22. HEPATOPULMONARY SYNDROME.

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Introduction. Nowadays hepatopulmonary syndrome (HPS) is a common and important cause of lung disease for the patients with cirrhosis of the liver. Extrahepatic complications include changes in many systems and organs and can be considered the multiorgan dysfunction syndrome. The most common complications in hepatopulmonary syndrome are: the complications of portal, splanchnic, and systemic hemodynamics in liver cirrhosis which are related to inflammatory and fibrotic processes in the liver, as well as intestinal bacterial translocation.

Aim of study. Highlighting the pathogenesis of hepato-pulmonary syndrome. Identification of the main and also alternative causes that occur in hepato-pulmonary syndrome.

Methods and materials. This review represents an analysis of actual information about hepatopulmonary syndrome from online biomedical sources, found with the research motors PubMed, Google Scholar, etc.

Results. From the pathophysiological perspective, the characteristic feature for the hepato-pulmonary syndrome is the remarkable dilation of the precapillary and capillary vessels, in association with the increased number of intrapulmonary vessels that suffer from these dimensional changes. The appearance of intrapulmonary arterio-venous communications and the development of porto-pulmonary anastomoses explain the symptoms of hepato-pulmonary syndrome. Another change which is observed in patients with cirrhosis complicated by the hepato-pulmonary syndrome is the absence of intrapulmonary vasoconstriction in response to the stimulus represented by hypoxemia, the so-called Euler reflex. A considerable role in the pathogenesis of the development of hepato-pulmonary syndrome and porto-pulmonary hypertension is attributed to an altered balance between vasodilator (Nitric Oxide) and vasoconstrictor (Endothelin 1) intrapulmonary substances, the main tools involved being macrophages, vascular endothelium and vascular smooth muscle. Proinflammatory cytokines are thought to play a key role in its occurrence. The studies have shown that endothelin-1 and its interaction with tumour necrosis factor (TNFα) from the pulmonary vessels contribute to the development of HPS. Another source of TNFα production is caused by stimulation of the endotoxin located in the Kupffer cells in the affected liver. The ascension of the diaphragm in ascites cirrhosis and the possible presence of a pleural collection also contribute to the deterioration of the lung function.

Conclusion. Knowing the mechanisms in development of complications in liver cirrhosis is essential in choosing the vasoactive pharmacological option with specific effects on fibrogenesis and portal hypertension syndrome.