

Evaluation of Microvascular Function of Residual Viable Myocardium in Infarct Zone after Percutaneous Coronary Intervention: Quantitative Myocardial Blood Flow Measurement Using Magnetic Resonance

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Introduction

For patients with acute myocardial infarction (AMI), the extent of residual viable myocardium in the infarct zone and the corresponding microvascular function are important determinants of subsequent functional recovery, left ventricular remodeling, and long-term prognosis (1). To date, the microvascular function of the residual viable myocardium is still inaccessible non-invasively. Therefore, the aim of this study was to quantify the zonal-dependent myocardial perfusion and the extent of viable myocardium with cardiovascular MR (CMR) imaging, and to explore the feasibility of assessing the microvascular function of the residual viable myocardium in the infarct zone.

Materials and Methods

Study population Twenty-nine patients who had first MI caused by single-vessel disease were enrolled. All patients received successful primary PCI, defined as TIMI 3 flow and final residual luminal stenosis $\leq 20\%$.

Image acquisition The CMR study was performed under ECG-gating on 3T MR system. All subjects received first-pass contrast-enhanced (FPCE) MRI at rest and during hyperemia for myocardial perfusion measurement and delay-enhanced (DE) MRI for myocardial scarring. For FPCE MRI, three short-axis images at basal, mid LV and apical levels were acquired using SR-Turbo FLASH pulse sequence (TR/TE/TI/flip angle = 1.08 ms/0.98 ms/90 ms/10°; slice thickness = 8 mm; field of view (FOV) = 400 mm \times 240–300 mm; matrix size = 192 \times 144). After the first FPCE study, dipyridamole (0.14 mg/kg/min of dose) was infused for 4th min and a second FPCE study during hyperemic status was performed at 7th min. After the FPCE studies, contrast medium was given by slow infusion, amounting to a total dose of 0.2 mmole/kg body weight. Ten minutes after the Gd administration, DE MRI was performed at the same short-axis planes as those in FPCE. The pulse sequence was inversion-recovery prepared segmented turboFLASH sequence (TR/TE/flip angle = 1.6 ms/1.52 ms/20°; FOV = 240 \times 350 mm; matrix size = 205 \times 256). The inversion time was adjusted to null the normal myocardium and was typically in the range of 200–300 ms.

Image analysis

1. **Residual viable myocardium quantification:** From DE MRI, the infarct zone was defined by a sector bounded by two ends of hyperenhanced region and the rest of the myocardium was defined as the remote zone. In the infarct zone, a pixel was counted as scar if its signal-intensity (SI) was higher than two standard deviations (SD) above the mean SI of the remote zone. The residual viable-myocardial ratio, VMR, of the infarct zone was quantified by the percentage of the total pixels of non-scar pixels relative to the total pixels in the infarct zone:

$$VMR = \frac{\text{total pixels of infarct zone - scar pixels}}{\text{total pixels of infarct zone}} \times 100\%$$

2. **Regional blood flow quantification:** Myocardial blood flow (MBF) was measured in the same zones as those determined on DE MRI (Fig 1). The MBF at rest and hyperemic status, in the unit of ml/min/g, was quantified by model-independent deconvolution method (2). (MPR = hyperemic MBF/rest MBF)
3. **Estimation of microvascular function in residual viable myocardium:** The microvascular function in the residual viable myocardium was evaluated by estimating the MBF augmentation during maximal vasodilatation. Regional MBF in the infarct zone (Pregion) can be considered as a result of linear combination of the perfusion in the scar tissue (Pscar) and the perfusion in the viable myocardium (Pviable). Pviable can be estimated as follows: $Pviable = (Pregion - (1-VMR)Pscar) / VMR$. Assuming that the scar tissue failed to augment during the hyperemic challenge and that MBF measured during resting is similar for both residual viable myocardium and scar tissue, Pscar can be replaced by the rest MBF in the infarct zone. In this way, Pviable can be estimated based on the measured regional MBFs at rest and stress as well as VMR.
4. **Relationships between regional myocardial perfusion and extent of residual viable myocardium:** To study the relationships between regional myocardial perfusion and the extent of the residual viable tissue in the infarct zone, MBF during resting, MBF during hyperemic status and MPR were correlated against VMR.

Results

The mean VMR of the infarct zone determined from DE MRI was $45.33 \pm 19.94\%$ (range 6.56–87.20%). Compared with the non-infarct remote zone, infarct zone showed significant impairment of regional MBF at rest (0.966 ± 0.271 vs. 1.151 ± 0.282 ml/min/g; $P=0.024$), during hyperemia (1.789 ± 0.732 vs. 2.753 ± 0.806 ml/min/g; $P<0.0001$), and MPR (1.923 ± 0.678 vs. 2.486 ± 0.836 ; $p<0.0001$). The microvascular function of the residual viable myocardium was found to be relatively conserved (Fig. 2). The relationships between myocardial perfusion and the extent of viability in the infarct zone showed that hyperemic MBF ($r=0.385$; $p=0.039$) and MPR ($r=0.434$; $p=0.018$) were significantly correlated with VMR.

Conclusion

In this study, we demonstrated that FPCE MRI in combination with DE MRI could effectively quantify the scar content, MBF and MPR in the infarct myocardium. Our results demonstrated significant impairment of the regional MBF in the reperfused infarct zone, and showed that this impairment was associated with the extent of scar tissue. In addition, the estimated microvascular function of the residual viable myocardium showed no significant difference from that in the remote zone. These data suggested that the microvascular function was generally impaired in the reperfused infarct zone, but more viability salvaged could preserve more microvascular function.

References

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2. Jerosch-Herold M et al. Med Phys 2002;29:886–897

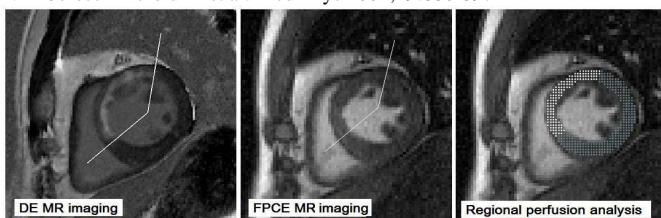


Fig 1. FPCE MRI in the infarct zone and remote zone (middle) was defined according to the same sectors determined from DE MRI (left). Regional MBF was measured from the infarct zone and remote zone (right).

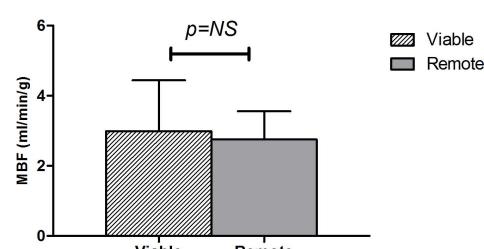


Fig 2. Hyperemia myocardial blood flow (MBF) in residual viable myocardium was not significantly different from that in the remote zone.