Microvascular Obstruction After Myocardial Infarction Affects Vasodilated Flow: A Quantitative Perfusion Study

W. Ingkanisorn[1], L-Y. Hsu[1], A. H. Aletras[1], A. E. Arai[1]

[1]Laboratory of Cardiac Energetics, NHLBI, National Institutes of Health, Bethesda, Maryland, United States

Background Despite successful epicardial revascularization for the treatment of acute myocardial infarction, an open infarct-related vessel does not ensure normal perfusion at the myocardial level [Int J of Card 2004; 96:389-96]. Although the "no reflow" phenomenon can be detected through TIMI frame counts, microvascular obstruction can be missed during catheterization. Furthermore, microvascular obstruction may contribute to adverse ventricular remodeling and worse prognosis. MRI first-pass gadolinium perfusion imaging is a high-resolution technique that can reproducibly quantify myocardial perfusion.

Hypothesis We hypothesized that despite successful acute percutaneous intervention (TIMI grade 3 flow), myocardial perfusion in regions of myocardial infarction would be abnormal during dipyridamole stress as quantified by a vasodilator dual-bolus first-pass MRI perfusion technique.

Methods **Study population:** Twelve patients were imaged following an acutely reperfused acute myocardial infarction (AMI). All patients underwent primary percutaneous coronary intervention (PCI) for the treatment of AMI, and all patients had TIMI Grade 3 flow at the end of the procedure. The patients then underwent dipyridamole MRI after successful PCI. **Imaging:** All studies were performed on a GE 1.5 T CV/i scanner, using a 4-element cardiac phased-array coil. Cine function, dipyridamole perfusion, and gadolinium delayed enhancement were performed as part of each study. The myocardial perfusion exam was performed after infusion of intravenous dipyridamole (0.56 mg/kg) using a volumetric saturation recovery, segmented hybrid echoplanar sequence (3 mm in-plane resolution, TI 60 ms, saturation pulse 90 degrees, readout RF flip angle 20 degrees). Three slices (basal, mid, and apical) were acquired gated to every R-R interval over 50 heart beats. A dual-bolus first pass perfusion technique using Gadolinium-DTPA 0.005 mmol/kg and 0.1 mmol/kg was performed. **Quantitative Analysis:** Regions of interest defining the endocardium and epicardium were performed on the perfusion slice (through all 50 phases) corresponding to the region of infarction as defined by the gadolinium delayed enhancement study, and the perfusion slice was divided either into 6 regions (at the basal and mid levels) or 4 regions (at the apex), according to a 16-segment model. Fermi function deconvolution was performed to determine myocardial perfusion. The territory of maximal infarction was compared to a reference normal segment in which there was no significant stenosis.

Results The mean age of the patients was 54 ± 10 years (11 males, 1 female), and the average time between the patients' AMI and the dipyridamole MRI was 12.6 ± 13.2 days. Myocardial infarctions occurred in all coronary territories: 4 left anterior descending, 4 circumflex, and 4 right coronary artery infarctions. The mean left ventricular ejection fraction was 50.5 ± 9.9%, and 5 of the 12 patients had overt visual evidence of microvascular obstruction on perfusion and delayed enhancement studies. The mean myocardial perfusion flow within a normal segment was 2.78 ± 1.20 mL/min/g, whereas, the mean flow within an infarcted segment was 1.87 ± 0.69 mL/min/g ($p < 0.001$) (See Figures 3 and 4). Contrast enhancement ratio was not statistically different between infarcted and normal segments but upslope ratio was significant. Ten of the 12 patients had a decrease of ≥ 20% in myocardial perfusion between the normal segments and the infarcted segments.

Conclusion Despite TIMI Grade 3 revascularization of an infarct-related coronary artery, territories of acute infarction have significantly decreased myocardial perfusion that is quantifiable by vasodilator dual-bolus first-pass MRI perfusion technique.

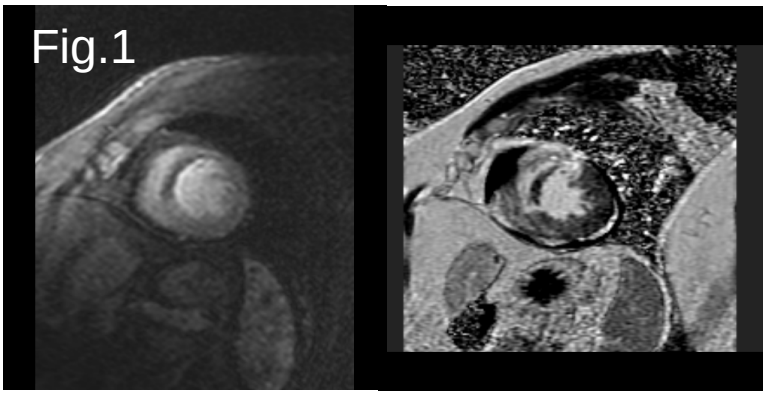

Figure 1. Standard FGRE-EPI perfusion study (left) with the corresponding delayed enhancement image (right). Note the evidence of microvascular obstruction within the anteroseptum. Within the normal region of the inferior wall, myocardial perfusion was quantified as 2.72 mL/min/g. Within the infarcted anteroseptal territory, perfusion was quantified as 1.50 mL/min/g.

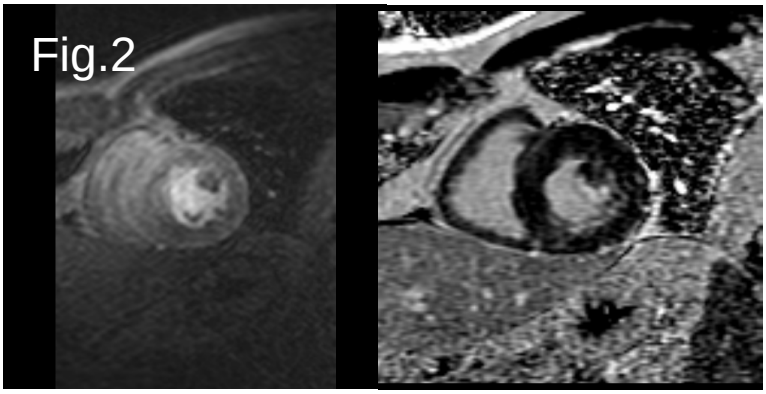

Figure 2. On the left, there is a qualitatively visible perfusion defect of the inferior and inferolateral walls. On the right is a small subendocardial infarction of the same region. Within the reference normal region of the anterior wall, the perfusion was quantified as 1.13 mL/min/g. The infarcted region had a measured perfusion of 0.87 mL/min/g.

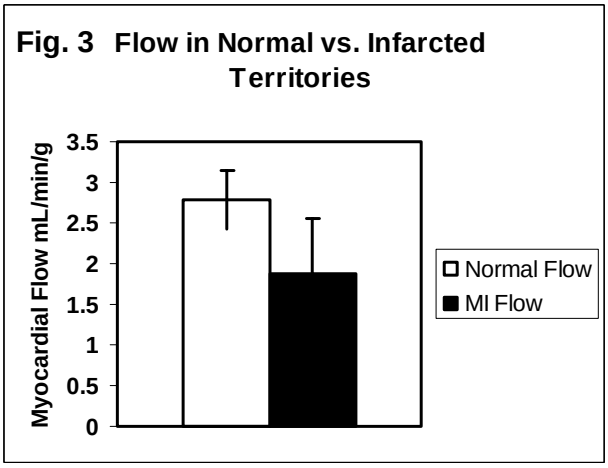

Fig. 4 Summary of mean quantitative perfusion findings in normal and AMI regions

	Flow (mL/min/g)	Contrast enhancement ratio	Upslope Ratio
Normal	2.79 ± 1.2	1.61 ± 0.28	0.79 ± 0.25
Infarct	1.87 ± 0.69	1.19 ± 0.48	0.60 ± 0.37
p value	<0.001	0.12	0.002