Microvascular Obstruction Related to Primary Angioplasty by Magnetic Resonance Imaging with Acute Myocardial Infarction

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Introduction: The failure of adequate microvascular perfusion in acute myocardial infarction (AMI) despite epicardial coronary artery recanalization has been termed no reflow phenomenon. No reflow phenomenon can be caused by any type of arteriolar or capillary occlusion within infarcted area. The precise etiology of this microvascular obstruction in AMI is not completely understood. However microthromboembolism is postulated as an etiology of microvascular obstruction after primary coronary intervention, angioplasty with stenting. Primary angioplasty with stenting without preceding thrombolysis is effective in restoring perfusion, but the presence of microvascular obstruction after the procedure has been associated with poor clinical outcome. With emergence of contrast-enhanced magnetic resonance imaging(MRI), microvascular obstruction related to primary angioplasty with stenting can be detected easily as hypoenhanced region within hyperenhanced lesion in contrast-enhanced MRI. We have investigated the microvascular obstruction related to primary angioplasty with stenting in AMI by means of contrast-enhanced MRI.

Methods: Consecutive 32 patients with AMI were enrolled.AMI was determined by their typical chest pain and CK-MB profile. 32 patients were divided into two groups. A group were treated by primary angioplasty with stenting without using protecting device for microembolization on admission (PA group, n=20, aged 61.8±9.5) and the other group initially treated by thombolytic therapy (TT group, n=12, aged 55.9±10.9). MRI was performed from 0 to 20 days (mean 4.4 days) after primary angioplasty and from 0 to 27 days (mean 6.1 days) after onset of symptom in TT group. The patients were imaged with a 1.5-T clinical imaging unit. (Gyroscan intera; Philips Medical systems, Best, the Nerthelands) Perfusion MRI was performed with TFE-EPI single shot pulse sequence in 3-4 short-axis slices for 50-60s with contrast-enhancement. Contrast (Omniscan; Nycomed Ireland Ltd, Cork, Ireland, 0.4cc/kg) injection rate is 3 - 4ml/s. Delayed-enhanced MRI encompassing the entire left ventricle was performed with a multi-shot, TFE, inversion prepulse , 10 minutes after contrast administration (TR=5.4ms, TE=1.6ms, FA=15, voxel size=1.37x1.37x10mm). An obvious perfusion deficit was evidenced by a darker region at infarcted region in perfusion MRI. In the center of affected myocardium, there is often a core of myocardium that does not enhance injection in delayed-enhanced MRI, remaining at the same signal intensity as the normal myocardium prior to contrast. This area was defined as “hypoenhanced zone” by microvascular obstruction (fig.1). The prevalence of microvascular obstruction in each group was compared. Infarct-size, level of cardiac enzymes(CK-MB and troponin-T) and degree of stenosis of infarct-related artery on coronary angiography were compared between the PA and TT groups and between the group with microvascular obstruction and the rest.

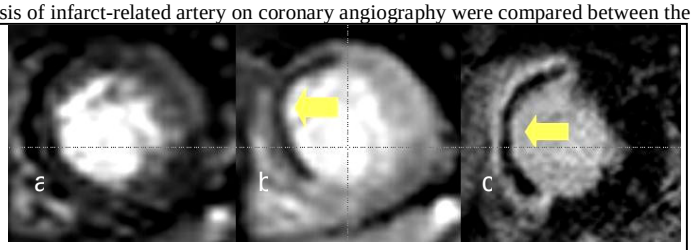

Fig1. Microvascular obstruction with contrast-enhanced MRI. Hypoenhanced region in early perfusion through delayed enhancement was defined as presence of microvascular obstruction. (a) before enhancement, (b) early perfusion, (c) delayed enhancement

Results: Nine patients (45%) in the PA group showed hypoenhancement within hyperenhanced region in delayed-enhancement MRI, which showed early hypoenhancement in perfusion study too. No patients in the TT group showed hypoenhancement in delayed-enhancement imaging even in the case of hypoenhanced region in perfusion study. The microvascular obstruction was observed on the exactly same territory of primary angioplasty and stenting. The degree of stenosis of infarct-related artery was not different between the TT and the PA group(92±9%, 89±11%, p=0.5). Peak value of CK-MB and Troponin-T were significantly higher in the PA group than in the TT group (330.8±270.0>100.46±122.60 p=0.009), (5.72±7.97>0.60±0.70, p=0.021). However, the level of the enzymes was not significantly different between in patients with microvascular obstruction and in patients without in the PA group (CK-MB: 360.7±266.0, 306.3±283.7, p=0.666), (Troponin-T: 4.79±6.93, 4.40±8.19, p=0.913). Therefore CK-MB and Troponin-T did not predict the presence of microvascular obstruction. Totally-transmural infarction determined by MRI (n=5) was observed only in patients revealing microvascular obstruction. Transmural extent of infarct-size measured in delayed images in patients with microvascular obstruction was larger than that in the TT group and that in patients without microvascular obstruction in the PA group (84±19%> 55±31%, 62±13% respectively, p<0.05). From this we could conclude that microvascular obstruction was linked to larger infarct-size. However, while microvascular obstruction was absent even when the transmural extent was as high as 90% in TT group, microvascular obstruction was present in 40% transmural extent in PA group. In order to control transmural extent of infarct-size, the patients having 20-80% transmural extent of infarction in each group (10 of 20 in PA group, 10of 12 in TT group) were compared. The transmural extent was not statistically different (63±11% in PA group and 55±22% in TT group (p=0.23)). While 3 of the 10 patients in PA group showed mircovascular obstruction, none from TT group did.

Conclusions: Microvascular obstruction was more prevalent with primary angioplasty with stenting after acute myocardial infarction than with thrombolytic therapy only. The occurrence of microvascular obstruction was partially related to the difference of transmural extent of infarction. However, microvascular obstruction was more prevalent with primary angioplasty than with thrombolytic therapy even under control of infarct size. Primary angioplasty related microvascular obstruction is present and it can be detected with contrast-enhanced MRI, perfusion and delayed imaging.

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