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EDITORIAL

Reperfusion injury components and manifestations determined by cardiovascular MR and MDCT imaging

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Abstract

Advances in magnetic resonance (MR) and computed tomography (CT) imaging have improved visualization of acute and scar infarct. Over the past decade, there have been and continues to be many significant technical advancements in cardiac MR and multi-detector computed tomography (MDCT) technologies. The strength of MR imaging relies on a variety of pulse sequences and the ability to noninvasively provide information on myocardial structure, function and perfusion in a single imaging session. The recent technical developments may also allow CT technologies to rise to the forefront for evaluating clinical ischemic heart disease. Components of reperfusion injury including myocardial edema, hemorrhage, calcium deposition and microvascular obstruction (MO) have been demonstrated using MR and CT technologies. MR imaging can be used serially and noninvasively in assessing acute and chronic consequences of reperfusion injury because there is no radiation exposure or administration of radioactive materials. MDCT is better suited for assessing coronary artery stenosis and as an alternative technique for assessing viability in patients where MR imaging is contraindicated. Changes in left ventricular (LV) volumes and function measured on cine MR are directly related to infarct size measured on delayed contrast enhanced images. Recent MR studies found that transmural infarct, MO and peri-infarct zone are excellent predictors of poor post-infarct recovery and mortality. Recent MR studies provided ample evidence that growth factor genes and stem cells delivered locally have beneficial effects on myocardial viability, perfusion and function. The significance of deposited calcium in acute infarct detected on MDCT requires further studies. Cardiac MR and MDCT imaging have the potential for assessing reperfusion injury components and manifestations.

Key words: Calcium deposits in myocardium; Magnetic resonance imaging; Multi-detector computed tomography; Myocardial micro and macro-infarct; Reperfusion injury; Vascular injury

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INTRODUCTION

Ischemic heart disease remains the leading cause of

death worldwide and accounts for the majority (almost 70%) of congestive heart failure cases. Reperfusion therapy, which includes thrombolytic therapy, angioplasty and stent placement, is the greatest advancement in the treatment of acute myocardial infarction. On the other hand, reperfusion therapy induces myocardial injury. Recent studies have shown that interstitial edema, infarct dimensions (size, circumferential extent and transmurality), peri-infarct zone, microvascular obstruction (MO), interstitial hemorrhage and calcium deposition are major components of reperfusion injury and these components predict the short and long term survival.

The common denominator magnetic resonance (MR) sequences used in clinical viability protocols involves evaluating left ventricular (LV) function on cine techniques and myocardial infarct size on delayed contrast enhancement (DE) imaging. Other pulse sequences, such as first pass perfusion, tagged, velocity encoded cine, T2-weighted turbo spin echo and T2*-susceptibility MR imaging have also been used for assessment of the consequences of post-infarct reperfusion. MR imaging has also been useful in defining the etiology of non-ischemic diseases, such as amyloidosis[1], viral myocarditis[2] and hypertrophic cardiomyopathy[3]. DE-MR imaging also eliminates the exposure of patients to ionizing radiation used in computed tomography (CT) imaging[4].

On the other hand, the clinical indications for implantable cardiac defibrillators and biventricular pacing therapy continue to expand, and the development and validation of alternative imaging modalities with similar abilities for assessing LV function, perfusion and viability, are needed to accommodate such a growing population of patients who are unfavorable candidates for MR imaging. The application of multi-detector computed tomography (MDCT) does not suffer from relative contraindications (such as implanted active permanent pacemakers or defibrillators (or retained components of either, due to their potential to become dysfunctional) and/or unwanted conductors (e.g. induction of ectopy or heating capable of burning) within the rapidly changing magnetic and radiofrequency environments during imaging commonly confronting MR imaging in routine clinical cardiac imaging. Furthermore, there is no limitation of basic life-support and physiologic-monitoring equipment in the vicinity of the CT scanner. MR and MDCT imaging have been used to assess cardiac anatomy[5], measure left and right ventricular volumes and function[6], regional perfusion[7,8] and myocardial viability[9,10]. The focus of this review is on the manifestations of reperfusion injury using MR and MDCT imaging.

COMPONENTS OF REPERFUSION INJURY

Myocardial edema

Reperfusion of previously ischemic myocardium causes edema related to the leakage of blood macromolecules into interstitium. Edema is one of the features of the salvageable area at risk[11,12]. The increase in mobile water content (edema) within the ischemic region causes a prolongation of T2 relaxation time[13]. The salvaged area at risk in reperfused infarct has been visualized on T2-weighted turbo spin echo MR imaging as a hyperintense area (Figure 1)[14-18].

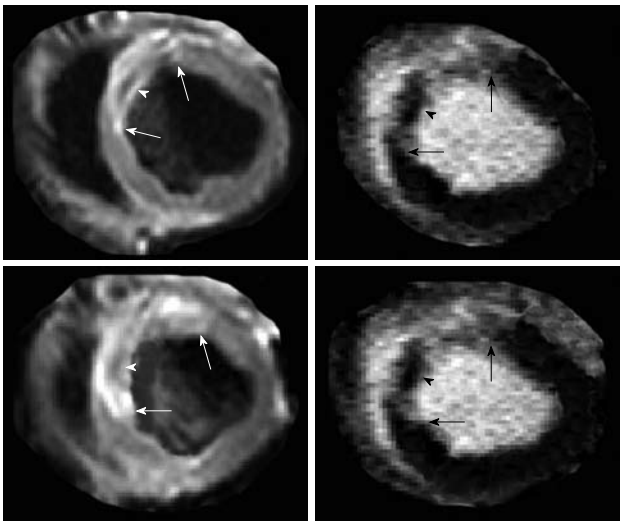

Figure 1 Multi-slice T2-weighted turbo spin echo imaging demonstrates the hyperintense edematous area at risk (white arrows in the left images) with hypoenhanced zone, which may represent microvascular and/or interstitial hemorrhage zone (arrowhead in the left images). Delayed contrast enhancement magnetic resonance (DE-MR) imaging confirmed the presence of microvascular and/or interstitial hemorrhage zone in the core (black arrowhead in the right images) of hyperenhanced infarcted myocardium (black arrows in the right images).

Accurate assessment of the area at risk is required to compare different revascularization techniques or for studies aimed at improving myocardial salvage[19-22] as an end point. MR imaging has documented regional myocardial edema in patients with normal coronary angiograms[23] and stunning[24]. Kwong *et al*[25] combined the assessment of LV function, myocardial perfusion and infarct in patients who presented to the emergency room with chest pain. The investigators found that MR imaging is the strongest predictor for the diagnosis of acute coronary syndrome compared with a standard workup. The sensitivity and specificity of MR imaging for detecting acute coronary syndrome was 84% and 85%, respectively. Moreover, multiple logistic regression analysis revealed that MR imaging had independent diagnostic value over clinical parameters, including ECG and initial troponin I levels. In another adenosine perfusion MR study, Ingkanisorn *et al*[26] evaluated the diagnostic value of adenosine in 135 patients who presented to the emergency room with chest pain with no elevation in troponin levels. MR imaging data indicated that there was no evidence of significant ischemic heart disease in these patients. Patients were contacted 1 year later to determine the incidence of coronary artery stenosis (> 50%) on invasive coronary angiography, abnormal correlative stress test, new infarct, or death. Based on this survey, MR perfusion imaging showed 100% sensitivity and 93% specificity for the detection of myocardial ischemia. It was concluded that MR imaging had significant prog-

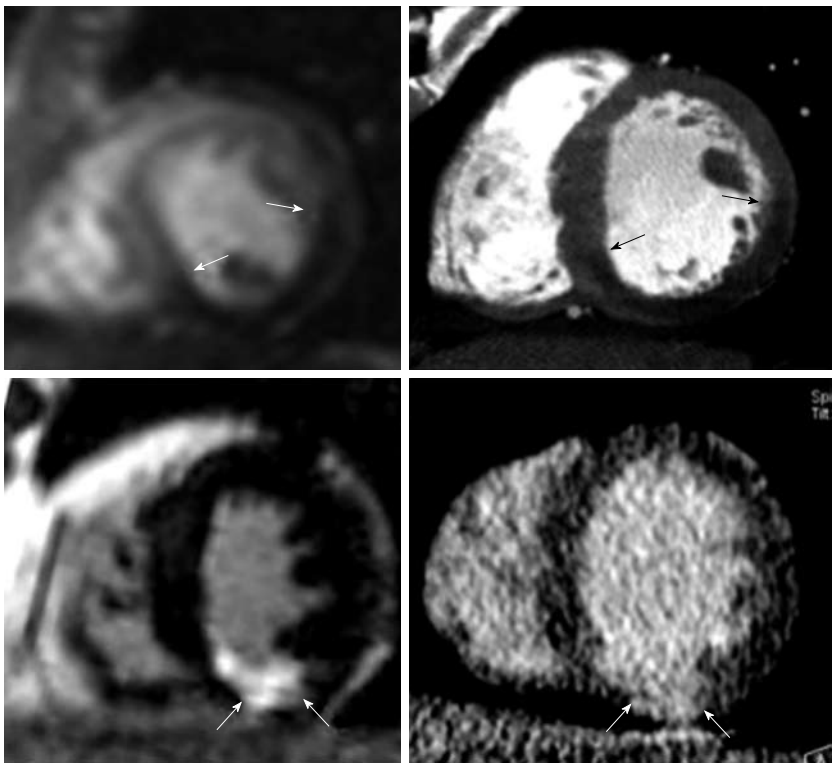

Figure 2 Head-to-head comparison between first pass perfusion MR (top left) and first pass multidetector computed tomography (MDCT) imaging (top right) in a 42-year-old man with acute reperfused infarct. Ischemic myocardium (arrows) appears as a hypoenhanced region with comparable extent on both imaging modalities. Bottom: Head-to-head comparison of DE-MR (bottom left) and DE-MDCT imaging (bottom right) shows a bright region comparable in size to enhanced inferior infarct (arrows). Note that the enhanced infarct on DE-imaging is substantially smaller than ischemic myocardium[38].

nostic value in predicting a future diagnosis of ischemia, infarct, or death. First pass perfusion imaging can be used to discriminate ischemic myocardium. A recent MR-impact study in 234 patients reported improved detection of ischemic myocardium distal to coronary stenosis compared to single photon emission computed tomography in a multicenter and multivendor randomized trial[27].

MDCT imaging has also been used in the evaluation of cardiac function, myocardial viability and plaque morphology[28-30]. A preclinical study demonstrated that this modality has the potential to detect infarct heterogeneity in the peri-infarct zone[31]. Recent experimental studies using modern MDCT technology confirmed the potential of the technique in depicting ischemic myocardium during the first pass perfusion of iodinated contrast media[10,32].

HOMOGENEOUS MYOCARDIAL INFARCT

Several studies indicated that there is a close correlation between homogeneous myocardial infarct size, dimensions (size, circumferential extent and transmurality) and LV remodeling. Inversion-recovery low-angle-shot MR imaging and helical MDCT imaging have been recently introduced and performed following the intravenous administration of contrast media with a delay of 5-10 min to define myocardial infarct dimensions[7,9,10,33-39] (Figure 2). Investigators found that differentially contrast enhanced regions on MR and MDCT imaging correlate well with areas of decreased flow[32,40] and dobutamine stress on echocardiography[41]. Furthermore, the combined use of cine and DE-MR imaging are able to differentiate regional transitional dysfunction in stunned and hibernating myocardium from permanent dysfunction on contrast enhanced infarct[42,43]. A recent study showed multicontrast MR imaging enables simultaneous assessment of wall motion, MO and viability[44].

Cine and DE-MR imaging have been used to determine contractile reserve in transmural and non-transmural infarct[45,46]. These studies have also indicated the substantial improvement in regional function in segments with 50% transmural enhancement and global LV improvement in transmural enhancement of less than 25% of LV wall thickness[45-47]. Others found that 75% of the patients with transmural enhancement died within 26-36 mo of diagnosis[48]. Tarantini *et al*[49] demonstrated in 76 patients with reperfused infarct that transmural enhancement using DE-MR imaging is associated with LV remodeling. These findings were confirmed by Roes *et al*[50] who showed that the size of the infarct scar in 231 patients is a stronger predictor of all-cause mortality than LV ejection fraction and LV volumes. Thus, extensive transmural enhancement is an excellent predictor of poor recovery.

Contrast enhanced T1-weighted and non-contrast T2-weighted MR imaging is useful in discriminating acute from chronic myocardial infarct[51]. In a study of 73 patients with acute and chronic infarct by Abdel-Aty *et al*[51] MR imaging was effective (96% sensitive) in discriminating acute from chronic infarct. In a preclinical study, Saeed *et al*[52] observed lack of deferential enhancement of chronic infarct after administration of blood pool MR contrast media, but not after clinically approved extracellular MR contrast media. Unlike acute reperfused infarct, chronic infarct lacks edema, MO or hemorrhage because they are resorbed.

Expanding the use of coronary MDCT into clinical

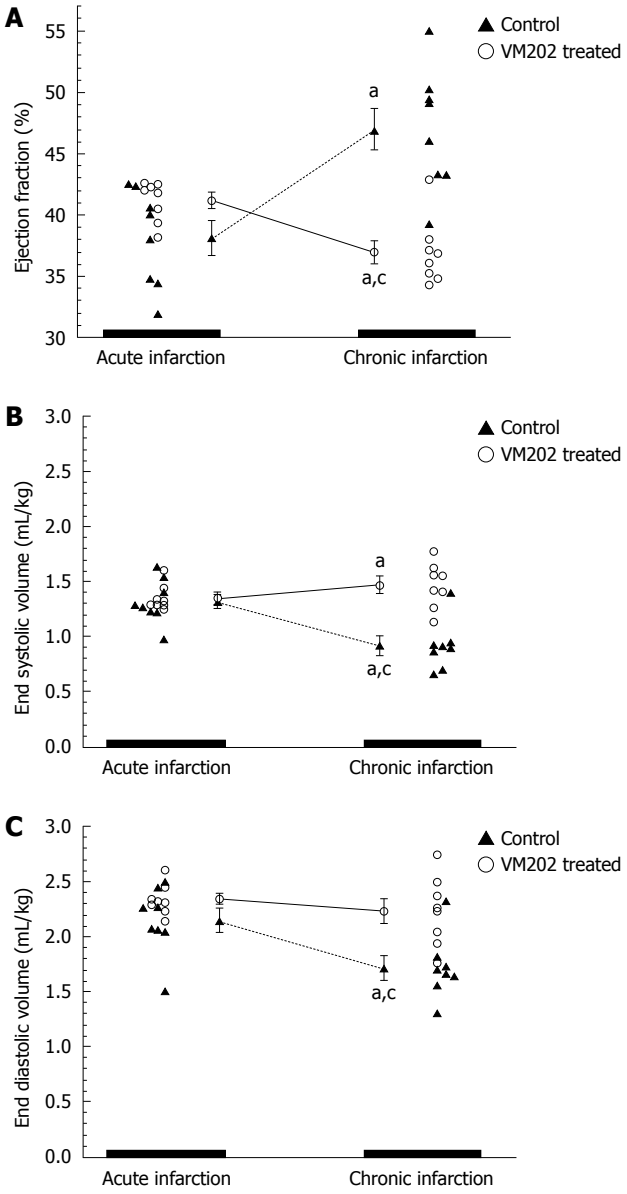

Figure 3 The ejection fraction (A), end systolic volumes (B) and end diastolic volumes (C) are shown for control and hepatocyte growth factor gene (VM202) treated animals. The hepatocyte growth factor gene administered at 3 d after reperfusion significantly decreased end diastolic (mL/kg) and end systolic volumes at 8 wk compared to 3 d infarct ($^{a}P < 0.05$) and control group ($^{c}P < 0.05$). Control animals at 8 wk showed a significant decrease in ejection fraction and significant increase in end systolic and end diastolic volumes compared with 3 d infarct[59].

practice has sparked interest in using the modality for assessing myocardial viability[53]. Gerber *et al*[9] showed the similarity between infarct size measured on DE-MDCT and DE-MR imaging in a series of patients. The investigators demonstrated good agreement (82%, $k = 0.61$, $P < 0.001$) between the two measurements (Figure 2). Nikolaou *et al*[54] demonstrated the diagnostic power of MDCT in assessing the presence, age, and size of myocardial infarct in 106 patients. Myocardial infarct was found in 27 of 106 patients. MDCT detected 23 of 27 patients with infarct with a sensitivity of 85%, specificity of 91% and accuracy of 90%.

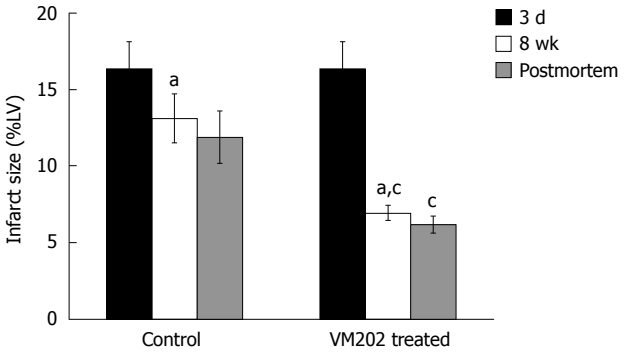

Figure 4 Histogram shows the difference in infarct size prior to intramyocardial gene delivery at 3 d and 8 wk after infarction in control animals (left block) and animals treated with hepatocyte growth factor gene (VM202) (right block) measured on DE-MR imaging (black and white bars) and postmortem (gray bars). Note the decline in infarct size was greater in gene treated animals compared with control animals. $^{a}P < 0.05$ compared with 3 d acute infarction. $^{c}P < 0.05$ compared with 8 wk chronic infarction in control animals. %LV: Percentage of LV mass[59].

Transfer of angiogenic genes to ischemic myocardium is a promising approach under development for the treatment of myocardial infarct. MR and CT imaging may be a useful tool for defining myocardial infarct and for use in targeting the infarct for gene and stem cell therapies[55-60]. Catheter-based fluoroscopic MR and MDCT imaging has been recently used for delivering these therapies transendocardially[37,58]. Sequential cine and DE-MR imaging showed great sensitivity in detecting improvement in ejection fraction, reduction of LV volumes and infarct size (Figures 3 and 4) after intramyocardial delivery of different angiogenic genes[58-60]. Figure 5 demonstrates the increase in vascular density of infarcted myocardium 8 wk after intramyocardial delivery of vascular growth factor gene. Thus, MR imaging provides great promise in evaluating gene and cell therapies[58,61-63].

A preliminary experimental study investigated MDCT for the assessment of the efficacy of stem cells in infarcted myocardium and showed that this technique has the capability to elucidate new therapies[37]. The radiation doses in MDCT may limit such application in patients because therapeutic studies need a minimum of two imaging sessions. The potential advantages of using MDCT in assessing myocardial viability may be related to faster acquisition time compared with cardiac MR imaging and the ability to scan claustrophobic or uncooperative patients. Additionally, MDCT angiography is the method of choice for direct visualization of the coronary arteries, coronary calcium and atherosclerosis in its earliest stages; when treatment can be most effective in preventing subsequent heart attacks or sudden death. On the other hand, MR imaging has other advantages over MDCT including: (1) the absence of radiation exposure; (2) the lack of nephrotoxic iodinated contrast media; and (3) it allows for repeated scans, particularly in pediatric patients. It should be noted that MR contrast media cause nephrogenic systemic fibrosis in patients with compromised renal function[64].

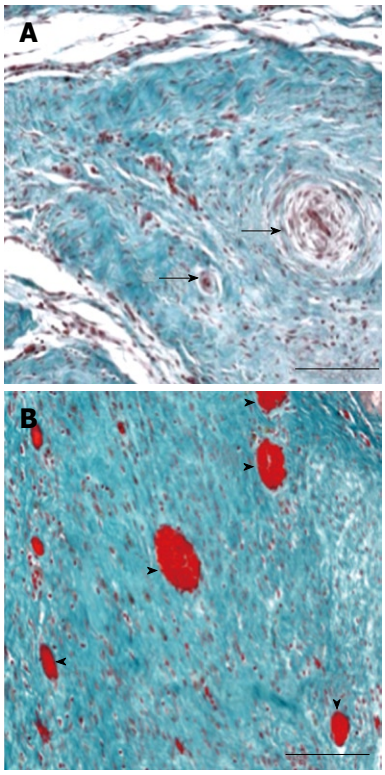

Figure 5 Micrographs of representative infarcts in control and VEGF-gene treated swine. A: The infarct in the control animal shows no appreciable angiogenesis and residual blood vessels have been remodeled, as shown by the thick vascular wall and small lumen (black arrows). B: VEGF-gene treated animal contains numerous blood vessels (arrowheads) in linear array representing injection track (calibration bar = 200 μm)[60].

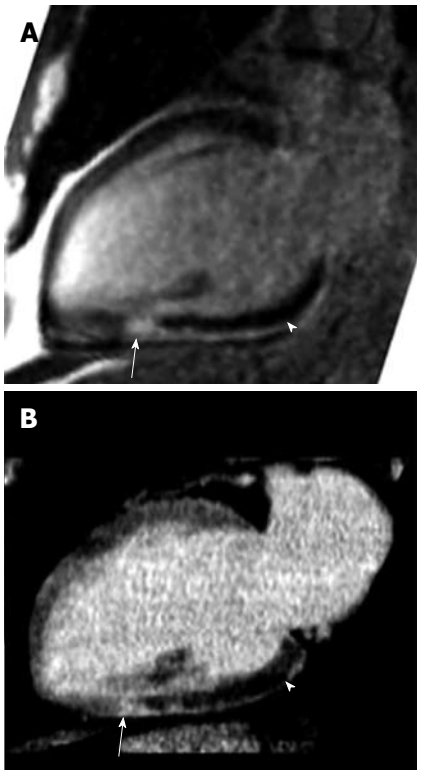

Figure 6 Head-to-head comparison of DE-MR (A) and DE-MDCT images (B) showing the dark MO zone (arrowheads) surrounded by a bright enhanced infarct in a reperfused patient (arrows)[38].

MANIFESTATIONS OF REPERFUSION INJURY

MO zone

In the setting of an acute myocardial infarction, treatment strategies have primarily focused on the management of culprit occlusions in the epicardial coronary arteries[65]. Interventional cardiologists, however, found that the benefits of revascularization of the epicardial coronary artery is limited and later discovered that MO is a major component of infarction, which is frequently seen after revascularization of the epicardial coronary artery. Investigators found that the formation of MO is related to plaque emboli, endothelial swelling, inflammation, extravascular edema and microvascular spasm[66].

How best to measure MO in terms of predictive values is an important question. A variety of techniques, flow or frame count[67,68], myocardial blush grade[69] coronary Doppler imaging[70], contrast echocardiography[68], contrast-enhanced MR imaging[71,72] and contrast enhanced MDCT[32,38], have been used to detect MO zone in patients with TIMI (thrombosis in myocardial infarction). The quality of some of these techniques, however, is suboptimal due to poor spatial resolution.

MR and MDCT imaging delineated MO as a hypoenhanced zone in the core of acutely reperfused infarct (Figure 6). The delineation is attributed to inadequate contrast media delivery during first pass perfusion (17 s blood recirculation time)[46], early (1-2 min; equilibrium phase of contrast medium in the blood and tissue interstitium)[72] and delayed (10 min; peak enhancement of myocardial infarct) MR imaging[73]. The extent of MO after bolus administration of contrast media is time dependent and varies between first pass, early contrast enhancement and DE-imaging because it is governed by 2 processes namely: perfusion and passive diffusion. Figure 6 illustrates the comparable MO extent measured on DE-MR and DE-MDCT imaging in a patient subjected to reperfusion.

Both early and delayed persistent MO has been shown to predict post-infarct LV remodeling and outcome in patients with ST-elevation myocardial infarction (STEMI)[72-76]. A recent study showed that MO detected on DE-MR imaging is more frequently observed in patients with the most severe LV dyfunction[77]. A clinical study in 25 patients demonstrated that delayed persistent MO is also high (32%) in the No-STEMI population after successful percutaneous coronary intervention[78], but less than that observed in STEMI patients[73,79]. Recent studies indicated that MO is predictive of increasing recurrent myocardial infarct, congestive heart failure, stroke and death up to 16 mo after the event[72,73,75].

Preclinical studies showed that the extent of MO in reperfused infarct is less variable in the first 10 min after administration of blood pool MR contrast media, which may be attributed to slow convection of the contrast medium in the interstitium and its retention in the blood pool[80].

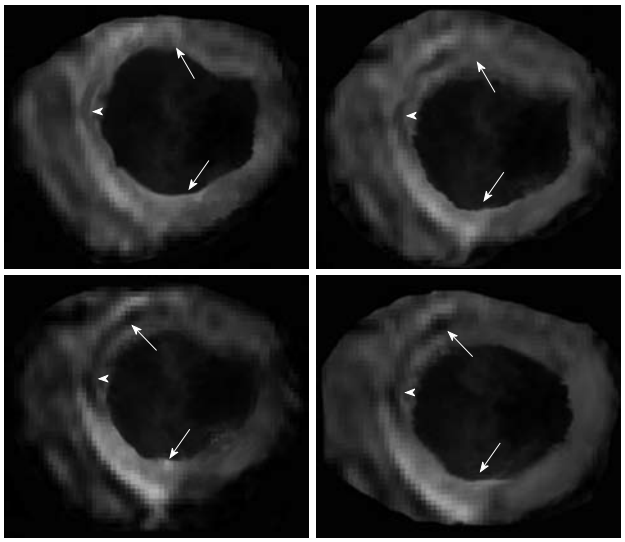

Figure 7 Multislice T2*-weighted (susceptibility) turbo spin echo images show severe interstitial hemorrhage in the core (arrowheads) of the hyperintense edematous area at risk (arrows) 3 d after reperfusion.

INTERSTITIAL HEMORRHAGE

Interstitial hemorrhage is another component of reperfusion injury in patients with ST-segment elevation due to acute infarct. Its presence is an important marker for myocardial and microvascular damage. Interstitial hemorrhage causes signal loss on T2*-weighted images, which depends on the status of hemoglobin (oxyhemoglobin, deoxyhemoglobin, or methemoglobin) and the presence of blood products such as ferritin and hemosiderin[15,81]. Figure 7 demonstrates intensive hypointense interstitial hemorrhage 3 d after reperfusion in a swine model on T2*-weighted (susceptibility) turbo spin echo MR imaging. O'Regan *et al*[82] quantified the extent of interstitial hemorrhage on T2*-weighted mapping and compared it with other indices of ischemic injury, such as area at risk and infarct size.

Although non-enhancing myocardium within the infarct is thought to represent MO[46], it is possible that the presence of blood products may also contribute to its low signal seen on MR images[15]. Ganame *et al*[83] also used T2-weighted MR imaging to measure the extent of hemorrhage and area at risk in 98 patients with a large reperfused infarct. Based on this technique, the investigators demonstrated a high prevalence of myocardial hemorrhage of 25% in this patient cohort, more common amongst patients with large transmural infarct and severe LV global and regional dysfunction.

PERI-INFARCT ZONE

A mixed population of viable and non-viable myocytes has been found around acutely infarcted myocardium, a territory previously described as the peri-infarct zone[84,85]. Microscopic studies indicated that the peri-infarct zone has leaky microvessels[84-88]. The peri-infarct zone has consistently been substantiated by a variety of modalities, including echocardiography[89], radiopaque bead arrays[90] and MR imaging[82]. The physiological correlates of the peri-infarct zone using MR imaging have been described[91].

MR or CT contrast media have been used to define the peri-infarct zone[31,88], identify patients who are susceptible to ventricular arrhythmias[92] and predict post-infarct mortality[93]. Using preclinical necrosis-specific (mesoporphyrin) and extracellular MR contrast medium in a seminal animal study, Saeed *et al*[94] demonstrated MR characterization of the peri-infarct zone. They found that the enhanced region on DE-MR imaging is larger than the true infarct delineated on TTC staining, which was identical to regions enhanced by the necrosis-specific contrast medium. The difference in enhancement regions demarcated by the 2 contrast media was considered the peri-infarct zone. At that time our findings were in contrast to other groups who demonstrated that differentially enhanced myocardium represents necrotic tissue. Recent clinical MR studies confirmed our findings[31,92,95] and went further to report the associations between infarct size, the peri-infarct zone and inducible ventricular arrhythmias[31,92,93,95]. Yan *et al*[93] found that the extent of the peri-infarct provides prognostic value for mortality incremental to that offered by ejection fraction and LV end-diastolic volume. On the other hand, the existence of viable myocytes in a large peri-infarct zone may raise an interesting hypothesis that reperfusion could be beneficial by reducing arrhythmogenic triggers, despite the apparent lack of measurable improvement in contractile function. Furthermore, implantable cardioverter-defibrillator therapy may be warranted in such high-risk patients identified by MR due to the creation of multiple action potential circuits derived from the peri-infarct zone.

CALCIUM DEPOSIT

Considering the deleterious effects of calcium overload in reperfused myocardium[96,97], the development of a noninvasive technique to visualize calcium deposits in infarcted myocardium may have clinical value. Noninvasive imaging techniques that directly incorporate the spatial distribution of calcium in infarcted myocardium may help our understanding of the relationship between calcium deposits in the myocardium, the rate of infarct resorption, and LV function[98].

Calcium deposited in infarcted myocardium has previously been used as a target for 99m-Tc pyrophosphate scintigraphy to delineate reperfused myocardial infarcts in patients[99,100]. For over a decade, electron-beam CT has been clinically used for calcium scoring in the coronary arteries of patients. In addition to calcium scoring and detection of coronary stenosis, modern MDCT scanners have been used to assess the extent of acute and chronic infarct[10,101].

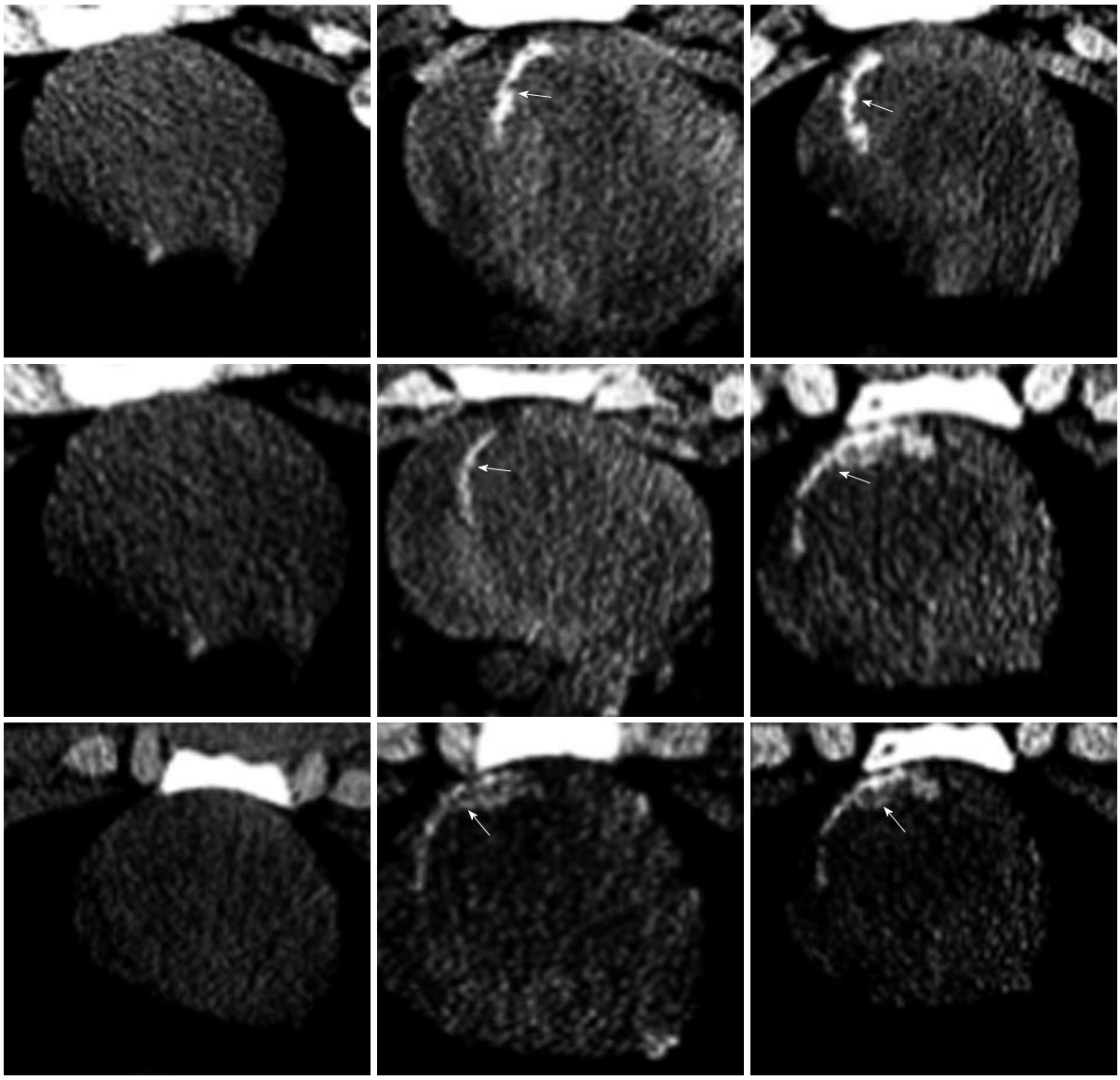

Figure 8 Non-contrast enhanced MDCT imaging demonstrates deposited calcium 7 d after reperfusion (right block arrows). In all 3 animals (the 3 rows) the deposited calcium was not evident at 2-3 h after reperfusion on non-contrast enhanced MDCT (left block)[102].

In a recent animal study, non-contrast MDCT images depicted calcium deposits as "hot-spots" 1 wk after reperfusion (Figure 8). The presence of calcium deposits on non-contrast MDCT images, however, was transient and specific to acute infarct because the calcium was resorbed from the infarct scar at 8 wk as shown on CT imaging and histopathology (Figure 9). Histopathology confirmed the engulfment of the deposited calcium by macrophages[102]. Noninvasive evaluation of the beneficial administration of calcium channel blockers on calcium overload during reperfusion may be possible using MDCT.

HETEROGENOUS MICROINFARCT

Heterogeneous microinfarct results from showers of microemboli shed following coronary intervention. Clinical studies showed that 42% of patients experience major cardiac problems, such as heart failure and sudden death, after percutaneous coronary angioplasty[103,104]. High incidences (30%-50%) of defects on myocardial perfusion scintigraphy have also been detected soon after coronary balloon angioplasty and with optimally implanted stents[105,106] and these events continued during follow-up[107-109]. The emboli sizes, collected by distal protection devices during percutaneous coronary intervention, differ widely (47-2503 μm)[110] and the size and number of ruptured atherosclerotic plaques is a key event in the pathogenesis of heterogeneous microinfarct[111]. A recent clinical study demonstrated that the volume of embolized material relates directly to the volume of new necrosis detected by delayed-enhancement MR imaging[112]. The American College of Cardiology and the European Society of Cardiology recently recognized the detrimental consequences of coronary microembolization in patients in their 2007 guidelines[113].

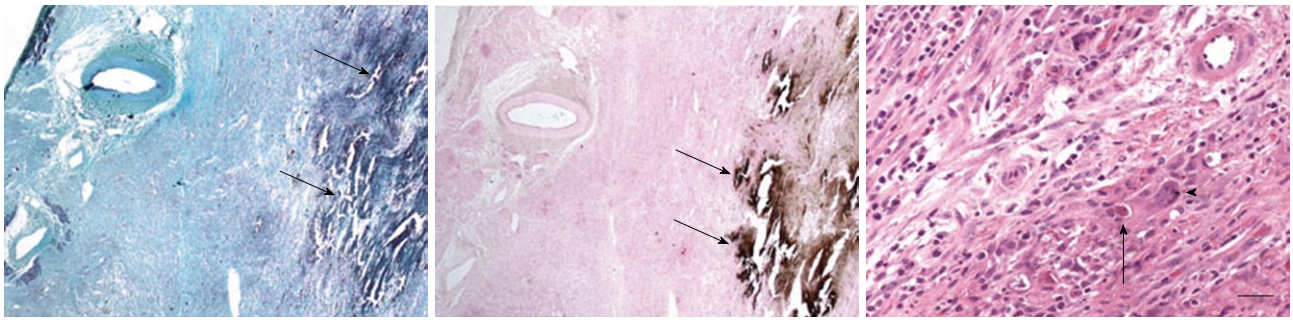

Figure 9 Histopathology from at 1-wk-old reperfused infarct shows calcium deposits as a black-brown precipitation product (arrows) on Masson trichrome (A) and the special calcium stain (von Kossa stain) (B). At 8 wk the stain shows traces of calcium deposits (C, arrow) surrounded by giant cell (arrowhead). Apparently the giant macrophages digest deposited calcium (calibration bars = 20 mm)[102].

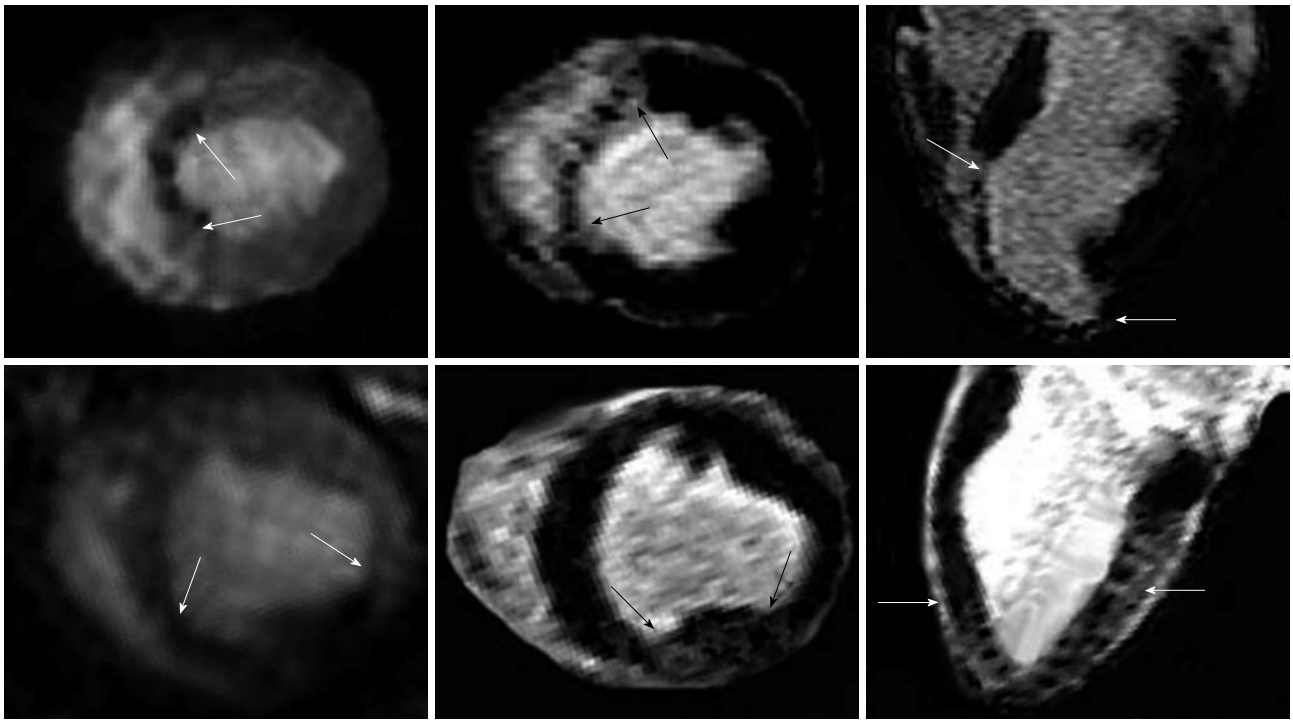

Figure 10 Preclinical study shows the patchy enhanced embolized region in two animals after delivery of embolic materials (7200 microsphere count, 100-300 μm diameters). Animal one received the embolic materials in the left anterior descending coronary artery causing perfusion deficit in the antero-septal wall 2-3 h after delivery of the embolic materials (top left, arrows), while animal two received the embolic materials in the left circumflex coronary artery causing perfusion deficit in the inferior wall (bottom left, arrows). Short (center images) and long (right images) axis views of DE-MR imaging illustrates the microinfarct of the same ischemic territory 7 d after embolization[114].

In a preclinical study, a microinfarcted region was detected on first pass perfusion imaging 2-3 h as well as 7 d after embolization[114]. DE-MR imaging failed to define microinfarct early but at 7 d it was clearly visible. Microinfarct was visualized as bright heterogeneous subregions on DE-MR imaging (Figure 8). Furthermore, DE-MR and DE-MDCT imaging is sensitive in detecting the direction of embolized vessels in experimental animals (Figure 9).

Several studies demonstrated the potential of DE-MR imaging in visualizing heterogeneous microinfarct in patients[102,112,114-118]. Ricciardi *et al*[118] and Choi *et al*[119] have demonstrated heterogeneous microinfarct in patients on DE-MR imaging. The investigators found a link between MR visualization of microinfarct and impaired myocardial perfusion (Figure 10)[119]. Selvanayagam *et al*[120] demonstrated that the extent of elevated troponin I levels 24 h after coronary intervention is directly related to the extent of microinfarct on DE-MR imaging. More recently, they examined myocardial perfusion and microinfarct serially after percutaneous coronary intervention using MR technique[117]. They found that myocardial perfusion is reduced in myocardial segments with new microinfarct 24 h after percutaneous coronary intervention. It has been shown that microinfarct causes severe and persistent LV dysfunction and in some cases sudden death[40]. Investigators concluded that even small amounts of infarct (microinfarct) detected on

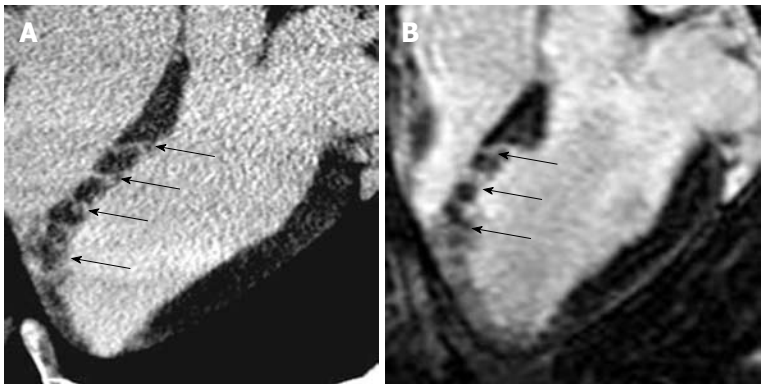

Figure 11 DE-multi-slice MDCT (A) and DE-MR (B) images from experimentally embolized LAD coronary artery show good correspondence between the modalities in defining heterogenous microinfarct. Both modalities show enhanced stripes (arrows) of microinfarct extending from the epicardium to the endocardium mapping occluded microvessels.

DE-images provides prognostic value beyond the routine clinical, angiographic and functional predictors[25].

Heterogeneous microinfarct is not limited to percutaneous coronary intervention for atherosclerosis but include a wide range of diseases, such as valvular disease, prosthetic valve, endocarditis, cardiomyopathy with mural thrombus, arrhythmias and during heart-lung-bypass[121-125]. This pathology has also been reported in patients with hypertension, diabetes[126], systemic lupus erythematosus[127] and sickle cell disease, where abnormally shaped erythrocytes obstructing the capillaries and small arterioles may cause myocardial fibrosis[128]. Therefore, early detection and subsequent effects of microinfarct need highly sensitive imaging modalities.

CONCLUSION

The clinical role of MR and MDCT imaging continues to expand supported by the advances in software and hardware. The strength of MR imaging relies on the variety of pulse sequences and the ability to noninvasively provide information on myocardial structure, function and perfusion in a single imaging session. The complementary use of both MR and MDCT imaging allows the components and manifestations of reperfusion injury including myocardial edema, interstitial hemorrhage, calcium deposition and MO to be visualized. MR imaging can be used serially and noninvasively in assessing the consequences of reperfusion injury because there is no radiation exposure or administration of radioactive materials. MDCT is better suited for assessing coronary artery stenosis and as an alternative technique for assessing viability in patients where MR imaging is contraindicated. Clinical MR studies found that the presence of transmural infarct, MO and peri-infarct are excellent predictor of poor post-infarct recovery and mortality[31,92,93,95,129]. Heterogeneous cardiac microinfarct detected on contrast enhanced MR and MDCT imaging has a prolonged effect on LV function and perfusion (Figure 11)[130]. The clinical significance of deposited calcium, detected on MDCT, in acute homogeneous infarct requires further studies. Recent preclinical MR studies provided ample evidence that angiogenic genes and stem cells, delivered transendocardially under MR-guidance, have beneficial effects on myocardial function, perfusion and viability. Imaging protocols are in progress to monitor the long-term efficacy of such therapeutic agents. Cardiac MR and MDCT imaging can characterize reperfusion injury components and manifestations.

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