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Combination Treatment for Aerovascular Hypertension Targeting the Nitric Oxide and Prostacyclin Pathways

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INTRODUCTION

Pulmonary arterial hypertension is a rare condition characterized by detrimental remodeling of the arterial tree leading to increased vascular resistance, resulting in increased right ventricular afterload and ultimately the development of heart failure. Nonspecific clinical manifestations and lack of knowledge of pathology lead to a poor prognosis, delaying diagnosis and initiation of treatment. In recent years, improvements in diagnostic procedures, the emergence of new specific therapies, and the creation of referral units dedicated to this condition have significantly improved the prognosis.

DESCRIPTION

Therefore, the opportunities for drug-drug interactions between pulmonary arterial hypertension targeted drugs and drugs that may be used to treat comorbidities increase. This review provides an overview of drug metabolism by cytochrome and describes important drug-drug interactions of Food and Drug Administration-approved drugs for his pulmonary arterial hypertensions in the nitric oxide, endothelin, and prostacyclin pathways. Among the nitric oxide pathway targets, critical interactions with nitrates, protease inhibitors, and other phosphodiesterase inhibitors can cause severe hypotension. In the endothelin pathway, bosentan is associated with more drug-drug interactions through inhibition. The interaction between macitentan and ambrisentan is less important.

Pulmonary arterial hypertension is further divided into subgroups based on underlying etiology, consisting of idiopathic Pulmonary hypertension, heritable Pulmonary hypertension, drug and toxin-associated Pulmonary hypertension, pulmonary veno-occlusive disease, Pulmonary hypertension in long-term responders to calcium channel blockers, and persistent PH of the newborn, as well as Pulmonary hypertension associated with other medical conditions including connective tissue disease, HIV, and congenital heart disease. Early symptoms are nonspecific and the typically consist of dyspnea and fatigue on exertion. Currently approved treatments for the pulmonary hypertension consist of drugs that potentiate agonists of the nitric oxide-cyclic guanosine monophosphate biological pathway, antagonists of the prostacyclin pathway, and the endothelin pathway. Current treatment consists of combination drug therapies targeting multiple biological pathways, Nitric oxide cyclic guanosine monophosphate and endothelin metabolic pathways, demonstrating clear improvements in morbidity and mortality compared with previous conventional single-pathway targeted monotherapies. Pulmonary arterial hypertension is a chronic progressive disease characterized by the vascular remodeling of the small pulmonary arteries, leading to increased pulmonary vascular resistance and eventual right ventricular failure. Combination therapy of pulmonary arterial hypertension, either proactively or sequentially, has become a widely used therapeutic strategy or can simultaneously target multiple of these signaling pathways involved in disease progression. Much of the current therapeutic landscape focuses on first-line combinations of ambrisentan and tadalafil, endothelin receptor antagonists and phosphodiesterase-5 inhibitors [1-4].

CONCLUSION

As a result, for patients with pulmonary arterial hypertension, clinicians often consider combination therapy with other drugs or drug classes that may be clinically appropriate. Although prospective data are lacking, preclinical data and results from secondary data analyses of clinical trials targeting these pathways suggest that, as a reasonable and sometimes preferred alternative approach to combination therapy in pulmonary arterial hypertension, this alternative combination therapy may provide new insights. This overview of preclinical and clinical data describes our current understanding of combination therapies that simultaneously target the nitric oxide and prostacyclin pathways and highlights the clinical benefits and theoreti-

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cal biochemical interactions of these agents.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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