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Linking Exercise-Induced Pulmonary Hypertension with Increased Cardiovascular Risk in Scleroderma

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Introduction

Scleroderma, or Systemic Sclerosis (SSc), is a rare, complex autoimmune disease characterized by fibrosis and vascular abnormalities that can affect multiple organ systems, including the skin, lungs, kidneys, and cardiovascular system. It is a heterogeneous condition, often marked by progressive tissue hardening, vascular occlusion, and, in severe cases, organ failure. One of the most life-threatening complications of scleroderma is Pulmonary Hypertension (PH), particularly Exercise-Induced Pulmonary Hypertension (eiPH), which has garnered increased attention due to its association with worsened cardiovascular outcomes.

Pulmonary hypertension refers to a condition characterized by elevated pressure in the pulmonary arteries, which can lead to right heart failure and significantly compromise overall cardiovascular health. While scleroderma-related pulmonary hypertension is well-documented, less attention has been given to its specific manifestation during physical activity. Exercise-induced Pulmonary Hypertension (eiPH) is a phenomenon in which an increase in pulmonary arterial pressure occurs during exercise, even in the absence of resting PH. This condition may be particularly significant for individuals with scleroderma, as exercise, typically beneficial for cardiovascular health, could exacerbate existing vascular and cardiac dysfunction, thus increasing the overall cardiovascular risk [1].

Description

Scleroderma is a chronic autoimmune disease that primarily affects the skin, resulting in thickening and hardening. However, it can also involve internal organs, leading to severe complications. The disease is often classified into two types: Limited Cutaneous Systemic Sclerosis (lcSSc) and diffuse cutaneous Systemic Sclerosis (dcSSc), each with distinct clinical features and progression. The cardiovascular system is one of the primary targets in scleroderma, and individuals with this disease are at a heightened risk for various cardiac and vascular complications. Cardiac involvement in scleroderma is multifaceted and can manifest as arrhythmias, pericarditis, myocardial fibrosis, and heart failure. However, pulmonary hypertension (PH), which is characterized by elevated pressure in the pulmonary arteries, is the most significant and common cardiovascular complication in scleroderma. In fact, PH is the leading cause of morbidity and mortality in patients with systemic sclerosis, with estimates suggesting that approximately 10-15% of individuals with SSc develop clinically significant pulmonary hypertension. Pulmonary hypertension in scleroderma can be secondary to pulmonary interstitial fibrosis (pulmonary arterial hypertension or PAH), or it can arise independently as a result of vascular remodeling and endothelial dysfunction [2].

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Exercise-induced pulmonary hypertension refers to a condition in which the pulmonary arteries experience abnormal pressure increases during physical activity. Unlike resting PH, which can be detected at baseline, eiPH is only apparent during stress, such as exercise or physical exertion. This phenomenon is particularly concerning in patients with scleroderma, as it indicates underlying dysfunction of the pulmonary vasculature and right ventricle, which may not be evident under resting conditions. In scleroderma, vascular abnormalities are a hallmark feature. Endothelial dysfunction leads to an imbalance between vasoconstrictor and vasodilator substances, impairing the normal response to physical exertion. Vascular remodeling, characterized by smooth muscle hypertrophy, intimal thickening, and fibrosis of the pulmonary arteries, results in an increased vascular resistance. As a result, during exercise, the pulmonary vasculature struggles to accommodate the increased cardiac output, leading to elevated pulmonary pressures. The right ventricle (RV) is responsible for pumping blood through the pulmonary circulation. In scleroderma, both the pulmonary vasculature and the right ventricle may be compromised. RV dysfunction, characterized by reduced contractility and impaired diastolic filling, limits the heart's ability to meet the demands of exercise. This can lead to exercise-induced right heart failure, contributing to the development of pulmonary hypertension during physical activity. Scleroderma often leads to microvascular changes that affect the pulmonary capillary bed. These changes reduce the ability of the pulmonary vasculature to dilate and accommodate increased blood flow during exercise. This microvascular dysfunction further contributes to elevated pulmonary pressures during physical exertion [3].

The presence of exercise-induced pulmonary hypertension in scleroderma poses significant clinical challenges, as it may lead to early cardiovascular decline in patients who otherwise appear stable. Patients with scleroderma who develop pulmonary hypertension-whether resting or exercise-induced-are at an increased risk of mortality. The combination of PH and impaired right ventricular function can accelerate the progression to heart failure with preserved ejection fraction (HFpEF) or right heart failure, both of which significantly reduce life expectancy. Exercise-induced PH further complicates this scenario, as it may be indicative of early-stage right ventricular dysfunction that could eventually progress to overt heart failure. Exercise intolerance is a common symptom in scleroderma, affecting up to 70% of patients. The development of exercise-induced pulmonary hypertension exacerbates this problem, as patients may experience disproportionate dyspnea, fatigue, and chest discomfort during physical exertion. This limitation in exercise capacity negatively impacts their overall quality of life and contributes to the development of deconditioning, obesity, and other comorbid conditions. The heart in scleroderma is often affected by fibrosis and inflammation, leading to diastolic dysfunction and arrhythmias. Exercise-induced pulmonary hypertension further stresses the cardiovascular system by imposing increased workload on both the left and right ventricles. Over time, this added strain can worsen pre-existing cardiac dysfunction and contribute to the development of congestive heart failure or arrhythmogenic complications [4,5].

Conclusion

Exercise-induced pulmonary hypertension in scleroderma represents a critical challenge in managing cardiovascular risk in these patients. The interplay between pulmonary vascular changes and right heart strain during physical activity contributes to a worsening cardiovascular prognosis. Early

recognition of EIPH through diagnostic tools such as cardiopulmonary exercise testing and exercise echocardiography is essential for preventing the progression of cardiovascular complications. By understanding the pathophysiology of exercise-induced pulmonary hypertension in scleroderma, healthcare providers can better manage the cardiovascular health of these patients, offering pharmacological treatments, exercise regimens, and other interventions aimed at reducing the burden on the heart and improving overall outcomes. With appropriate monitoring and tailored management strategies, patients with scleroderma and pulmonary hypertension can experience improved exercise capacity, better quality of life, and reduced cardiovascular risk.

Acknowledgement

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Conflict of Interest

None.

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