PEAK TROPONIN I RELEASE CORRELATES WITH INFARCTION SIZE MEASURED WITH DELAYED ENHANCEMENT MRI

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Synopsis

This study compared the peak chemical levels of the cardiac isoform contractile protein troponin I with MR estimates of infarct volume in a chronic canine infarct model. Results indicate a strong correlation between peak troponin levels and infarct size with MRI.

Introduction

The extent of injury, following a myocardial infarction is routinely predicted by measuring the elevation of cardiac troponin I (TnI). The relation between serum levels of troponin I and actual infarct extent has been evaluated by measuring defect size through scintigraphic techniques [1], and by assessing ventricular function measured with echocardiography [2,3]. Limited spatial resolution, and hibernation and stunning of still viable myocardium may have compromised previous studies. MR imaging of myocardial delayed enhancement (DE) after administration of an extra-cellular contrast agent overcomes these limitation. This goal of this study was a comparison of a standard serological marker for infarct size (TnI) against infarct size measured by MRI of DE for relatively small infarcts (<10%) where other imaging modalities with limited spatial resolution may be sub-optimal.

Methods

Seven dogs (n = 7) underwent a thoracotomy procedure for ligation of the proximal left anterior descending coronary artery (LAD). Peak cardiac Tn1 levels were measured in blood samples taken approximately 14 hours after ligation. Imaging was performed two weeks following surgery using a 1.5 T Siemens Sonata system. Delayed enhancement (DE) images were acquired 15-20 minutes following the injection of extracellular contrast agent (0.3 mmol/kg Gd-DTPA) using a T1-weighted 3D FLASH sequence with inversion recovery preparation, and with the following parameters: TR = 450-700 ms; TE = 1.79-1.83 ms; TI = 220-300 ms; α = 52-65^{o}; receiver bandwidth = 975 Hz/pixel; acquisition matrix = 256x 168; phase encodings = 130; pixel size = 1.17 x 1.17 x 2.0 mm. Image analysis was performed with software (CARMA[3]) for global function analysis and mapping of delayed enhancement and regional wall motion defects to a 3D model of the LV. Signal enhancement was calculated for pixels within the endo- and epicardial borders relative to a remote region of interest (Fig 1). Extent was calculated as the ratio of the infarct volume and the total LV volume.

Results

A significant correlation was found between peak TnI and MR infarct extent (r = 0.91; P=0.0046). Infarct sizes ranged from 0.30mL -7.53mL (0.51% -7.83% of the LV) measured by DE MRI. By comparison, to extent of enhancement, the correlation between ejection fraction (EF) and peak TnI was weak (r = 0.53; P=0.35) along with the relationship between infarct extent and EF (r = 0.37; P=0.54).

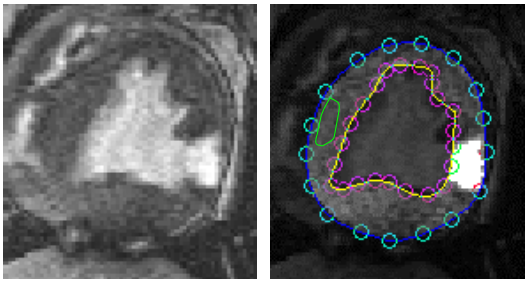

Figure 1. Example DE image from a canine (left) with a MI in the lateral wall and segmented with infracted pixels highlighted (right).

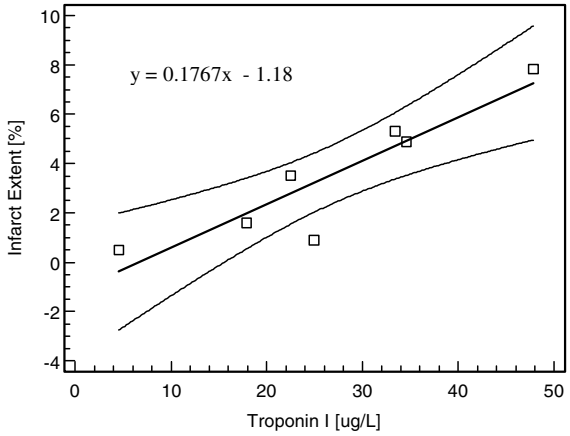

Figure 2. Infarct extent vs. peak TnI values for a chronic canine infarct model

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Discussion

The findings of this study indicate that the serological estimation of infarct size, in particular for small infarcts, may be better than previously reported for imaging modalities with poorer spatial resolution than MRI. Scintigraphic studies have based the determination of infarct size on myocardial segment counts, with approx. 30 segments in a heart. The present study suggests that the serological marker TnI may correlate more tightly with infarct size than previously reported. This suggests that an evaluation of other serological markers should be based on delayed enhancement MRI.

References

1.Wagner, MJ, *et. al.*, Eur J Clin Chem Cline Biochem, 1995; 33(11): 869-72.
2.Wu, AHB, et. al. Clin Chem, 1996; 42:651-2.
3.Apple, FS, *et. al.*, Clinica Chimica Acta, 1998; 272:59-67.