Journal of Cardiovascular Magnetic Resonance

Meeting abstract

1075 Assessment and relevance of ventricular wall stress in dilated cardiomyopathy

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from 11th Annual SCMR Scientific Sessions
Los Angeles, CA, USA. 1–3 February 2008

Published: 22 October 2008

Journal of Cardiovascular Magnetic Resonance 2008, **10**(Suppl 1):A200 doi:10.1186/1532-429X-10-S1-A200

This abstract is available from: http://jcmr-online.com/content/10/S1/A200

Background

Ventricular loading conditions are crucial determinants of cardiac function. Dilatation of the heart during progression of heart failure is associated with a poor prognosis. In parallel, wall stress increases which has various adverse consequences for energy metabolism and gene expression. Since, wall stress cannot be measured directly in vivo, appropriate models have to be used for its assessment. B-type natriuretic peptide (BNP) is mainly stored in the ventricular myocardium and is released in response to an increased ventricular filling pressure. We examined therefore the relation of BNP to left ventricular (LV) wall stress.

Methods

A thick-walled sphere model based on volumetric analysis of the LV using cardiac magnetic resonance imaging (CMR) was compared with an echocardiography based approach to calculate LV wall stress in 39 patients with dilated cardiomyopathy (DCM) and 21 controls. Serum BNP was used as in vivo marker of raised wall stress. Nomograms of isostress lines were established to assess the extent of load reduction that is necessary to restore normal wall stress.

Results

The CMR based volumetric analysis for wall stress calculation was superior to the echocardiographic based approach which underestimated LV wall stress systematically. Although LV hypertrophy was correlated with the enddiastolic and endsystolic volume ($r = 0.73$, $P < 0.001$; $r = 0.70$, $P < 0.001$), patients with DCM exhibited increased LV wall stress. Both enddiastolic and endsystolic LV wall stress were correlated with the enddiastolic LV volume ($r = 0.54$, $P < 0.001$; $r = 0.81$, $P < 0.001$). LV enddiastolic wall stress was correlated with pulmonary pressure (capillary: $r = 0.69$, $P < 0.001$; artery: $r = 0.67$, P 8 kPa: 587 ± 648 pg/ml, P 12 kPa: 715 ± 661 pg/ml, $P < 0.001$; normal = 4 kPa: 124 ± 203 pg/ml). Analysis of variance revealed LV enddiastolic wall stress as the only independent hemodynamic parameter influencing BNP ($P < 0.01$). When compared with the CMR based wall stress analysis, the echocardiography based method underestimated LV wall stress systematically.

Conclusion

The CMR based method is appropriate to analyze LV wall stress in vivo. LV wall stress was shown to be increased in patients with DCM. A correlation of BNP concentration with LV wall stress was observed in vivo. Using the present mechanistic CMR based approach, nomograms with 'isostress' curves were established which permit to assess the extent of load reduction required to restore normal LV wall stress. Since an increased wall stress is causative for cardiac dilatation, early diagnosis and adequate treatment is expected prevent worsening of heart failure.