Significance of right ventricle function assessment and pulmonary vascular resistance after heart transplantation

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Introduction: To test the effect of pretransplant pulmonary vascular resistance (PVR) on posttransplant right ventricle (RV) size and function as well as to address the possible causes, except cardiomyopathy¹, affecting the RV function and its recovery trend after heart transplantation² (HTx). Also to compare invasive and echocardiographic RV function measurements and their impact on survival.

Patients and Methods: PreHTx right heart catheterization (RHC) as well as postHTx ECG, echo and RHC were performed in 84 adult heart transplant pts. RV longitudinal function was assessed by tricuspid annular plane systolic excursion (TAPSE). Data on PVR, transpulmonary gradient (TPG), RV stroke work index (RVSWI) as well as graft ischemic time (GIT), extracorporeal circulation time (ECCT) and time on mechanical ventilation were collected.

FIGURE 1. Tricuspid annular plane systolic excursion - TAPSE (in millimeters) in follow-up after heart transplantation. The most pronounced recovery is present during the second month after heart transplantation.

FIGURE 2. Systolic pulmonary artery pressure trend (by echocardiography) after heart transplantation. Significant decrease occurs during the first six months, with rise during the long-term follow-up.

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Results: The development of RBBB after HTx was not related with longer GIT or ECCT, and had no effect on posttransplant RV dilatation or function and survival. RV was dilated in 36% of pts in the 1st month after HTx with mean TAPSE 12 ± 4 mm, which increased to 16 ± 4 mm during the 2nd month. 63% of pts had reduced initial TAPSE with further recovery in only 44% (**Figure 1**). Longer GIT, ECCT and duration of mechanical ventilation had no influence on TAPSE or on the development of acute RV failure. Posttransplant RV function assessed both by TAPSE or RVSWI had no influence on survival. RVSWI was significantly reduced in early postHTx period ($5 \text{ gm}\cdot\text{m}/\text{m}^2$) but did not correlate with TAPSE. Pts with normal pretransplant PVR and TPG had similar posttransplant TAPSE and RVSWI in comparison to pts with reversibly increased PVR and TPG. PVR decreased for 43% after HTx (mean 203 ± 102 to 116 ± 45 dynes-sec-cm-5; $p < 0.001$). Systolic pulmonary arterial pressure assessed by echo was mildly to moderately elevated in early postHTx period (39 ± 11 mmHg) with trend to normalization within the first 6 months after HTx (**Figure 2**).

Conclusion: Posttransplant loss of the RV longitudinal systolic function was present in 60% of pts and recovered in almost half of them, but with no effect on patients' survival. Higher and reversibly elevated pretransplant PVR and TPG did not add to the more pronounced decrease in RV longitudinal function posttransplant. Interestingly, post-transplant RVSWI as an invasive measure of RV function did not correlate with TAPSE.

LITERATURE

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