Long-Term Efficacy of Treat and Repair Strategy for Atrial Septal Defect with Pulmonary Artery Hypertension

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Introduction: Therapeutic strategies for atrial septal defect (ASD) with pulmonary artery hypertension (PAH) are controversial. Recently, the efficacy of treat and repair strategy (PAH-specific medical therapy and transcatheter ASD closure) is introduced; however, the long-term effects remain unknown. The purpose of this study was to evaluate the long-term efficacy of the treat and repair strategy for ASD with significant PAH (mean pulmonary artery pressure (mPAP) >25 mmHg and PVR >3 wood units).

Methods: A total of 616 adult patients who underwent transcatheter ASD closure were divided into 3 groups: PAH/specific-medical therapy (n = 11), PAH/no-specific-medical therapy (n = 43), no-PAH (n = 562). The endpoint was defined as cardiovascular mortality and hospitalization for heart failure.

Results: Mean pulmonary artery pressure (PAP) before the treat and repair strategy was 56 ± 21 mmHg in PAH/specific-medical therapy group. Initially, the PHM group had higher PVR compared with non-PHM group (9.6±3.8 vs. 4.2±1.0 Wood units, P<0.01). After treatment with PAH-specific medications, PVR in this group decreased to 4.0±0.8 Wood units (P<0.01). During a median follow-up of 24 months (1-110 months), the event-free survival rate in PAH/specific-medical therapy group was inferior compared to that in no-PAH group (log-rank test, p < 0.01), however it was not different from that in PAH/no-specific-medical therapy group (p = 0.87). More than 90% of patients with PAH/specific-medical therapy had no cardiovascular events. In the PHM group, during a treatment period of 52±48 months, the World Health Organization Functional Classification significantly improved (3.0±0.5 to 2.0±0.0, P<0.01), as well as in the non-PHM group (2.1±0.6 to 1.5±0.5, P<0.01).

Conclusions: Treat and repair strategy for ASD with severe PAH can be considered as a safe and valuable therapeutic option even in patients complicated with significant PAH.