CHANGES IN THE SMALL INTESTINE IN PULMONARY FIBROSIS. THE BODY'S RESPONSE TO EXPERIMENTAL PULMONARY FIBROSIS. THE RESULT OF THE EXPERIMENTS

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Abstract

In this review, we discuss three broad areas which have been explored that may be responsible for the combination of altered lung fibroblasts, loss of alveolar epithelial cells, and excessive accumulation of ECM: inflammation and immune mechanisms, oxidative stress and oxidative signaling, and procoagulant mechanisms.

The goal - Pulmonary fibrosis is a chronic lung disease characterized by excessive accumulation of extracellular matrix (ECM) and remodeling of the lung architecture. Idiopathic pulmonary fibrosis is considered the most common and severe form of the disease, with a median survival of approximately three years and no proven effective therapy. Despite the fact that effective treatments are absent and the precise mechanisms that drive fibrosis in most patients remain incompletely understood, an extensive body of scientific literature regarding pulmonary fibrosis has accumulated over the past 35 years. In this review, we discuss three broad areas which have been explored that may be responsible for the combination of altered lung fibroblasts, loss of alveolar epithelial cells, and excessive accumulation of ECM: inflammation and immune mechanisms, oxidative stress and oxidative signaling, and procoagulant mechanisms. We discuss each of these processes separately to facilitate clarity, but certainly significant interplay will occur amongst these pathways in patients with this disease.

Pulmonary fibrosis is a chronic lung disease characterized pathologically by excessive accumulation of extracellular matrix (ECM) and remodeling of the lung architecture, and additionally characterized by recognizable clinical, physiologic, and radiographic findings. Though some descriptions of fibrous diseases of the lungs can be found as early as the 5th century BC by Hippocrates , more modern descriptions of pulmonary fibrosis occurred in the early part of the 20th century with reports by Hamman and Rich of four patients with rapidly progressive diffuse interstitial fibrosis of the lungs . Although the prognosis of patients with diffuse pulmonary fibrosis is poor, it was subsequently realized that many patients did not have the extremely rapid deteriorating course that was described by Hamman and Rich. With further pathologic analysis, several distinct types of pulmonary fibrosis were described, and the terms diffuse fibrosing alveolitis, diffuse interstitial fibrosis, and idiopathic pulmonary fibrosis (IPF) were introduced to describe a more insidious, yet still debilitating form of chronic pulmonary fibrosis.

Material and methods. A total of 102 women of fertile age (19-49 years old) living in cities and villages of Bukhara region were involved in the research. All of them became those who were treated in the gynecology department of the Bukhara City maternity complex. Healthy women were included in the control group, who were made sure that no symptoms of (IDSPO) were observed in the last 6 months.

Currently, IPF is considered the most common and severe form of pulmonary fibrosis, with a disheartening median survival of approximately three years, with no proven effective therapy, and with lung transplantation remaining the only viable intervention in end-stage disease.

Keywords: Pulmonary fibrosis, immune system, idiopathic pulmonary fibrosis

Conclusions

In addition to altered mesenchymal cells, abnormalities of the alveolar epithelium in patients with pulmonary fibrosis have been noted from the earliest descriptions of the disease process.

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