RA-ILD: Pathogenesis and the Promise of Antifibrotics

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Rheumatoid arthritis- associated interstitial lung disease (RA-ILD) is one of the most important extra-articular manifestations of RA, and it contributes to considerable morbidity and mortality. The pathogenesis of RA-ILD is complex, because it involves chronic systemic inflammation, aberrant immune responses, and progressive fibrotic remodeling of the lung. And genetic susceptibility, environmental exposures such as smoking, and autoimmunity are thought to lead to epithelial injury, dysregulated repair and excessive fibroblast activation.

Recently, there has been growing interest in the use of antifibrotics such as nintedanib and pirfenidone in RA-ILD. The results of clinical trials and real- world studies suggest that antifibrotics may slow the rate of lung function decline and stabilize disease progression, especially in patients with fibrotic phenotypes. Although immunosuppressive agents remain the mainstay for inflammatory components of RA-ILD, the addition of antifibrotics can be a promising strategy to control the fibrotic component of RA-ILD. In the use of antifibrotics in RA-ILD, defining optimal patient selection, deciding the timing of initiation and combining strategies with conventional disease-modifying antirheumatic drugs and biologics are still challenging. Understanding the interplay between immune-mediated inflammation and fibrosis will be important in the treatment of RA-ILD.

In this lecture, I will review the knowledge of RA-ILD pathogenesis and the evidence on antifibrotics as a therapeutic option in RA-ILD.