Change in Respiratory Function after Balloon Pulmonary Angioplasty in Patients with Chronic Thromboembolic Pulmonary Hypertension

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ABBREVIATIONS
BPA: Balloon Pulmonary Angioplasty; CTEPH: Chronic Thromboembolic Pulmonary Hypertension; PAP: Pulmonary Arterial Pressure; PEA: Pulmonary Endarterectomy; PH: Pulmonary Hypertension

Chronic thromboembolic pulmonary hypertension (CTEPH) is characterized hemodynamically by a mean pulmonary artery pressure (mPAP) of ≥ 25 mmHg along with a pulmonary artery wedge pressure (PAWP) of ≤ 15 mmHg at rest, and is classified as Group IV pulmonary hypertension (PH) [1]. CTEPH is caused by non-resolving fibro-thrombotic obstructions of large pulmonary arteries, combined with small vessel arteriopathy in some patients. Both proximal and small vessel obstruction increase pulmonary arterial pressure (PAP), leading to progressive PH, right heart failure and ultimately death [1,2]. Not all patients with CTEPH have a history of acute pulmonary embolism, and it has been reported that the incidence of CTEPH after pulmonary embolism is 0.4% to 8.8% [3,4]. In CTEPH, dyspnea and hypoxemia are the common presenting symptom and considered to be related to increased dead space ventilation caused by pulmonary vascular obstruction [5]. The dyspnea initially occurs during exertion only, but then progresses to occur at rest and becomes more severe, and usually more troubling to patients who are active [6]. Dyspnea and hypoxemia leads to impair exercise performance and are associated with reduced quality of life [7]. Continuous long-term oxygen therapy has been recommended, and it may benefit during daily physical activities and training [8].

The gold standard in the treatment of CTEPH is a surgical procedure: pulmonary endarterectomy (PEA), which significantly improves the prognosis and dyspnea in this group of patients [9-12]. However, < 60% of patients with CTEPH can undergo PEA, and many patients experience persistent hypoxemia and residual PH [13]. Balloon pulmonary angioplasty (BPA) is a novel method of treating inoperable or persistent CTEPH. BPA has been reported to improve hemodynamics and functional capacity in patients with CTEPH who are not candidates for PEA and residual or recurrent PH after PEA [14-17]. However, patients still suffered dyspnea and hypoxemia on effort even after BPA, despite remarkably improvement in resting hemodynamics [18].

Adaptation of perfusion to ventilation is an important feature of pulmonary physiology; ventilation and pulmonary blood flow in different lung fields vary at rest and during exercise. BPA changes the ventilation/perfusion (V/Q) ratio by improving pulmonary blood flow, and the effect of BPA on respiratory function may vary depending on the BPA fields, such as lower or upper/middle lung fields at

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rest and during exercise. Recently, some investigators additionally observed that effect of BPA on respiratory function in patients with CTEPH [19-21].

BPA in the lower lung field may improves oxygenation and respiratory function parameters during exercise, such as VE/VCO₂ slope, because of remarkable improvement in hemodynamics. Moreover, BPA in the upper-middle lung field may improve oxygenation and respiratory function parameters at rest, such as % diffusing capacity for carbon monoxide (DLco), cause by improvement in V/Q mismatch. These findings showed that the effect of BPA on respiratory function in patients with CTEPH differed depending on whether an intervention was to the lower or to upper-middle lung fields [19]. Aoki, et al. investigated how BPA affects hemodynamics, respiratory functions, and intrapulmonary shunt before and after the BPA [20]. BPA improved not only hemodynamics but also oxygenation with a resultant decrease in intrapulmonary shunt. Especially, the percentage of patients who required home oxygen therapy were significantly decreased after BPA (Home oxygen therapy; 79% (n = 19) to 54% (n = 13), p = 0.01).

In pulmonary function testing, Takei, et al. reported that vital capacity (VC), forced vital capacity (FVC), forced expiratory volume in 1s (FEV1), total lung capacity (TLC), and functional residual capacity (FRC) increased significantly after BPA [21]. Moreover, Changes in TLC, VC, FRC, and FVC were significantly correlated with changes in pulmonary vascular resistance (PVR). However, DLco did not change significantly. It was seemingly caused by throughout the duration of BPA with treatment to both upper and lower lung fields, consequently, DLco was considered to be counterbalanced [22].

Advances in BPA may demand more refined definitions of CTEPH and pulmonary artery abnormalities. For example, the distribution of intra-arterial lesions, lesion types, number of chronic total occlusions, upper/lower or proximal/distal distribution of lesions and diameter of pulmonary artery branches should be reported that might be associated with respiratory function in CTEPH patients. BPA may be effective for CTEPH patients with dyspnea and hypoxemia.

Future studies required to demonstrate the benefits of BPA on respiratory function in patients with CTEPH.

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