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The role of antifibrotic agents in the management of pulmonary fibrosis: Current evidence and future directions.

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Introduction

Pulmonary fibrosis, particularly idiopathic pulmonary fibrosis (IPF), is characterized by chronic inflammation and irreversible scarring of the lung parenchyma. The disease has a poor prognosis, with a median survival of 3–5 years' post-diagnosis. Until recently, treatment options were limited to supportive care and lung transplantation. However, the development of antifibrotic agents has shifted the therapeutic landscape, offering new hope for patients [1].

Fibrosis in the lungs results from aberrant wound healing following alveolar epithelial cell injury. This leads to the activation of fibroblasts and myofibroblasts, excessive extracellular matrix deposition, and tissue remodeling. Antifibrotic agents aim to interrupt these processes, thereby slowing disease progression. Unlike anti-inflammatory drugs, antifibrotics directly target fibrogenesis, making them more suitable for managing fibrotic lung diseases [2].

Pirfenidone is a pyridone derivative that exhibits antifibrotic, anti-inflammatory, and antioxidant properties. It inhibits the synthesis of transforming growth factor-beta (TGF-β), a central mediator in fibrosis. Clinical trials such as CAPACITY and ASCEND have demonstrated that pirfenidone slows the decline in forced vital capacity (FVC) and improves progression-free survival in IPF patients. It has been approved in multiple countries and is considered a first-line therapy [3].

Nintedanib is a tyrosine kinase inhibitor that targets receptors involved in fibroblast proliferation, including platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), and fibroblast growth factor (FGF). The INPULSIS trials showed that nintedanib significantly reduces the annual rate of FVC decline in patients with IPF. It has also shown efficacy in other fibrosing interstitial lung diseases (ILDs), broadening its clinical utility [4].

Both pirfenidone and nintedanib are generally well tolerated but come with side effects. Pirfenidone is associated with gastrointestinal symptoms, photosensitivity, and liver enzyme elevations, whereas nintedanib commonly causes diarrhea, nausea, and hepatotoxicity. Dose adjustment and close monitoring are essential to minimize adverse events and enhance patient adherence [5].

Conclusion

Antifibrotic agents such as pirfenidone and nintedanib have transformed the management of pulmonary fibrosis by slowing disease progression and improving quality of life. As research advances, the development of new agents and combination therapies holds promise for more effective, personalized care. Addressing challenges related to diagnosis, access, and long-term management will be key to optimizing outcomes for patients with fibrosing lung diseases.

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