World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-17, December 2022
ISSN: 2749-3644

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

Hasanova D.A
Barnoev A.I
1 Bukhara State Medical Institute named after Abu Ali ibn Sino, Republic of Uzbekistan, Bukhara,

Article history:

Received: October 20th 2022
Accepted: November 20th 2022
Published: December 28th 2022

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. We discuss each of these processes separately to facilitate clarity, but certainly significant interplay will occur amongst these pathways in patients with this disease.

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.

Conclusions. The amount of leukocytes in the 1-2 days when the organism of patients women observed in the (IDSPO) fell to the stasionar in the immune system was convincingly higher than the indicators of the control group, a decrease in the relative indicators of lymphocytes was observed.

Keywords: Pulmonary fibrosis, immune system, idiopathic pulmonary fibrosis

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. 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 .

The pathologic findings in pulmonary fibrosis (excessive accumulation of ECM and remodeling of the lung architecture) are a consequence of disturbances in two physiologically balanced processes: proliferation and apoptosis of fibroblasts, and accumulation and breakdown of ECM. When the normal balance between ECM deposition and turnover is shifted toward deposition or away from breakdown, excessive ECM accumulates. When the balance between fibroblast

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-17, December 2022
ISSN: 2749-3644

proliferation and apoptosis is shifted toward accelerated proliferation or slowed apoptosis, fibroblasts - the primary ECM producers - accumulate. Several possible origins of ECM-producing mesenchymal cells have been described, and have included accumulation of resident lung fibroblasts, homing and fibroblastic differentiation of bone marrow-derived cells such as circulating fibrocytes or monocytes , or epithelial-mesenchymal transition (EMT). Independent of the source of fibroblast expansion in the lungs (resident or systemic), it seems agreed upon that the ultimate effector cell in pulmonary fibrosis is the myofibroblast, a differentiated fibroblast which has contractile properties similar to smooth muscle cells, and which is characterized by the presence of alpha-smooth muscle actin (α-SMA).

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. Loss of normal type I alveolar epithelium and replacement by hyperplastic type II cells or bronchiolar cuboidal cells is a consistent finding in patients with IPF. In addition to these observations, more recent mechanistic studies have focused on the interplay, or cross-talk, between damaged epithelial cells and lung mesenchymal cells. This epithelial-mesenchymal interplay lends support to a key theme in pulmonary fibrosis, in which altered lung mesenchymal cells coupled with alveolar epithelial cell injury result in the accumulation of ECM and remodeling of the lung architecture.

REFERENCES:

1. Takishima T, Shimura S. In: *Basic and clinical aspects of pulmonary fibrosis.* Takishima T, editor. CRC Press, Boca Raton, FL; 1994. Definition and Classification of Pulmonary Fibrosis; pp. 293–303.
2. Orr CR, Jacobs WF. Pulmonary Fibrosis. *Radiology.* 1926;**7**:318–325.
3. Hamman L, Rich AR. Fulminating diffuse interstitial fibrosis of the lungs. *Trans Am Clin Climatol Assoc.* 1935;**51**:154–163
4. Hamman L, Rich A. Acute diffuse interstitial fibrosis of the lungs. *Bull Johns Hopkins Hosp.* 1944;**74**:177–212.
5. Scadding JG, Hinson KF. Diffuse fibrosing alveolitis (diffuse interstitial fibrosis of the lungs). Correlation of histology at biopsy with prognosis. *Thorax.* 1967;**22**:291–304.
6. Crystal RG, Fulmer JD, Roberts WC, Moss ML, Line BR, Reynolds HY. Idiopathic pulmonary fibrosis. Clinical, histologic, radiographic, physiologic, scintigraphic, cytologic, and biochemical aspects. *Ann Intern Med.* 1976;**85**:769–788.
7. NA.Narzieva, N.Hasanova Communicative competence as a pedagogical modelin the classrooms, ACADEMICIA: An international Multidisciplinary Research Journal, volume 10(6),78-81,2020
8. NA Narzieva The concept of defined target technologies and their role in the educational process, Theoretical and Applied science, 2020
9. NA Narzieva. The concept of defined target technologies and their role in the educational process// Theoretical &Applied science, 356-360, 2020
10. NN Atakulovna FACTORS SUPPORTING TEACHING AND LEARNING ENGLISH IN NON-ENGLISH SPEAKING COUNTRIES, ResearchJet Journal of Analysis and Inventions, 2021
11. NN Atakulovna Teaching Vocabulary by Using Digital Technology to Non-Native Learners, " ONLINE-CONFERENCES" PLATFORM, 2021
12. NA Narzieva, ORGANIZING ENGLISH CLASSES REGARDING LEARNERS WISHES, Scientific progress, 2021
13. *Narzieva Nilufar Atakulovna, Teaching ESP to dentistry students, Web of Scientist: International Scientific Research Journal, 2022*