to the terminal bronchioles, leading to destructive and morphological changes in the alveolar walls.

Aim.

Studies of the etiological and pathological mechanisms of the formation of pulmonary emphysema, methods of diagnosis and differentiation with other pathologies of the pulmonary system and therapeutic options for the treatment of this pathology.

Materials and methods.

Digital access to the full-text and abstract databases of Web of Science, PubMed, Research Gate.

Results and discussion.

According to a meta-analysis, emphysema occurs in the adult population with a frequency of 0.5 - 5.7%

There are two groups of causes leading to the development of emphysema. The first group includes factors, violations of elasticity and strength of the elements of the structure of the lungs. For example, changing the properties of a surfactant, congenital deficiency of alpha-1-antitrypsin, pathological microcirculation, fine and coarse particles in the inhaled air, tobacco smoke, as well as gaseous substances (cadmium compounds, nitrogen oxides, etc.).

According to the European Lung Foundation, in European countries the

prevalence alpha-1-antitrypsin deficiency varies between 1 in 1800-2500 newborns, which is about 125 thousand people. These causes usually lead to primary diffuse emphysema. However, bronchial conduction during inhalation is not disturbed.

The factors of the second group include an increase in pressure in the respiratory section of the lungs and an increase in the stretching of the alveoli, alveolar ducts and respiratory bronchioles. Leading among them is airway obstruction, which occurs in chronic obstructive bronchitis.

The main role in the formation of emphysema is played by the protease antiprotease imbalance, due to which the destruction of the elastic lung skeleton occurs. Due to chronic inflammation and migration of macrophages and neutrophils, there is a release of proteolytic enzymes (serine and cysteine proteases, matrix metalloproteinases) and an increase in proteolytic activity in the lungs.

Neutrophil elastase destroys elastin, collagen, fibronectin, laminin, proteoglycans and other components of the extracellular matrix. Then, in turn, the small bronchi, which do not have their own cartilaginous framework, passively collapse, thereby increasing the bronchial resistance on expiration, increasing the pressure in the alveoli. Angiogenesis in the lungs is also suppressed, the balance between damaged and regenerating lung parenchyma is disturbed. There is an accelerated apoptosis, an increase in intraacinar pressure.

The characteristic symptoms are: barrel-shaped chest, expansion of the intercostal spaces, enlargement of the supraclavicular areas. Also, reduction of respiratory excursions, boxed percussion sound. Respiration significantly weakened. Percussion decreased area of relative dullness of the heart. Low standing of the diaphragm and a decrease in its mobility.

On the radiograph increased transparency of the lung fields. Symptomatic treatment of primary emphysema includes: oxygen therapy, smoking cessation and other harmful influences, including occupational ones, moderate limitation of physical activity, therapy with α1-antitrypsin inducers.

With secondary emphysema, therapy aimed at stopping respiratory and heart failure is required. In some cases, resection of the affected areas of the lung is

performed. Prevention of secondary emphysema is reduced to the prevention of chronic obstructive bronchitis.

Conclusions.

Emphysema is still a formidable adversary of humanity. However, modern science continues to study new approaches in the diagnosis of etiological factors and pathogenetic mechanisms of this disease and is struggling with this problem at all levels of disease prevention, treatment and prophylaxy of complications.