

Improvement of Left Ventricular Strain with Reduction of Mean Pulmonary Arterial Pressure in Pulmonary Hypertension: Treatment Effect Independent of Right Ventricular Volumetric Parameters.

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Target audience: Researchers, radiologists, and clinicians imaging or treating patients with pulmonary hypertension.

Introduction: Pulmonary hypertension, defined as a mean pulmonary arterial pressure (mPAP) greater than 25mmHg, is classified into several categories (1). Some types of pulmonary hypertension, such as central-type chronic thromboembolic pulmonary hypertension (CTEPH), can be curatively treated by surgical intervention (pulmonary endarterectomy). Whereas, other types such as idiopathic pulmonary arterial hypertension and distal-type CTEPH are not indicative for surgery other than lung transplantation. Since recent advances of medical and/or catheter intervention improved prognosis of patients with inoperable pulmonary hypertension (2, 3), long-term follow-up is mandatory to such patients. It is reported that among cardiac MR parameters, a decreased stroke volume (SV), an increased right ventricular (RV) end-diastolic volume, and a decreased left ventricular (LV) end-diastolic volume measured at baseline are associated with a poor prognosis (1). Even if LV ejection fraction (LVEF) is preserved, decline of LV systolic strain is also associated with mortality of pulmonary arterial hypertension (4). Effective pulmonary thromboembolectomy improves LV strain of patients with CTEPH (5). However, it is not well known that whether there is ventricular interdependence in treatment effect on inoperable pulmonary hypertension or not.

Purpose: In this clinical study, we aimed to evaluate biventricular function and LV strain in inoperable pulmonary hypertension before and after treatment and correlate the data with right heart catheterization; we also evaluated ventricular interdependence regarding changes of parameters.

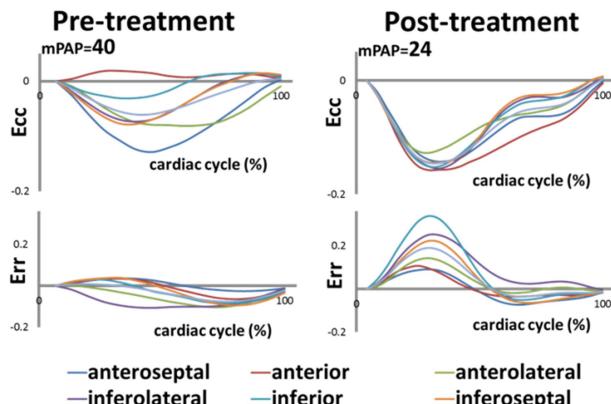
Materials and Methods: We enrolled 11 patients with distal-type CTEPH and one with idiopathic pulmonary arterial hypertension. The 11 patients with distal-type CTEPH were treated by pulmonary balloon angioplasty and medication such as vasodilators and anticoagulants. The patient with idiopathic pulmonary arterial hypertension was treated by medication. All patients underwent two MR examinations using a 3.0T whole body scanner (Trio A Tim System, Siemens Healthcare, Erlangen, Germany) before and after treatment (mean intervals 11.4 ± 6.2 month). Scan protocols included cine balanced steady-state free precession sequences in 2-, 3-, and 4-chamber views and a stack of short axis images in 20 cardiac phases per beat and short-axis cine-tagging imaging in mid LV. Cine-tagging imaging was acquired with the following parameters: ECG-triggered two-dimensional FLASH sequence with a resting grid pulse; TR/TE, 25 ms/ 3.85 ms; flip angle, 10° degrees; slice thickness, 8 mm; field of view, 320 mm; matrix size, 1.4x1.4x 8 mm ; tag grid, 6 mm; 13-25 cardiac phases/R-R interval on ECG.

Images were analyzed on a standalone workstation (ZioStation 2, Ziosoft, Tokyo, Japan). LV and RV volumes were analyzed on steady-state free precession cine images. Documented cine MR parameters included LVEF, LVSV, LV end-diastolic volume index (LVEDVI), LV end-systolic volume index (LVESVI), RVEF, RVSV, RVEDVI and RVESVI. LV strain analysis was performed on tagged images. Peak midwall circumferential strain (Ecc) and radial strain (Err) in anteroseptal, anterior, anterolateral, inferolateral, inferior and inferoseptal segments were analyzed.

Data are expressed as mean \pm standard deviation. Pearson's correlation coefficients (r) were used to compare between MR parameters and catheter-derived mPAP. Multivariate linear regression analysis was used to evaluate independent association of MR parameters and mPAP. $P < 0.05$ was used to designate statistical significance.

Results: Significant decrease in mPAP was observed after treatment (35.6 ± 10.2 to 27.4 ± 10.6 mmHg, $p=0.006$). Among MR-derived parameters, RVESVI ($r=0.633$, $p=0.036$) and RVEDVI ($r=0.603$, $p=0.049$) were significantly correlated with baseline mPAP. All patients demonstrated normal LVEF (64.9 ± 7.1 %) at baseline. None of LV function and strain parameters were significantly correlated with baseline mPAP. Change of peak Err in the inferolateral ($r=0.706$, $p=0.010$) and inferoseptal ($r=0.598$, $p=0.040$) segments were significantly correlated with change of mPAP. Changes of the following MR parameters tended to be correlated with mPAP demonstrating $p < 0.10$: RVEF, RVESVI and peak Ecc in the inferolateral segment. These 5 parameters were included in a multivariate regression model (stepwise method). The multivariate linear regression analysis revealed that changes of peak Err (coefficient for 10% increase, 4.08, 95% CI, 2.42, 5.72, $p=0.001$) and peak Ecc (coefficient for 10% increase, -8.41, 95% CI, -14.9, -1.89, $p=0.019$) in the inferolateral segment and RVESVI (coefficient for 10ml increase, -3.15, 95%CI, -4.40, -1.89, $p=0.019$) were independently associated with changes of mPAP after treatment.

LV strain before and after treatment



Discussion: Global LV function was preserved at baseline in our population. However, Err and Ecc in the inferolateral wall were improved after reduction of mPAP. Analysis of regional LV function is more sensitive to effective treatment than that of global function. Improvements of LV strain in the inferolateral segment and reduction of RVESVI occurred independently after treatment. These results indicate a need to monitor biventricular function in long-term management of patients with pulmonary hypertension. Larger longitudinal study including various types of precapillary pulmonary hypertension is needed to validate our preliminary results.

Conclusion: Changes of LV radial and circumferential strain in the inferolateral segment and RVESVI were independently correlated with change of mPAP in follow-up MR scans after effective medical and/or catheter intervention. LV strain as well as RV function analyses may be useful for management of patients with pulmonary hypertension.

References:

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