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

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Abstract
From the earliest descriptions of patients with pulmonary fibrosis, cellular inflammation in the lung parenchyma has been a consistent pathologic finding . Histologic analysis has shown varied accumulations of lymphocytes, macrophages, plasma cells, eosinophils and neutrophils, and the presence of lymphoid follicles with germinal centers has been observed in many patients in the lung interstitium . The initial terms ‘diffuse fibrosing alveolitis’ and ‘cryptogenic fibrosing alveolitis’ were used as a reflection of the inflammatory component of the pathologic process in pulmonary fibrosis , and most patients with usual interstitial pneumonia (UIP), the pathologic hallmark of IPF, will manifest a mild to moderate degree of chronic cellular inflammation in the lung.

Keywords: Pulmonary fibrosis, immune system, idiopathic pulmonary fibrosis.

Over the past 15 to 20 years, however, the role of inflammation has been questioned, and the hypothesis has been put forward that active cellular lung inflammation is not a major feature or requirement for the development of IPF. Recent pathologic descriptions of patients with UIP have emphasized the epithelial, mesenchymal, and ECM abnormalities while de-emphasizing the cellular inflammatory features. This paradigm has thus shifted emphasis in IPF towards a process of epithelial cell injury coupled with exaggerated wound healing, and inflammation has been relegated to a mechanistically less important and bystander role in the fibrotic process. Probably the major supporting argument for inflammation playing a non-causal role stems from the observation that anti-inflammatory therapies, particularly corticosteroids, have been uniformly ineffective in improving pulmonary function or survival in patients with IPF.
However, even though the role of inflammation in IPF has been recently de-emphasized, the original findings of cellular inflammation in the lung have been supplemented with an extensive accumulation of scientific studies which have implicated numerous inflammation-related cytokines and cell surface molecules in profibrotic mechanisms. It is also important to note that resistance to corticosteroids does not necessarily indicate or equate with a lack of inflammatory involvement, as there are several well-established diseases in which inflammation is clearly accepted to be the predominant underlying mechanism, but in which traditional anti-inflammatory therapy has been poorly effective. Stemming from the observations on inflammatory cells, cytokines, chemokines, and cell surface molecules, the inflammation hypothesis has dominated the field of pulmonary fibrosis for nearly four decades, and IPF

continues to be viewed by many authorities as a chronic inflammatory disease of the lung parenchyma.

It is difficult if not impossible to comprehensively review the evidence in support of the role of inflammation in pulmonary fibrosis. Such evidence is abundant from mechanistic studies in cell culture, experimental research in animals, and observations in human patients. Below, we provide an overview of such evidence and lines of thought, in hopes to provide a springboard for continued independent studies in the field.

CoOver the past 40 years of research into mechanisms of pulmonary fibrosis, an immense amount of literature has described alterations in cytokine expression and function in animals and patients with pulmonary fibrosis. Most of the findings have described a propensity for a variety of cytokines to promote and enhance the fibrotic process, whereas in some instances, up-regulation of a particular cytokine is associated with inhibition of fibrosis. The potential sources of these cytokines in the lung are numerous, and include resident or systemic epithelial, mesenchymal, or inflammatory cells (T lymphocytes, B lymphocytes, macrophages, neutrophils, eosinophils, and platelets).

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.

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