Laboratory Investigation

Detecting Acute Myocardial Infarction by Diffusion-Weighted

versus T2-Weighted Imaging and Myocardial Necrosis Markers

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We used a porcine model of acute myocardial infarction to study the signal evolution of ischemic myocardium on diffusion-weighted magnetic resonance images (DWI). Eight Chinese miniature pigs underwent percutaneous left anterior descending or left circumflex coronary artery occlusion for 90 minutes followed by reperfusion, which induced acute myocardial infarction. We used DWI preprocedurally and hourly for 4 hours postprocedurally. We acquired turbo inversion recovery magnitude T2-weighted images (TIRM T2WI) and late gadolinium enhancement images from the DWI slices. We measured the serum myocardial necrosis markers myoglobin, creatine kinase-MB isoenzyme, and cardiac troponin I at the same time points as the magnetic resonance scanning. We used histochemical staining to confirm injury. All images were analyzed qualitatively. Contrast-to-noise ratio (the contrast between infarcted and healthy myocardium) and relative signal index were used in quantitative image analysis.

We found that DWI identified myocardial signal abnormity early (<4 hr) after acute myocardial infarction and identified the infarct-related high signal more often than did TIRM T2WI: 7 of 8 pigs (87.5%) versus 3 of 8 (37.5%) (P=0.046). Quantitative image analysis yielded a significant difference in contrast-to-noise ratio and relative signal index between infarcted and normal myocardium on DWI. However, within 4 hours after infarction, the serologic myocardial injury markers were not significantly positive. We conclude that DWI can be used to detect myocardial signal abnormalities early after acute myocardial infarction—identifying the infarction earlier than TIRM T2WI and widely used clinical serologic biomarkers. ***(Tex Heart Inst J 2016;43(5):383-91)***

Key words: *Diffusion magnetic resonance imaging; disease models/animal; feasibility studies; image enhancement/methods; magnetic resonance imaging/methods; myocardial infarction/diagnosis/physiopathology; myocardium/pathology; predictive value of tests; signal-to-noise ratio; swine*

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Cardiac magnetic resonance (CMR) has been confirmed as useful in acute myocardial infarction (AMI) for evaluating infarct size, myocardial edema, and intramyocardial hemorrhage.[1] T2-weighted imaging (T2WI) sequences and late gadolinium enhancement (LGE) sequences are also used clinically to evaluate recent myocardial infarction (MI).[2] When combined with LGE, the area at risk on T2WI can be used to compute myocardial salvage[3] and differentiate acute from chronic myocardial ischemia.[4] There are several T2 imaging methods, such as short tau inversion recovery (STIR), turbo inversion recovery magnitude (TIRM) T2WI, and T2 mapping. The last method is relatively new and not used widely in clinics. Most often, STIR and TIRM T2WI sequences are used to identify myocardial edema.[5-9]

Several challenges hinder the widespread clinical acceptance of T2WI sequences. First, the sensitivity of T2WI in detecting MI is in doubt, ranging from 35% to 100%. Several authors have reported a low sensitivity of approximately 60%.[4,6,9-11] In addition, artifacts caused by slow blood flow make it difficult to distinguish myocardial edema from subendocardial blood.[12] Therefore, the usefulness of T2WI to detect AMI needs further confirmation.

In diffusion-weighted imaging (DWI), protons precess at the same rate if they experience the same magnetic field. A pulse gradient is applied before refocusing by a 2nd pulse gradient; the protons that have not moved will be fully rephased, causing no signal loss. Protons that have moved will be dephased and cause a drop in signal intensity (SI).

Various factors can influence the diffusion of water molecules (which is not consistent in the body), such as the extent of tissue cellularity and the presence of intact

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cellular membranes. During acute myocardial ischemia, the rapid failure of high-energy metabolism and associated ionic pumps leads to the migration of sodium and calcium into the cell. The subsequent influx of osmotically obligated water results in cellular swelling (cytotoxic edema) and a decrease in the extracellular volume fraction. The ischemic territory will be seen on DWI as a hyperintense region.

Investigators have used DWI to detect acute stroke.[13] However, in cardiac DWI, both the cyclical movement of the heart and respiratory movement will cause motion artifacts and image signal-to-noise ratio (SNR) loss. In the past 20 years, along with the development and application of new technology, in vivo cardiac DWI has become possible, and DWI has been acknowledged as an alternative to conventional T2WI for detecting recent MI.[9,10] Nevertheless, the study of applying DWI in AMI has focused on patients whose hemodynamic condition is stable, usually in the first few days after the onset of AMI.[9,10,14]

Whether DWI can detect AMI earlier after onset has not been determined. Therefore, our objectives in this study were to observe the signal patterns of infarcted myocardium dynamically, using DWI sequences, and to compare these findings with those of traditional T2WI sequences in an in vivo porcine model of acute-reperfusion MI.

Materials and Methods

This experimental animal study complied with the Helsinki declaration and was approved by the Southeast University Institutional Animal Care and Use Committee. Nine Chinese miniature pigs (4 female and 5 male; weight range, 20.6–27.5 kg; age range, 4–5 mo) were anesthetized with 4 to 5 mg of intramuscular ketamine. A continuous intravenous infusion of 3% thiopental sodium (6 g/200 mL physiologic saline solution) was used to maintain anesthesia throughout all experimental procedures. One 23.6-kg male pig died during intubation. The remaining 8 pigs underwent percutaneous transluminal coronary angioplasty (PTCA) to induce AMI[15]; this procedure reduced the risk of death and ensured sufficient infarcted area. After a coronary angiogram of the left coronary anatomy was obtained, a coronary angioplasty balloon (size range, 1.25–1.5 mm) was advanced into the distal left anterior descending coronary artery (LAD) or left circumflex coronary artery (LCx) (Fig. 1). The balloon was inflated for 90 min. Coronary angiography was used to confirm vessel occlusion. The balloon was then deflated, and the artery was reperfused. Cardiac function was monitored by means of continuous electrocardiography (ECG) and invasive arterial blood pressure monitoring. Direct-current defibrillation was applied when ventricular fibrillation occurred.

Magnetic Resonance Protocol. The CMR scans were performed preprocedurally and at 1, 2, 3, and 4 hours postprocedurally. All the pigs underwent CMR on a 3-T MAGNETOM Verio machine (Siemens Healthcare GmbH; Erlangen, Germany) with a flexible, 6-channel, phased-array surface coil combined with a 6-channel, phased-array, posterior spine coil, which resulted in a 12-element cardiac coil fully compatible with integrated parallel acquisition. Peripheral or ECG gating was achieved with use of an in vivo magnitude physiologic monitor through a plethysmographic tracer placed on the pig's tail or through electrodes placed on the anterior chest wall. True free-induced steady-state precession was used to locate the anatomic axis of the heart. During repeated periods of suspended respiration (10–15 s), ECG-gated cine images encompassing the entire left ventricle

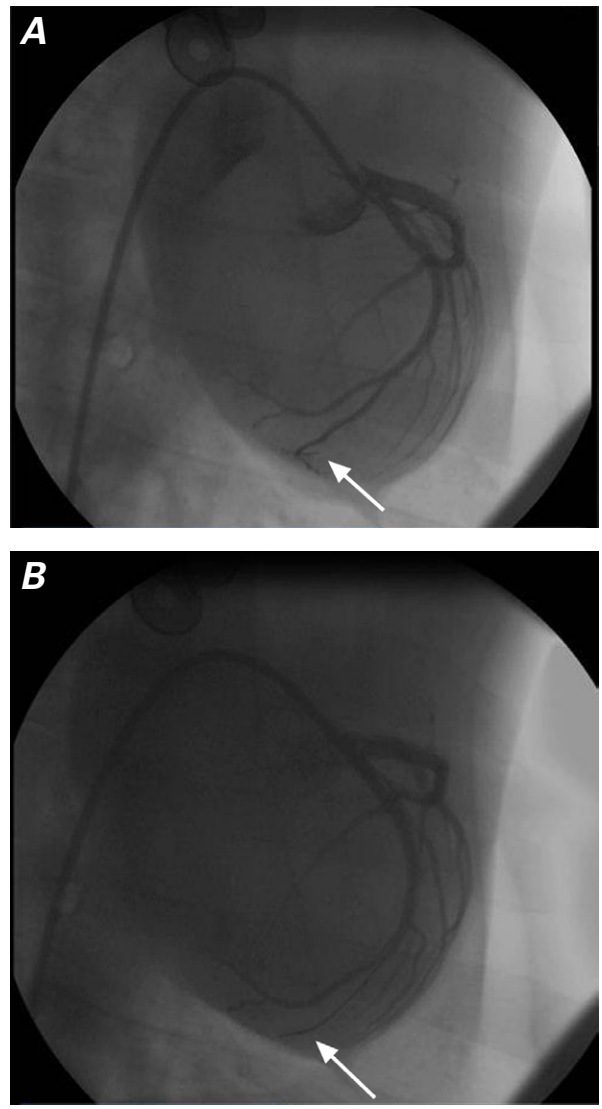

Fig. 1 *Coronary angiograms.* ***A****) Before balloon inflation, perfusion of the remote branch of the left anterior descending coronary artery is normal (arrow).* ***B****) After inflation, flow in that branch disappears (arrow).*

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were acquired, to examine the suitable time for trigger delay.

Table I shows the details of the 3 sequences. A DWI single-shot echo-planar imaging (SS-EPI) sequence with a bipolar+ diffusion scheme lasting 15 ms was acquired in the short-axis view with 3 b values (0, 50, and 100 s/mm^2). The b values indicate the sensitivity of each sequence in measuring the diffusivity of water molecules. The images were acquired during suspended respiration in the pigs. Slice thickness was 6 mm with no gap between the 7 slices covering most of the heart. Each slice was obtained at end-diastole by means of ECG-triggered segmented data acquisition. A generalized, autocalibrating, partially parallel acquisition technique of 3 with 24 autocalibration lines was chosen for acceleration. An SS-EPI sequence with an EPI factor of 156 was selected. A spectral adiabatic inversion recovery pulse that automatically calculated inversion time was used for fat saturation.

The TIRM T2WIs were acquired at the same locations and time points as the DWIs. Postprocedural images were acquired 15 min after a 0.2-mmol/kg bolus intravenous injection of Magnevist® gadolinium-diethylenetriamine pentaacetic acid (Bayer Pharma AG; Berlin, Germany). The LGE images were acquired at the same locations, via phase-sensitive inversion recovery. A dedicated scouting sequence was used to adjust the optimal inversion recovery time.

Serum Myocardial Necrosis Markers. Blood samples (7 mL) were collected for the measurement of serum myocardial necrosis markers before intervention and at the 4 time points of the DWI scanning.

Myocardial necrosis myoglobin, the creatine kinase-MB isoenzyme (CK-MB), and cardiac troponin I (cTnI) (with minimum detection concentrations of <3 ng/mL, 1.6 U/L, and 16 pg/mL, respectively) were detected in serum with use of enzyme-linked immunosorbent assay kits: myoglobin and cTnI, from MyBioSource, Inc. (San Diego, Calif); and CK-MB, from Santa Cruz Biotechnology, Inc. (Dallas, Texas). Each serum sample was tested twice, to calculate the average values. The kits simultaneously detected the restructuring of natural porcine myocardial protein without cross-reaction with other porcine cytokines; coefficients of variation between board and board were all <10%.

TABLE I. Settings for Image Acquisition

Variable	DWI	TIRM T2WI	LGE
Repetition time	300 ms + RR	2–RR	2–RR
Echo time (ms)	65	47	1.1
Field of view (mm)	370 × 300	340 × 265	340 × 255
Resolution (mm)	192 × 158	256 × 133	192 × 102
Voxel size (mm^3)	1.9 × 1.9 × 6	2 × 1.3 × 6	2.5 × 1.8 × 6
Bandwidth (Hz/pixel)	1,860	310	1,532
Echo space (ms)	0.64	6.71	2.6
b value (s/mm^2)	0/50/100	—	—
Inversion time (s)	—	—	280–380
Fat saturation	SPAIR	SPAIR	—

DWI = diffusion-weighted imaging; LGE = late gadolinium enhancement; SPAIR = spectral adiabatic inversion recovery; TIRM T2WI = turbo inversion recovery magnitude T2-weighted imaging

Postmortem Histochemical Staining. The pigs were euthanized by intravenous infusion of a potassium chloride overdose. The hearts were harvested immediately and placed into a plastic receptacle. The cavities and receptacle were filled with 3% agarose solution (Sigma-Aldrich Corp.; St. Louis, Mo), to preserve ventricular shape and volume. The excised hearts were sliced into 6-mm sections from the apex to the base with use of a commercial meat-slicer. The specimens were immersed in 1% 2,3,5-triphenyltetrazolium-chloride solution (TTC) (Sigma-Aldrich) at 37 °C for 15 min. The TTC-stained areas were defined as noninfarcted myocardium, and the TTC-unstained areas, as infarcted myocardium. The specimens were then stored in 10% formalin solution for 12 hr. Photographs of each specimen were obtained with a digital camera.

Image Analysis. All CMR images were independently analyzed on a dedicated workstation (Siemens Medical Solutions, Inc.) by 2 of the authors, who had 10 and 5 years of experience in CMR imaging. Qualitative and quantitative image analyses were performed. To avoid interpretation bias, TIRM T2WIs and DWIs were presented to each analyst in random order. First, the images were evaluated in regard to whether high-signal myocardial areas could be detected.

For qualitative image analysis,[10] overall image quality and the quality of blood suppression were scored in terms of the delineation of the left ventricular wall, the high-signal area within the myocardium, and the SI of healthy myocardium. The uniformity of blood-signal inhibition in the left cavity was also examined. Scoring was on a 4-point scale: 1, poor; 2, moderate; 3, good; and 4, excellent.

For quantitative image analysis, the left ventricular endocardial and epicardial borders were manually traced with a dedicated pointer. Depending upon the standard deviation of SI, the regions of interest were defined as infarcted segments, remote normal segments, and the background for each pig heart. Remote normal myocardium was defined as segments with TTC stain but no LGE. Infarcted myocardium was present when the myocardial SI exceeded the mean value + 2 × SD of remote normal myocardium.[9] The background was defined as a 1-cm^2 region of interest positioned in the background air 2 to 3 cm anterior to the pigs' chest

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wall. The DWIs with b values of 50 s/mm^2 showed the highest SI for infarcted and normal myocardium, so these were selected to compare with the T2WIs (Fig. 2). The contrast-to-noise ratio (CNR) between regions of infarcted and healthy myocardium and the relative signal index (RSI) were calculated as follows[10]:

$$CNR = (SI_{high\ signal} - SI_{healthy\ myocardium})/\text{mean of } SI_{noise},$$

and

$$RSI = (SI_{high\ signal} - SI_{healthy\ myocardium})/SI_{healthy\ myocardium},$$

where $SI_{high\ signal}$ is the mean SI of the infarcted segments, $SI_{healthy\ myocardium}$ is the mean SI of the remote normal myocardial segments, and SI_{noise} is the mean background SI.

Because of the differences in voxel size and bandwidth between the DWI and TIRM T2WI sequences, the T2WI signal was multiplied by a correction factor of 0.5.[9,10]

Statistical Analysis

SPSS software version 13.0 (IBM Corporation; Endicott, NY) was used for statistical analysis. Data are reported as mean ± SD. Normality was confirmed by means of the Shapiro-Wilk test. The CNR and RSI between DWIs and T2WIs were compared with use of paired-sample *t* tests. One-way analysis of variance for repeated measurements was used to compare the CNR at different time points in DWIs. *P* values <0.05 were considered to be statistically significant. The inter- and intraobserver agreements for evaluating high-signal areas for DWI and TIRM T2WI sequences were calculated with use of the kappa coefficient.

Results

Qualitative Image Analysis. All 8 pigs that underwent PTCA were confirmed to have infarcted areas consistent with the anatomic perfusion territory of the culprit arteries by TTC staining. Three pigs were positive for both sequences, with one supplied by the LAD and 2 by the LCx. Three of the 4 pigs with a high signal on the DWI images but normal TIRM T2WIs had regional distributions consistent with the LAD territory; in the 4th of those pigs, the LCx artery was affected. The 8th pig was negative for both sequences; TTC stain showed that the infarcted area was supplied by the LCx. High-signal areas were detected in 7 of 8 pigs (87.5%) with use of DWI, and in 3 of 8 pigs (37.5%) with use of TIRM T2WI (*P*=0.046).

Upon evaluation, the overall quality of the DWIs versus TIRM T2WIs was not significantly different. The mean quality of blood suppression was better on the

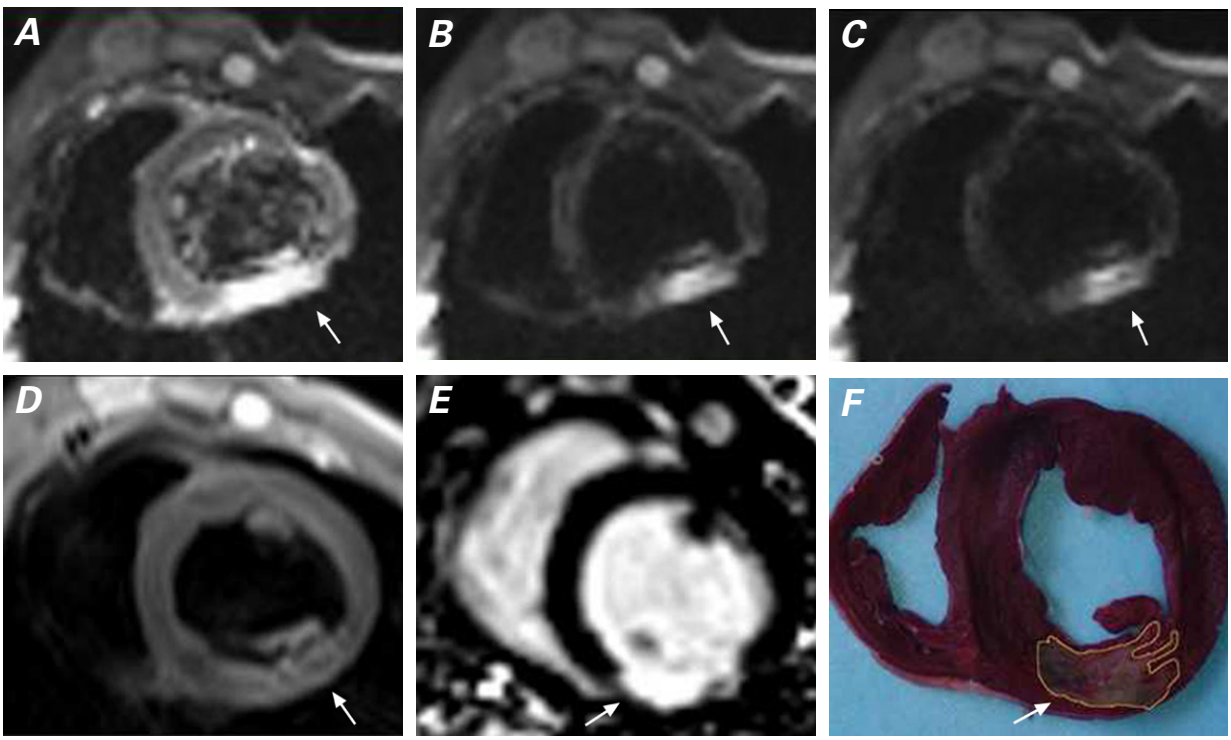

Fig. 2 *Diffusion-weighted, T2-weighted, and late gadolinium enhancement images from one pig, obtained 2 hours after acute myocardial infarction in the territory of the left circumflex coronary artery. Diffusion-weighted images (short-axis views) with* ***A****) b=0,* ***B****) b=50, and* ***C****) b=100 s/mm^2 show hyperintense areas (arrows) in the inferior left ventricular wall.* ***D****) Turbo inversion recovery magnitude T2-weighted image shows a mildly hyperintense area (arrow).* ***E****) Late gadolinium enhancement and* ***F****) 2,3,5-triphenyltetrazolium chloride histochemical staining confirm the infarction.*

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DWI images than on the T2WI images (3.29 ± 0.76 vs 2.14 ± 0.9; P=0.047) (Table II).

Quantitative Image Analysis. At each time point, the CNR and RSI on the DWIs were significantly higher than on the TIRM T2WIs (Tables III and IV). The mean values of CNR on the DWIs at the 2nd time point had the highest value, but the difference was not statistically significant among the 4 time points (Fig. 3). The respective inter- and intraobserver kappa values between the image analysts were 0.78 and 0.80 (DWI) and 0.82 and 0.85 (T2WI).

Serum Myocardial Necrosis Markers. At the beginning of hour 2, all the pigs had slightly elevated myoglobin levels but within the test kit's normal range (10–92 ng/mL) (Fig. 4). The CK-MB concentration was normal (<25 U/L) for 4 hours. Serum cTnI was undetectable during serial measurement in the 8 pigs.

TABLE II. Comparative Qualitative Analysis of the Imaging Methods*

Variable	DWI	TIRM T2WI	*P* Value
Image quality	3.29 ± 0.76	3.43 ± 0.53	—
Blood suppression	3.29 ± 0.76*	2.14 ± 0.9	0.047

DWI = diffusion-weighted imaging; TIRM T2WI = turbo inversion recovery magnitude T2-weighted imaging

*Grade: 1 = poor, 2 = moderate, 3 = good, 4 = excellent.

Data are presented as mean ± SD. P <0.05 was considered statistically significant.

TABLE III. Comparison of CNR between the Imaging Sequences over Time

Time (hr)	DWI	TIRM T2WI	*t*	*P* Value
1	39.7 ± 18.52	4.57 ± 2.32	5.041	0.002
2	45.36 ± 27.45	5.8 ± 1.32	3.176	0.009
3	39.41 ± 16.53	5.78 ± 2.39	4.87	0.003
4	33.36 ± 7.14	6.08 ± 2.55	11.064	—

CNR = contrast-to-noise ratio; DWI = diffusion-weighted imaging; TIRM T2WI = turbo inversion recovery magnitude T2-weighted imaging

Data are presented as mean ± SD. P <0.05 was considered statistically significant.

TABLE IV. Comparison of RSI between the Imaging Sequences over Time

Time (hr)	DWI	TIRM T2WI	*t*	*P* Value
1	1.67 ± 0.79	0.03 ± 0.01	5.377	0.002
2	1.6 ± 0.48	0.04 ± 0.02	8.491	—
3	2.01 ± 1.25	0.03 ± 0.01	4.162	0.006
4	1.88 ± 0.49	0.04 ± 0.01	9.681	—

DWI = diffusion-weighted imaging; RSI = relative signal index; TIRM T2WI = turbo inversion recovery magnitude T2-weighted imaging

Data are presented as mean ± SD. P <0.05 was considered statistically significant.

Discussion

In this study, the DWI sequence detected myocardial high-signal areas more often than did the TIRM T2WI sequence in pigs with periacute MI. The CNR and RSI on the DWIs were significantly higher than on the TIRM T2WIs, indicating that DWI enabled higher infarcted-to-normal myocardium contrast resolution than did TIRM T2WI. In comparison with serum myocardial injury markers, DWI detected MI earlier. The effect of the DWI sequence on inhibiting the blood-pool signal was better than that of the TIRM T2WI sequence; this agrees with previously published work.

Abdel-Aty and colleagues[16] reported that T2WI sequences could detect abnormal signals in dogs with myocardial ischemia for 26 min. The mean CNR on the T2WIs in their study was approximately 12.8 ± 9.6, slightly different from our findings. The reported CNR with a large SD indicated that T2WI detects AMI with substantial uncertainty. Because different sequencing parameters were used, those results cannot be compared with ours; nevertheless, the CNR on the T2WIs was significantly lower than that on the DWIs.

Different animal models might produce different experimental results. Maxwell and associates[17] reported that pigs have no intercoronary connections. In that case, coronary occlusion would lead to a zone of severe ischemia, which, absent reperfusion, would rapidly deteriorate to irreversible injury and cell death. In contrast, in species such as the dog, extensive collateral connections might slow cellular injury and deliver sufficient blood to enable survival in at least part of the original ischemic zone. The abundant collateral blood flow can aggravate the myocardial edema. Therefore, in dogs with early coronary artery ligation, high signals can appear on T2WIs. Because AMI anatomy in human beings and pigs is similar, use of the porcine model is feasible, although true AMI perfusion territories should be used.[18,19]

Kociemba[9] and Deux[10] and their respective colleagues performed imaging in patients who had sustained recent MI (within a mean 3.5 ± 2 and 2.5 ± 1.52 d, respectively), and showed that DWI had a higher sensitivity than T2WI; however, the difference in CNR between infarcted and healthy myocardium was not statistically significant. Imaging during different phases after AMI

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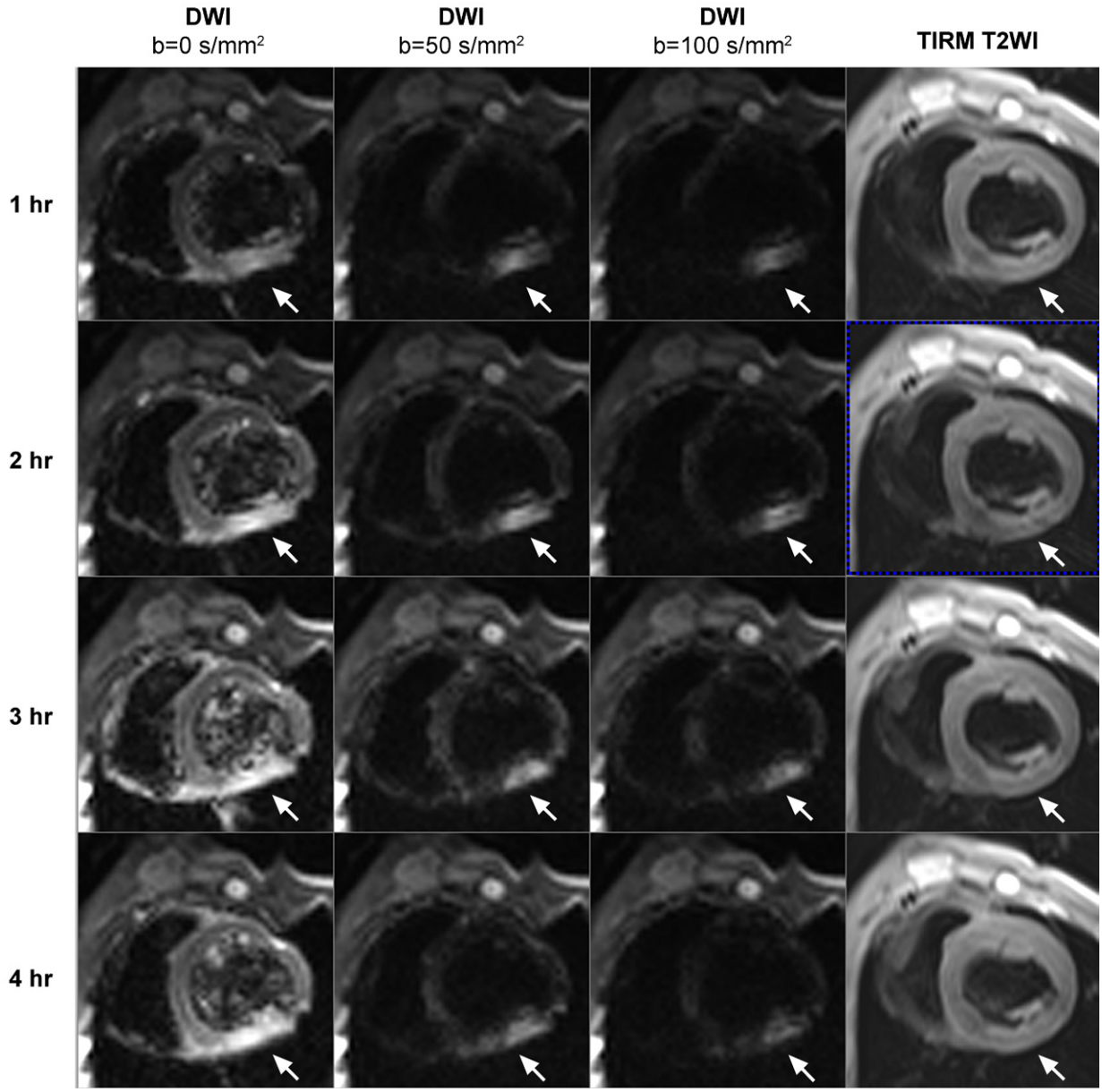

Fig. 3 *Cardiac magnetic resonance images from one pig, obtained after acute myocardial infarction in the territory of the left circumflex coronary artery (arrows), show findings from diffusion-weighted imaging (DWI) and turbo inversion recovery magnitude T2-weighted imaging (TIRM T2WI).*

might account for this difference between their findings and ours. According to strong evidence, myocardial edema is predominantly intracellular early in the course of ischemic injury, and T2WI might be unable to detect bound water molecules in the case of intracellular edema.[20] The long T2 relaxation time of protons bound in free water indicates regional myocardial edema and is the primary cause of changes in T2WI signal intensity.[16] According to Nilsson and co-authors,[21] myocardial edema was greatest one week after the infarction and then gradually declined. Jeanette and associates[20] reported no relevant intramyocardial SI changes in T2WI within 1 hr after therapeutic septal artery embolization. These authors also found that no significant edema was detectable 7 hr after the intervention. Upon reperfusion, the flow of normal osmotic blood into this hyperosmotic region worsened the edema.[21] Therefore, the extent of edema in infarcted myocardium might increase as the extent of reperfusion increases, possibly increasing the T2 values.

The optimal b values for tissue characterization depend on the tissue or organ that is being evaluated. Diffusion-weighted sequences with high b values (>1,000 mm^2/s) are widely used to detect acute stroke.[22] As b values increase, the SNR is reduced significantly and is more sensitive to the homogeneity of the magnetic

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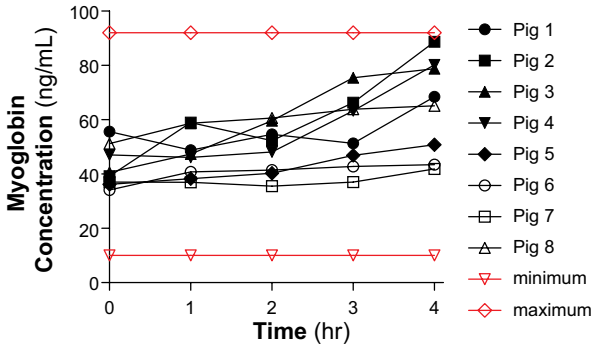

Fig. 4 *Graph shows blood myoglobin concentration in the 8 pigs before acute myocardial infarction (hour 0) and at each hour after infarction. The red lines show the normal range of myoglobin (10–92 ng/mL) according to the testing kit.*

field. In addition, the periodic movements of hearts can hinder high resolution for in vivo DWI. Therefore, the study of hearts with use of DWI sequences has chiefly focused on low-b-value conditions. When low b values are used, the blood flow within the capillary network and the T2 shine-through effect cannot be avoided. Both of these factors are partly responsible for the high signal. The apparent diffusion coefficient (ADC) map and exponential DWI can be obtained by using DWIs with different b values. These methods can be used to remove the contribution from the T2 SI of the tissue under examination, thereby increasing the sensitivity of detecting infarction.[23,24] However, the mechanisms of the signal pattern in AMI are not fully understood. The analysis of ADC maps and exponential DWI is beyond the scope of this paper.

Myocardial injury markers will appear to be abnormal only when the myocardial membrane has been damaged and related proteins are released into the serum.[16] The myoglobin levels in our pigs began to increase from the 2nd postprocedural hour. However, most samples remained lower than the reference limit during the 4 time points of CMR. According to Vikenes and colleagues,[23] in the nonischemic experimental animal (with only a femoral artery incision), myoglobin and CK-MB concentrations increased, but cTnI was unaffected. Moreover, the increase in conventional markers was highly correlated with injured skeletal muscle. Therefore, the rise in myoglobin and CK-MB concentrations cannot be fully representative of myocardial cell damage. In comparison with open surgery, animal models of PTCA have less damage and agree more with normal pathologic processes of disease. In addition to a small number of false positives, cTnI cannot be detected in healthy human beings.[25] Vikenes and colleagues[23] observed that elevation of cTnI above the reference limit (0.4 ng/mL) occurs 4 to 9 hours after myocardial ischemia, and the maximal increase is observed at 12 to 14 hours. Our study similarly revealed no detectable increase in cTnI within 4 hours after AMI. The objective of myocardial injury-marker detection was to illustrate that serologic measurements would not detect ischemic injury when DWI signal changes were readily visible.

Several acute heart diseases are notable for substantially increased water content in the injured myocardium: ischemia,[10] myocarditis,[26,27] heart-transplant rejection,[28] and takotsubo cardiomyopathy.[29] Diffusion-weighted sequences can track the motion of water molecules in the tissue to obtain information about extracellular space and cell membrane integrity. Abnormal myocardial-cell energy metabolism causes cellular swelling to occur within a few minutes after ischemia; the extracellular space becomes smaller, and the free diffusion of water molecules is restricted.[30,31] This mechanism is probably related to the unusually high signal on DWIs. According to Mahrholdt and associates,[32] the major difference between MI and myocarditis is that areas of tissue damage in myocarditis are usually smaller than in MI. These authors also assumed that myocarditis occurs predominantly from the epicardial quartile of the wall and is most often located in the lateral free wall. In comparison, MI typically originates from the subendocardial portion of the wall.[33,34]

Lymphocytic and monocytic infiltration, the hallmark of acute transplantation rejection, is followed by endothelial cellular injury, structural tissue deterioration, and myocardial necrosis. Wu and co-authors[28] reported that in vivo cellular CMR for macrophages and lymphocytes, in combination with functional CMR and strain analysis, could be used to diagnose heart-transplant rejection at the cellular and functional levels. In the acute phase of takotsubo cardiomyopathy, DWIs have shown hyperintense areas when the LGE images appeared to be normal.[29] In addition, angiographic normalcy of the coronary arteries is an essential characteristic of this disease.[27] It is therefore necessary to identify the cause of the high signal on DWI images.

Our study has some limitations. First, we focused on signal traits within 4 hours after the onset of occlusion; the characteristics of damaged myocardium beyond 4 hours have been described.[9,10] Second, the arteries were reperfused immediately after 90 min of balloon occlusion; it is unknown how the DWI signal might have evolved had there been no reperfusion. Third, because of the relatively low SNR on DWIs, it was difficult to measure the infarcted area exactly or to compare it with the infarcted area as confirmed by TTC staining. We used DWI to confirm the presence of AMI; however, quantification of the infarcted area is needed to further improve the SNR of DWIs. Fourth, different T2WI sequences (such as T2 mapping and bright-blood T2 imaging) and imaging parameters are crucial for comparison. We used TIRM because it is the chiefly used T2WI sequence. Unified standards of imaging need further research. We used a small number of pigs, which

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might have limited the power of our analysis. Therefore, we did not analyze the sensitivity of the different infarct locations with regard to the DWI sequence. The findings in this study must be confirmed through clinical application.

Conclusion

In this study, we showed the feasibility of using DWI sequencing to identify infarcted myocardium in a porcine model of AMI. In comparison with TIRM T2WI sequences, DWI sequences more frequently detected high signals caused by AMI. In comparison with serologic myocardial necrosis markers, DWI sequences revealed abnormalities earlier and with greater specificity. Relatively few studies involve diffusion imaging of the heart, especially in the acute phase of AMI, so our findings warrant further investigation.

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