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by 故人

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INTRODUCTION

Coronavirus Disease 2019 (COVID-19) is a respiratory illness caused by the SARS-CoV-2 virus. It has been observed that COVID-19 patients may develop pulmonary fibrosis (PF), a serious complication that affects their quality of life even after recovery. PF is characterized by damage to the lung tissue, excessive scarring, and impaired lung function. This often leads to respiratory failure and can be fatal. It is important to explore pharmacotherapy interventions that can prevent or reduce fibrosis damage in COVID-19 patients.

PATHOGENESIS OF COVID-19-INDUCED PF

The pathogenesis of COVID-19-induced PF is complex and involves various molecular mechanisms. Transforming growth factor-beta (TGF- β) and phosphatidylinositol 3-kinase (PI3K)/AKT signaling pathways play important roles in the development of PF. The activation of these pathways leads to fibroblast proliferation, migration, and conversion into myofibroblasts, resulting in excessive scarring. Cytokine storm, characterized by an overactive immune response, also contributes to the activation, proliferation, and migration of fibroblasts. Additionally, the epidermal growth factor receptor (EGFR) pathway has been implicated in COVID-19-related PF.

PHARMACOTHERAPY INTERVENTIONS

MEDICATIONS COMMONLY USED IN CLINICAL TREATMENT

Pirfenidone and nintedanib are currently approved for the treatment of idiopathic pulmonary fibrosis (IPF) and have shown efficacy in COVID-19-induced PF. Pirfenidone inhibits fibroblast proliferation and extracellular matrix deposition, while nintedanib slows down the development of fibrosis. Both

medications have similar efficacy in reducing lung function decline. However, pirfenidone may cause liver injury, and nintedanib is not recommended for patients with moderate or severe liver injury.

MEDICATIONS LESS COMMONLY USED IN CLINICAL TREATMENT

N-acetylcysteine (NAC) and mesenchymal stem cell (MSC) therapy have shown potential as adjuvant treatments for COVID-19-induced PF. NAC replenishes glutathione levels, and MSCs have anti-inflammatory and regenerative properties. DPP-4 inhibitors and statins, commonly used for diabetes and cholesterol management, respectively, may also prevent fibrosis. Anakinra, Xuanfei Baidu Decoction (a traditional Chinese medicine), nimotuzumab, nanoceria, and vitamin D supplementation have shown promising results in treating COVID-19-induced PF.

POTENTIAL MEDICATION THERAPIES WITH LIMITED EVIDENCE

Several medications, such as natural polysaccharides, baicalin, the endocannabinoid system, dihydroartemisinin, and EGCG, have shown anti-fibrotic effects in preclinical trials but lack clinical trial dates. Tocilizumab and baricitinib combination therapy has shown effectiveness but has controversial safety concerns. PI3K inhibitors hold promise but require further exploration for safe use.

CONCLUSION

Pulmonary fibrosis (PF) remains a challenging problem without a cure. Pharmacotherapy interventions aimed at delaying disease progression and improving quality of life are crucial. Pirfenidone and nintedanib are currently the mainstay of treatment, while other medications serve as potential adjuvant

therapies. Rational use of DPP-4 inhibitors, statins, NAC, anakinra, vitamin D, nintedanib, and nintedanib may prevent or control the progression of COVID-19-induced PF. Traditional Chinese medicine and other experimental medications require further research and clinical trials to evaluate their efficacy and safety. Continued studies and follow-up are necessary to explore anti-fibrosis pharmacotherapy interventions for COVID-19-induced PF.

ACKNOWLEDGEMENTS

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