Short Communication

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Exercises for Pulmonary Arterial Hypertension: Friend or Foe?

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Abstract

Pulmonary arterial hypertension (PAH) is a group of disorders defined by a progressive elevation of pulmonary vascular resistance in the small pulmonary arteries and arterioles. The objective of this short communication was to assimilate the evidence for exercises in pulmonary arterial hypertension through an informed overview of published reviews in PubMed. Evidence evolved from studies on exercise-induced pulmonary hypertension to exercise testing in PAH and more recently, exercise training and its proposed beneficial effects in people with PAH. There is need for more studies in this area, and the limited evidence is too insufficient to draw conclusive effects.

Keywords: Pulmonary Arterial Hypertension; Cardio-Vascular Rehabilitation; Exercise Training; Exercise Rehabilitation.

Pulmonary arterial hypertension (PAH) is a group of disorders defined by a progressive elevation of pulmonary vascular resistance in the small pulmonary arteries and arterioles. The objective of this short communication was to assimilate the evidence for exercises in pulmonary arterial hypertension through an informed overview of published reviews in PubMed.

The pathophysiology of PAH was described by Waxman et al [1] as follows; "In pulmonary artery hypertension (PAH) vascular remodeling contributes to a sustained elevation of pulmonary vascular resistance (PVR) and pulmonary artery pressure (PAP) as a result of vascular remodeling characterized largely by vascular smooth muscle cell proliferation and medial hypertrophy, and endothelial cell proliferation resulting in lumen obliteration. The loss of pulmonary arterial compliance and development of elevated PVR puts an excessive burden on the right ventricle due to the increased workload necessary to overcome the downstream pressure, ultimately leading to right-sided heart failure."

Endothelial factors (NO and endothelin), neurohumoral factors (noradrenaline, acetylcholine), natriuretic peptides, reactive oxygen species and phosphodiesterase activity can influence resting pulmonary vascular tone (PVT), and alterations in PVT influences Pulmonary hypertension, thus resulting in blunted pulmonary vasodilatation response during exercise and impaired exercise capacity [2].

Exercise produces stress on the pulmonary circulation through increases in cardiac output (.Q) and left atrial pressure. Exercise has to be dynamic to avoid the increase in systemic vascular resistance and intrathoracic pressure that usually occur with resistive exercise. Recent studies suggest that exercise-induced increase in Pulmonary arterial pressure (PAP) to a mean higher than 30 mm Hg may be associated with dyspnea-fatigue symptomatology [3]. Recent studies had shown that exercise-induced increase in pulmonary artery pressure was associated with dyspnea-fatigue symptomatology, thus validating the notion of exercise-induced pulmonary hypertension [4].

Exercise testing in pulmonary arterial hypertension (PAH) provides important information on degree of this limitation, disease severity, and

prognosis. Together with ventilatory expired gas analysis, exercise testing acts as a valuable diagnostic tool. Emerging evidence indicates that exercise training was beneficial in patients with PAH [5].

Cardiopulmonary exercise testing (CPX) provides valuable diagnostic information in pulmonary arterial hypertension (PAH) and secondary pulmonary hypertension (PH) on the degree of ventilation/ perfusion mismatching secondary to altered pulmonary hemodynamics [6]. The practicing clinician uses various exercise modalities in evaluating and managing patients with PAH such as the 6-minute walk test (6MW), cardiopulmonary exercise testing, and exercise echocardiography. Exercise testing had diagnostic, therapeutic and prognostic implications based upon the nature of a patient's exercise limitation [7].

The functional capacity limitation in PAH is multifactorial and includes changes such as right heart failure, which leads to a limited increase in cardiac output during exercise, and hyperventilation with a reduced perfusion of properly ventilated alveoli. Arterial blood gas analysis abnormalities include hypoxemia and hypocapnia, related to an abnormal ventilation / perfusion match, gas diffusion abnormalities, low mixed venous oxygen saturation and to the development of intra- and extra-pulmonary right-to-left shunts. The 6-minute walking test for exercise tolerance, and functional capacity assessment by cardiopulmonary exercise test (CPET) estimates the severity of disease and assesses patients' prognosis and response to therapy [8].

Zhu et al [9] in their meta-analysis of seven trials on 768 patients that evaluated the effect of combination therapy on exercise capacity [measured with 6-minute walk distance (6MWD)] and clinical worsening in PAH patients. The combination therapy increased 6MWD by 21.59 meters and reduced the risk of clinical worsening compared to monotherapy.

Exercise training was a safe and effective method for treating patients with pulmonary arterial hypertension [10]. Evidence evolved from studies on exercise-induced pulmonary hypertension to exercise testing in PAH and more recently, exercise training and its proposed beneficial effects in people with PAH. There is need for more studies in this area, and the limited evidence is too insufficient to draw conclusive effects.

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