Low dose dobutamine adds incremental value to delayed enhancement cardiac MR in the prediction of adverse remodelling following acute myocardial infarction.

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Background

The development of left ventricular dilatation, measured as changes in left ventricular end-diastolic volume (LVEDV), in the months following acute myocardial infarction (AMI) is known as 'adverse remodelling'. This adverse remodelling is a poor prognostic factor and well established as a predictor of increased morbidity and mortality.

The use of delayed enhancement cardiac magnetic resonance (DE CMR) in the early post-infarct period has previously been examined for its utility in predicting adverse remodelling at six months.[1,2,3.] Infarct size, as determined by DE CMR predicts changes in LVEDV and wall thinning six months post AMI.[1,2.] The presence of hypoenhancement on first pass perfusion imaging has been shown to be independently predictive of cardiovascular complications and adverse left ventricular remodelling even after controlling for infarct size.[1] The combination of infarct size, presence of persistent microvascular obstruction (PMO) and transmurality of infarction as measured by CMR predict adverse remodelling as defined by an increase of LVDEV of 20% or greater.[3]

Low dose dobutamine (LDD) CMR allows quantitation of contractile reserve in the early post-infarct period. Its utility for the prediction of LV remodelling post AMI is yet to be established. We hypothesised that a comprehensive CMR examination including quantitation of baseline LV dimensions, first pass perfusion kinetics, delayed enhancement, persistent microvascular obstruction and response to dobutamine would yield a model more highly predictive of adverse remodelling than utilising data from the DE component of the scan alone. We therefore examined both qualitative and quantitative variables derived from combined LDD and DE CMR, in order to delineate the most predictive model for the determination of cardiac remodelling post-AMI. In addition we examined the relative value of using data from either the DE component or the LDD component alone.

Methods

55 patients presenting with AMI and a new severely hypokinetic or akinetic segment on screening echocardiogram were recruited. CMR examinations were performed on a GE 1.5T Signa CVi scanner (Waukeshau, USA) 2-6 days after presentation (baseline) and again at 6 months (follow-up). LVEDV at baseline and follow-up was measured using the validated 'sum of discs' method. Qualitative and quantitative variables were recorded at baseline across five categories: 1- baseline dimensions, 2- contractile response to dobutamine, 3 – extent of delayed enhancement, 4- extent of persistent microvascular obstruction , 5 – first pass perfusion kinetics. Quantitation was performed using MEDIS 6.0.1 software (Leiden, Netherlands). Statistical analysis was performed using SPSS version 15.0.

Results

Examining all variables from the combined LDD and DE components of the scan with forwards linear regression revealed a combination of 6 parameters (*volume of PMO, average end-systolic wall thicknes (ESWT)s at rest in the infarct zone, improvement in left ventricular quantitative wall motion in response to dobutamine, improvement in ESWT in the infarct related territory with dobutamine, improvement in thickening with dobutamine in infarct related territory and average transmurality of DE in the infarct zone*) most accurately predicted changes in LVEDV between baseline and follow-up ($r=0.881$, $r^2=0.777$).

The most predictive univariable was volume of PMO ($r=0.62$, $r^2=0.384$).

Examination of the best combination of variables utilising one from each of the 5 categories showed a model encompassing *percentage of myocardium occupied by PMO, left ventricular improvement in quantitative wall motion in response to dobutamine, average ESWT at rest in the infarct zone, average transmurality of DE in the infarct zone, and the number of segments exhibiting hypoenhancement on first pass perfusion*, is also highly predictive of changes in LVEDV ($r=0.842$, $r^2=0.709$).

Linear regression, using variables derived only from examination of the DE component of the scan, derived a model encompassing *volume of DE and average transmurality of DE enhancement in the infarct zone* which also predicted changes in LVEDV ($r=0.66$, $r^2=0.435$) but with less accuracy than the aforementioned combined models.

Linear regression using variables derived only from the LDD component of the scan derived a model encompassing *percentage change in LVEDV in response to dobutamine, improvement in ejection fraction in response to dobutamine, improvement in ESWT in reponse to dobutamine, average quantitative wall motion in the infarct related territory at 10mcg/kg/min of dobutamine infusion, and visual wall motion score at 10mcg/kg/min of dobutamine infusion,* predicted changes in LVEDV with a similar accuracy to the optimal combined model ($r=0.875$, $r^2=0.766$)

Conclusions

A comprehensive CMR examination accurately predicts adverse remodelling post AMI. LDD significantly increases the predictive power of DE CMR and is independently predictive of adverse remodelling. LDD CMR may be effectively utilised alone or in combination with DE CMR for accurate prediction of remodelling in this patient population.

References

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