The Effects of Trimetazidine on the Enhancement Patterns of Multi-Detector Computed Tomography in a Porcine Myocardial Infarction Model: What is the Meaning of the MDCT Enhancement Pattern in this Myocardial Infarction Model?

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The recently introduced multidetector-row CT (MDCT), with sub-second rotation times and dedicated cardiac reconstruction algorithms, offers a new opportunity in cardiac CT imaging; in particular, as it can perform high-resolution MDCT coronary angiography. The improved spatial and temporal resolutions of MDCT have made it possible to assess the myocardium.1-3

Contrast-enhanced, two-phase MDCT reveals an early myocardial perfusion defect and late enhancement of acute myocardial infarction (MI), which is consistent with the findings observed in nuclear imaging and on contrast-enhanced MRI.4 The infarct size determined by delayed enhanced MDCT and delayed enhanced MRI shows good correlation with that found in the pathology of an animal model.5

An early defect revealed on MDCT reflected a decrease in the volume of the vascular bed, i.e. a decrease in the myocardial blood flow. Infarct assessment based on the detection of an early defect by MDCT has important limitations. Although it is reasonable that an early defect reflects the region of infarcted tissue in a model of coronary occlusion without reperfusion, this relationship does not apply to reperfused infarcts, which have become much more common following the introduction of reperfusion therapy.6

The mechanism of late enhancement in acute MI is similar to that proposed for delayed gadolinium-enhanced MRI. Under conditions of normal myocyte function, sarcolemmal membranes serve to exclude iodine from the intracellular space. After myocyte necrosis, however, membrane dysfunction ensues, and iodine molecules are able to penetrate the cell. As 75% of the total myocardial volume is intracellular, large increases in the volume of distribution are achieved, thereby, resulting in a marked hyperenhancement.7,8

In a study by Koyama et al.,9 the delayed hyperenhancement in reperfused acute MI was divided into two groups: delayed hyperenhancement (DE) without a residual defect (RD), and delayed hyperenhancement (DE) with a residual defect (RD). They explained that the area of RD might correspond to myocardial necrosis, with an extensive capillary disorder, whereas, DE might correspond to the layer where the blood supply was preserved to some degree. In a long-term follow-up study of the two groups, the percentage decrease in the wall thickness in the DE with RD group was significantly greater than that in the DE without RD group. They concluded that two-phase, contrast-enhanced MDCT proved useful in predicting left ventricle function recovery and the wall thickness in a reperfused acute MI.7

Currently, myocardial late enhancement on MDCT correlates well with delayed enhancement MR imaging during the different stages of MI, and allows for reliable assessment of a reperfused MI during the acute, subacute and chronic stages of MI.8 In chronic MI, hyperenhancement is considered to be the simple consequence of an expanded distribution volume of the contrast material within the scar tissue.

The myocardial enhancement pattern on MDCT is still not widely accepted and still presents as a challenging and controversial field, because MDCT has important limitations, such as the use of iodinated contrast, its lower temporal resolution compared to MRI, and increased exposure to radiation. Therefore, most studies regarding the myocardial enhancement pattern on MDCT are experimental. Lee et al.9 adopted the myocardial enhancement pattern detected by MDCT to evaluate the effect of trimetazidine in a porcine myocardial infarction model. This was a very innovative idea, which
provided a new era for the applications of MDCT. More experimental studies using the myocardial enhancement pattern of MDCT should be expected in the near future. In reviewing other experimental reports, there are certain points that need to be considered. Firstly, the optimal time for imaging myocardial delayed enhancement. Most authors consider image acquisition obtained approximately 5 min or more after contrast injection to be best suited for MDCT visualization of the myocardial delayed enhancement. Secondly, how objectivity can be obtained for the imaging data. Differences in animal weights and body compositions, cardiac outputs and the presence of artifacts induced by cardiac motion, or beam hardening through the contrast bolus in the left ventricle, may induce different imaging conditions. Estimation of interobserver and intraobserver variabilities in imaging interpretation, or calculation of the contrast ratio between infarcted and viable myocardium, might help to overcome these limitations.

It is obvious that MDCT has great potential as a single comprehensive method for examination of the heart when combined with MDCT coronary angiography and a functional analysis. The myocardial enhancement pattern of MDCT is an indication of what the future holds in cardiac imaging.

REFERENCES