AJPMHS GLOBAL RESEARCH NETWORK

AMERICAN Journal of Pediatric Medicine and Health Sciences
Volume 01, Issue 07, 2023 ISSN (E): 2993-2149

CHRONIC OBSTRUCTIVE LUNG DISEASES

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Abstract: Several lung diseases are characterized by impaired lung function, impaired airway permeability, and difficulty in exhalation. Among these lung diseases, chronic bronchitis, especially bronchiolitis, pulmonary emphysema, bronchial asthma, bronchiectasis have a special place and cause very specific anatomical changes. In this article, chronic obstructive pulmonary disease information about diseases.

Key words: obstructive lung disease, chronic bronchitis, cough, periacinar, emphysema.

The prevalence of chronic obstructive pulmonary disease is increasing worldwide. Including, in 1995, 12.5 million children and 1.65 million people had emphysema in the United States. As a person grows older, the risk of developing this disease increases. Including in Uzbekistan, it reached 3-6% of people under the age of 10, and 24.1% under the age of 70. This indicates the urgency of the disease. In Uzbekistan, the number of diseases of the respiratory organs of the population takes the first place. In the last 10 years, the incidence rate of non-specific lung diseases has increased by 2.5 times. The number of hospitalized patients with pulmonological diseases exceeded the number of hospitalized patients with cardiovascular and endocrine diseases. 52% of ambulatory applications, 63% of emergency medical care applications, 33% of hospitalizations.

Chronic bronchitis is a chronic inflammation of the wall of the bronchi, which usually begins in large bronchi or small bronchi with a diameter of at least 2 mm. The main feature of bronchiolitis in chronic bronchi is mucus hypersecretion. Etiology and pathogenesis. Cigarette smoking, air pollution with nitrogen dioxide, sulfur dioxide, and dust particles play a role in the onset of chronic bronchitis. These can be directly or indirectly affected. When indirectly affected

by poison, they blind the mucous glands in the neurohumoral way, causing mucus hypersecretion to begin. Mucous hypersecretion is based on the hyperplasia of mucous glands and the transformation of epithelial cells of the airways into goblet cells. In the place of mucus hypersecretion, mucus plugs appear and clog the airways. Mucus in the small bronchi can cause them to become completely blocked, and they become even more blocked due to developing emphysema. Microbial infection and adenoviruses play a lesser role in the development of chronic bronchitis. Clinical manifestations of the disease depend on airway obstruction. It is characteristic to cough and produce a lot of sputum. In the initial stages of the disease, the ventilation function of the lungs is not disturbed. However, as the process worsens, panting begins, hypoxia and hypercapnia occur, which lead to the onset of cyanosis, which is not well saturated with oxygen in the blood. At the same time, chronic hypoxia causes irreversible vasoconstriction in the lungs, and the lung tissue causes heart failure (cor pulmonum). Along with cyanosis, right heart failure may begin, and sneezes may appear in the body.

Vesicular pulmonary emphysema. According to modern concepts, vesicular pulmonary emphysema is an organic lung disease characterized by a sharp expansion of air-conducting lung structures distal to the terminal bronchioles. This disease continues with the destruction of the barriers between the alveoli. Sudden swelling of the lungs should be distinguished from vesicular emphysema. When the lungs are at rest and suddenly inflated, the volume of the air spaces in the respiratory section of the lungs increases due to the elasticity of the walls of the alveoli without signs of destruction. In emphysema, four main types of emphysema are distinguished depending on the location of the disease process: 1) centroacinar (centrolobular), 2) panacinar, 3) periacinar (paraseptal, subpleural), 4) irregular (perifocal, next to the scar) emphysema. This classification reflects that the structural-functional unit of the lung respiratory department of the process is located within the working acinus of the department. Acinus consists of respiratory bronchioles, alveolar ducts and alveoli of all orders.

Centrolobular (centroacinar) emphysema is characterized by the exacerbation of the disease in the central part of the acinus, that is, in the respiratory bronchioles. At the same time, in the early stages of the disease, the distal sections of the acinus remain intact, that is, undamaged. As the process worsens, the distal alveoli expand and their walls erode. For this type of emphysema, it is typical that the process is stronger in the upper parts of the lungs. Centrolobular emphysema is often observed in men, smokers, and sometimes non-smokers. In panacinar emphysema, the entire acinus is damaged, and the alveoli and alveolar ducts become equally enlarged. In this case, the walls of the alveoli are also eroded. As the process progresses, it becomes diffuse in nature. Panacinar emphysema is thought to be caused by a genetic deficiency of the inhibitory substance alpha-1-antitrypsin. This type of emphysema is more common in older people.

Periacinar (paraseptal or subpleural) emphysema is characterized by damage to the peripheral parts of the acinar wall. The process is located in the subpleural area and along the septa between the lung lobes (Fig. 37). This type of emphysema is limited in nature and rarely changes lung function. This type of emphysema sometimes causes the appearance of bullous bubbles directly under the pleura, such bubbles can burst into the pleural cavity and cause pneumothorax in young people. Of the four types of emphysema described above, centrolobular emphysema and panacinar emphysema, which are based on chronic obstructive pulmonary disease, are more common. This type of emphysema, especially at the beginning of centrolobular emphysema, bronchitis and bronchiolitis, smoking cigarettes is also important.

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