

Effects of exercise training on myocardial perfusion reserve in patients with post myocardial infarction

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Introduction

Exercise-based rehabilitation has been demonstrated to improve myocardial perfusion and reduce recurrent cardiovascular events (1). Although it has been proved that exercise-induced angiogenesis increases the capillary surface area and blood flow in the ischemic myocardium, the effect on the non-transmural infarct myocardium is unclear. In this study, we aimed to investigate the perfusion response to exercise rehabilitation in patients with post-myocardial infarction (MI).

Materials and Methods

Study protocol A total of 39 patients presenting with a first acute MI at least 6 months ago were recruited and randomized into two groups, a training group (N=20, age: 52±8 yrs) who attended a 3-month exercise program and a control group (N=19, age: 52±9 yrs; p=NS) who did not join the exercise program. The training group received two MR studies, one before and one after the 3-month exercise program. The control group also received two MR studies which were three months apart. The first MR studies served as a baseline for comparing with the second MR studies.

Cardiac rehabilitation protocol Exercise sessions were performed three times a week for 3 months. The exercise included a 5-min warm up, a 20-min bicycle exercise combined with treadmill walking, and a 5-min cool down. The exercise intensity was prescribed according to the ventilatory threshold (51–59% of maximal heart rate reserve) and the Borg's scale 12 rating of perceived exertion of 12 to 13 (fairly light to somewhat hard).

MR acquisition All subjects underwent both rest and stress first-pass contrast-enhanced MR studies on a 3T MR scanner (Trio, Siemens, Germany). Three short-axis slices at basal, mid left ventricle (LV) and apical levels were acquired using an SR-prepped Turbo FLASH pulse sequence. Right after the scanning started, gadolinium-DTPA (0.025mmole/kg) was bolus injected via left antecubital vein at a rate of 4–6ml/sec. The stress study was performed approximately 10 min after the rest study. Vasodilator (dipyridamol, 140µg/kg/min) was infused intravenously for 4 min and the image acquisition began at the 7th min when the maximal vasodilatation was achieved. Gadolinium-DTPA (0.2 mmole/kg) was administered after the perfusion study, and late gadolinium enhancement (LGE) MR imaging was performed 10 min after to assess the myocardial viability.

Image analysis LV myocardium were segmented semi-automatically and divided into infarct and remote segments according to the LGE results (Fig. 1). Transmural infarct segments were excluded to avoid confounding bias due to thin wall thickness. After the background signal correction, myocardial perfusion was quantified by measuring the maximum upslope (Upslope) of the first pass signal-time curve from the LV myocardium, normalized to the Upslope in the LV cavity. Myocardial perfusion reserve (MPR) index was calculated by dividing the Upslope at stress by the Upslope at rest. In each subject, MPRs calculated at two MR studies were compared segment by segment. Comparison was also made between different segments in the same MR study.

Statistical Analysis

Data were presented as mean±SD. Paired difference in MPR between two MR studies were tested using a paired Kolmogorov-Smirnov test segment by segment. The differences in MPR between infarct and remote myocardium were also analyzed using a 2-sample Komogorov-Smirnov test. Statistical significance was considered if p<0.05.

Results

As listed in Table 1, baseline MPR showed no significant difference between control and training group at the infarct segments (1.44±0.37, 1.38±0.36; p=0.82) and at the remote segments (1.52±0.41, 1.49±0.39; p=0.79). After 3 months, significant difference was found at both infarct segments (1.44±0.35, 1.73±0.47; p=0.05) and remote segments (1.47±0.32, 1.96±0.54; p=0.01). Figure 2 showed that after 3 months of exercise training, the training group had a significant increase in MPR at the remote segments (1.49±0.39, 1.96±0.54; p=0.03) and a moderate increase at the infarct segments (1.38±0.36, 1.73±0.47; p=0.08).

Discussion and Conclusions

Regular physical activity has been documented to improve symptoms, myocardial perfusion and reduce mortality in patients with coronary heart disease (2). Using MRI, we found that the improvement in perfusion in the remote myocardium was more significant than that in the non-transmural infarct myocardium. Our results support a previous study using thallium-201 scintigraphy (1), and further characterize the difference in perfusion improvement between the remote and non-transmural infarct regions.

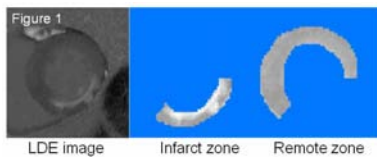


Figure 1. The LGE image (left) and its infarct and remote segments (right).

Table 1

		Baseline	3-month
Infarct	Trained group	1.38±0.36	1.73±0.47
	Control group	1.44±0.37	1.44±0.35
		p=NS	p=0.05*
Remote	Trained group	1.49±0.39	1.96±0.54
	Control group	1.52±0.41	1.47±0.32
		p=NS	p=0.01*

The MPR changes between trained group and control group at infarct and remote myocardium

References

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- Hambrecht R, Adams V, Erbs S, et al. Regular physical activity improves endothelial function in patients with coronary artery disease by increasing phosphorylation of endothelial nitric oxide synthase. *Circulation* 2003;107(25):3152-3158.

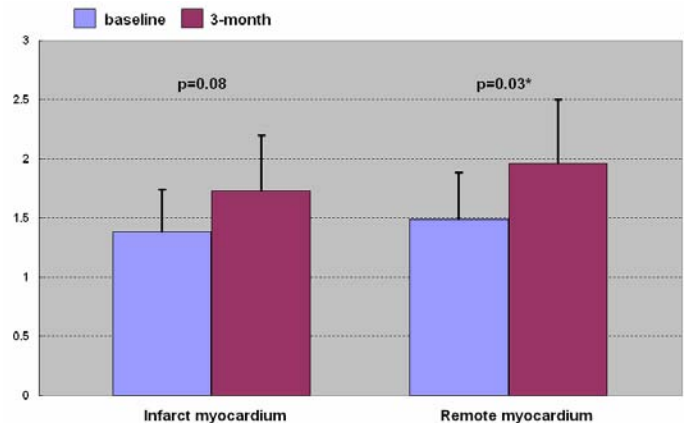


Figure 2. After 3 months of exercise, the training group had a significant increase in MPR in remote myocardium (p=0.03) and a moderate increase in infarct myocardium (p=0.08).