Journal of Cardiovascular Magnetic Resonance



Poster presentation

Open Access

Prolonged pulmonary transit time by cardiac MRI is a marker of hemodynamic derangement in patients with congestive heart failure

Jie J Cao*, Yi Wang, Jeannette McLaughlin, Elizabeth Haag, Michael Passick, Rena Toole, Joshua Cheng, Justine Lachmann and Nathaniel Reichek

Address: St Francis Hospital, Roslyn, NY, USA

from 13th Annual SCMR Scientific Sessions Phoenix, AZ, USA. 21-24 January 2010

Published: 21 January 2010

Journal of Cardiovascular Magnetic Resonance 2010, 12(Suppl 1):P96 doi:10.1186/1532-429X-12-S1-P96

This abstract is available from: http://jcmr-online.com/content/12/S1/P96

© 2010 Cao et al; licensee BioMed Central Ltd.

Introduction

Cardiac MRI (CMR) plays an important role in evaluation of congestive heart failure (CHF). However, despite the effectiveness of routine clinical CMR in assessment of cardiac structure and function, CMR does not directly evaluate hemodynamic variables critical to CHF management.

Purpose

We sought to evaluate the role of pulmonary transit time (PTT) by first pass perfusion CMR in the assessment of hemodynamic derangement in CHF patients

Methods

Subjects were prospectively enrolled and brain natriuretic peptide (BNP) and N-terminal proBNP (NT proBNP) obtained prior to CMR examination. First-pass perfusion was performed in sagital and coronal planes covering the main pulmonary artery and left atrium using a saturation recovery SSFP sequence with 0.005 to 0.01 mmol/kg gadopentetate. PTT was measured as the time interval between peak signal intensity in the main pulmonary artery and peak signal intensity in the left atrium and normalized to heart rate. SSFP cine imaging was performed for ventricular structure and function evaluation. All participants also underwent echocardiography within 2 hours of CMR. Right ventricular (RV) systolic pressure was estimated using Doppler tricuspid regurgitant velocity. Tissue Doppler was used to determine the mitral E/e' ratio.

Results

Of 31 subjects enrolled, 12 were normal controls and 19 CHF patients. Most with CHF (n = 17) were stable outpatients with class I to III NYHA functional class. Compared to controls, CHF patients had lower LVEF (37% vs 56%, p < 0.001), and stroke volume (74 ml vs 96 ml, p = 0.005). There was no significant difference in cardiac output (5.8 $L/\min vs 5.0 L/\min, p = 0.077$). However, PTT was significantly prolonged in CHF patients: 9.0 ± 2.9 s vs 5.7 ± 0.9 s in controls (p < 0.001). Prolonged PTT was closely associated with BNP and NT proBNP(r = 0.746 (p < 0.001), r = 0.789 (p < 0.001), respectively). PTT was also associated with low LVEF (r = -0.653, p < 0.001), low RVEF (r = -0.653) 0.605, p < 0.001), reduced cardiac output (r = -0.533, p = 0.002), RV systolic pressure (r = 0.459, p = 0.032) and mitral E/e' (r = 0.615, p = 0.002). In multivariate models including PTT, cardiac output and LVEF, prolonged PTT was the only predictor of increased BNP (p = 0.003) and NT proBNP (p = 0.001), suggesting an independent relationship between PTT and BNP.

Conclusion

PTT was significantly prolonged in CHF patients. Prolonged PTT correlated with important biomarkers and hemodynamic indices of CHF such as increased BNP, NT proBNP, mitral E/e' and RV systolic pressure. These findings suggest that PTT by CMR is a valuable hemodynamic marker, likely reflecting total pulmonary resistance increases due to abnormalities of both the pulmonary vasculature and left atrial pressure.

^{*} Corresponding author