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Perspective

Pulmonary Angioplasty: Treatment for Thromboembolic Pulmonary Hypertension

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ABOUT THE STUDY

An innovative method for treating persistent thromboembolic pulmonary hypertension is Balloon Pulmonary Angioplasty (BPA). Cardiologists are familiar with the idea of balloon angioplasty, but BPA presents unique difficulties. BPA was first developed and established to treat congenital pulmonary stenosis in children. In 1988, it was subsequently used for the first time to treat Chronic Thromboembolic Pulmonary Hypertension (CTEPH). Although there was a significant mortality rate, the initial results of conducting BPA in inoperable CTEPH were described in 2001. The National Hospital Organization Okayama Medical Center in Japan, where the process was improved and smaller balloons were utilized, has been the primary source of reports on the effects of BPA since 2012. In addition, the numbers of balloon inflations per session were carefully limited to one or two lung vessel segments with targeting only one lung lobe during each session. Intravascular imaging was also introduced.

Acute Pulmonary Emboli (PE) that have been treated with anticoagulation for three months are thought to fail to disintegrate *via* autolysis, leading to the development of Chronic Thromboembolic Disease (CTED). When examined utilizing lung perfusion scintigraphy 3-12 months after an acute PE, residual abnormalities can be seen in up to 50% of patients. Anticoagulation or thrombolytic treatment cannot clear the thromboembolic material because it is replaced by fibrotic material, forms an endothelial coating, and cannot be removed. The incidence of pulmonary hypertension, despite the presence of remaining intravascular material, is reported to be less than 1% in clinical practice and 2-3% in the prospectively observed Pulmonary Embolism Thrombolysis (PEITHO) trial.

Chronic Thromboembolic Pulmonary Hypertension (CTEPH), which is frequently accompanied by severe effort limitation, is present when pulmonary pressures are elevated to abnormal levels in addition to CTED. This has been redefined as a Mean Pulmonary Artery Pressure (mPAP) of greater than 20 mmHg with a Pulmonary Vascular Resistance (PVR) of less than 3WU since the 6th World Symposium on Pulmonary Hypertension (WSPH). However, up until 2019, it was customary to identify pulmonary hypertension only in cases where the mPAP was below 25 mmHg.

The amount of obstructive material in CTEPH does not always correspond with pulmonary hypertension, which is instead made worse by a second vasculopathy that appears months to years after the first occurrence. When someone with CTEPH experiences an acute PE, their pulmonary hypertension typically does not go away and their dyspnea persists and worsens over time as the underlying vasculopathy worsens. There is no clear acute prior incident in 23% of instances. It is not yet known if this suggests a subclinical initial incident followed by a progressive vasculopathy.

Surgical pulmonary endarterectomy is the standard treatment for CTEPH (PEA). Technically possible in 60% of CTEPH patients, this has outstanding symptomatic, hemodynamic, and prognostic results. While the major centres now report a 30-day mortality of 2.2%, the international CTEPH registry has recorded an in-hospital mortality of roughly 5%. According to reports, operated patients have a three-year survival rate of 89%compared to unoperated patients' 70%. Only 55% of patients who were declared surgically operable but chose not to get treatment reached the age of 5 years, as opposed to 83% of those who had PEA.

The only approved treatment for CTEPH that has been demonstrated to improve 6-minute walk distance and moderately lower mPAP (10%) in patients with inoperable or post-PEA CTEPH is medicinal therapy with riociguat. The BAY63-2521 Long-term Extension Study in Patients with Chronic Thromboembolic Pulmonary Hypertension (CHEST-2) trial has long-term results that demonstrate prognostic advantage, with an estimated 93% 2-year survival rate among the 62% of the study group who continued medication for at least 2 years. In the multinational CTEPH registry, unapproved medicinal treatment (phosphodiesterase inhibitors and endothelin receptor antagonists) was not linked to increased survival.

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