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Meeting abstract

216 Beyond late gadolinium: ventricular dysfunction in adults with congenital heart disease is associated with diffuse myocardial fibrosis as shown by the volume of distribution of gadolinium

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Introduction

Heart failure is common in adult congenital heart disease (ACHD) and may be secondary to myocardial fibrosis.

Purpose

We sought to quantify diffuse fibrosis using the volume of distribution of Gadolinium (VdGd).

Methods

Consecutive ACHD patients (N = 15) were studied with cardiac MRI (Philips Achieva 3.0 Tesla) to quantify ventricular volume and function, as well as late gadolinium enhancement (LGE). T1 measurements were made with a Look-Locker technique, ie GRE sequence with a non-slice selective inversion pulse followed by segmented GRE acquisition for 20–24 cardiac phases. (temporal resolution 40 ms, TR/TE/FA = 3.4/1.7/12, slice thickness 8 mm, 170 × 140 matrix, FOV 400 × 320 – 400, SENSE factor 2) The sequence was performed before and at various intervals 3–15 minutes after injection of Gadolinium-DTPA (total dose 0.1 mmol/kg). For each T1 sequence a region of interest was defined for the myocardium of the systemic ventricle and blood pool. Intensity vs. time curves were used to define T1, and 1/T1 or R_{T1}. The slope of the linear relationship of R_{T1} values for myocardium vs. blood gave the partition coefficient of Gadolinium (lambda). VdGd was calculated as lambda × (1-Hct/100). Results were compared to normal controls (N = 10) and patients with acquired heart failure (N = 4).

Results

VdGd was significantly elevated in ACHD patients compared to controls (32 ± 5% vs. 25 ± 2%, $p < 0.001$). There was a negative correlation between VdGd and systemic ventricular ejection fraction (R = -0.43). VdGd was comparable to patients with acquired heart disease (29 ± 5%, p = ns; p = 0.016 vs. controls), similar to our previously reported values in cardiomyopathy patients. No large macroscopic areas of LGE were present to explain the findings. VdGd was highest in patients with transposition of the great arteries (35 ± 5%) and Eisenmenger syndrome (34 ± 5%).

Conclusion

ACHD patients have evidence of diffuse, microscopic myocardial fibrosis, similar to patients with acquired heart disease such as cardiomyopathy, not shown by LGE. VdGd may facilitate much needed studies on mechanism and therapies for myocardial fibrosis in these patients.