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Potential utility of global left ventricular end-systolic wall stress measured by CMR

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Introduction

Global left ventricular (LV) end-systolic wall stress (LVESWS) is related to remodeling and can be quickly and reliably assessed by cardiac magnetic resonance (CMR). LVESWS has previously been shown to inversely correlate with left ventricular ejection fraction (LVEF). However, the relationship of these markers in patients with different types of LV dysfunction has not been well demonstrated.

Purpose

To describe patterns of LVESWS in different subgroups of LV dysfunction and their relationship to other parameters of LV structure and function in a large group of patients referred for clinical CMR.

Methods

Patients referred to the CMR centre from July 2005-September 2009 were retrospectively screened for whether regional wall motion abnormality (RWMA) and late gadolinium enhancement (LGE) were assessed. LVESWS was compared between patients with and without RWMA, with this analysis repeated for subgroups categorized as having normal (greater or equal to 55%) or abnormal LVEF (<55%). Comparison was also made between those with normal/abnormal LGE. The relationship with cardiac output (CO), LVEF and left-ventricular end-diastolic volume index (LVEDVI) was also assessed.

Results

In the population as a whole (n = 3048), LVESWS significantly correlated with LVEF (r = -0.76, $p < 0.001$), and weakly with CO (r = -0.12, $p < 0.001$) and LVEDVI (r = 0.15, $p < 0.001$). LVESWS was significantly higher in those with RWMA (n = 1172) compared to those with no RWMA (n = 1876) (56.9 ± 21.2 vs. 47.5 ± 16.1, $p < 0.001$). A similar finding was observed between those with/without RWMA and reduced LVEF (63.5 ± 21.6 vs. 60.7 ± 19.0, $p = 0.004$), but not in those with normal LVEF (41.7 ± 8.9 vs. 41.2 ± 9.3, $p = 0.75$). When patients were divided based on the presence or absence of LGE, LVESWS was shown to be significantly greater in LGE+ve patients (52.4 ± 20.7 vs. 49.1 ± 15.1, $p < 0.001$). LVESWS correlated highly with LVEF (r = -0.66) and LVEDVI (r = 0.62) in patients with abnormal EF ($p < 0.001$). In patients with normal EF, LVESWS correlated only with LVEF (r = -0.50, $p < 0.001$). Differences in LVESWS between those with/without LGE were still significant even after accounting for difference in LVEF ($p < 0.001$).

Conclusion

Assessment of LVESWS by CMR is feasible in large clinical referral populations and correlates well with conventional measures of LV structure and function. Global LVESWS is related to wall motion abnormalities and to the presence of fibrosis as defined by LGE, with the relationship to LGE remaining after accounting for the effect of LVEF. CMR assessment of global LVESWS should be investigated further for potential incremental value as a prognostic marker in patients with cardiovascular disease.