12. Measurement of Pulmonary Arteries by Cardiac Magnetic Resonance Imaging: A Simple and Useful Tool for the Detection of Pulmonary Hypertension in Systemic Sclerosis Patients without Overt Cardiac Microvascular Perfusion Defects or Fibrosis.

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Background/Purpose: Pulmonary hypertension (PH) is a major complication of systemic sclerosis (SSc). We observed that a majority of our SSc patients with PH as diagnosed by right heart catheterization (RHC) had PH due to left heart disease (LHD) (15/26, 58%). We hypothesized that LHD in these patients could be explained by cardiomyopathy secondary to fibrosis. The aim of our study was to detect fibrosis, as well as useful parameters for PH diagnosis, by cardiac magnetic resonance imaging (cMRI) in SSc patients with and without PH.

Methods: A retrospective analysis of our cohort of 432 SSc patients was performed. All patients routinely underwent screening for PH, and diagnosis of PH was proven by RHC in all suspected cases. Data from clinical, cardiopulmonary and serological investigations were analyzed. All living patients with PH (n=18) as well as a control group of 19 consecutive SSc patients without clinical suspicion of PH underwent a cMRI.

Results: Twenty-six SSc patients (26/432; 6%) had PH diagnosed by RHC. Eighteen of these patients (11 with PH due to LHD [61%], 4 with PAH [22%], 3 from other causes) and 19 without clinical suspicion of PH underwent cMRI. Age, disease duration, gender, ethnicity, disease subtypes and autoantibody profiles were similar between the two groups. Systolic pulmonary artery pressure by transthoracic echocardiography as well as DLCO were statistically significantly different between the two groups. Cardiac MRI showed statistically significant differences between SSc patients with and without PH, respectively, for the measurement of the diameter of the main pulmonary artery (PA) (30,90±5,03 mm vs 26,30±3,86 mm, p=0,006), the right PA (23,50±4,11 vs 18,60±2,90 mm, p=0,001), and the ratio of the main PA to the ascending aorta (0,97±0,10 vs 0,84±0,10, p=0,002). There was a trend toward significance for the measurement of the left PA (21,60±3,52 mm vs 19,80±1,98 mm, p=0,07). None of the 37 patients had significant myocardial hypersignal in T2 STIR nor delayed gadolinium enhancement.

Conclusion: Cardiac MRI did not show overt evidence of myocardial fibrosis to explain PH secondary to LHD in our SSc cohort. However, cMRI measurement of the diameter of the main PA, the right PA and possibly of the left PA, as well as the ratio of the main PA to the ascending aorta, seem to be simple and reliable methods for PH diagnosis in SSc patients, and may prove to be useful non-invasive tools in the investigation of PH.