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Coronary collaterals: The role of MCP-1 during the early phase of acute myocardial infarction

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Abstract

Background: The collateral growth (arteriogenesis) of an individual may vary from complete to absent during the early phase of acute myocardial infarction (AMI). However, the mechanisms underlying the large differences in the extent and adequacy of collateralization remain unclear. We hypothesized that shear stress-induced activation of monocyte chemoattractant protein-1 could potently contribute to the development of coronary collaterals during the early phase of AMI.
Methods: We enrolled forty patients with AMI who did not receive reperfusion therapy within 24 h after the onset of chest pain and who also underwent coronary angiography (CAG) from 1 to 7 days after admission (mean duration: 3.6±2.2 days). The grades of the collateral development were angiographically defined and grouped according to the grade of collaterals as absent (score 0, $n=20$) or well-developed (score 2, $n=20$) collateral circulation. The plasma concentrations of vascular endothelial growth factor (VEGF), endostatin, monocyte chemoattractant protein-1 (MCP-1), and stromal cell-derived factor-1 (SDF-1) were assessed by enzyme-linked immunosorbent assay and then these values were compared between the two groups.
Results: There were no differences in the demographic and angiographic characteristics except for the number of total occlusion in culprit lesion. The plasma MCP-1 levels were significantly higher in the group with well-developed collateral circulation compared to the group with absent collateral circulation (262±216 vs. 151±88 pg/ml, respectively, $p=0.043$). However, the plasma levels of VEGF, endostatin and SDF-1 were not different on comparisons between the groups (VEGF; 369±377 vs. 324±363 pg/ml, endostatin; 1.74±1.71 vs. 1.49±1.15 ng/ml, SDF-1; 1806±508 vs. 2091±772 pg/ml, respectively).
Conclusion: During the early phase of AMI, the plasma levels of MCP-1 were significantly increased in the patients with well-developed collateral circulation as compared to those patients with absent collateral circulation. These findings suggested that the shear stress-induced overexpression of MCP-1 contributes significantly to the development of coronary collaterals during the early phase of AMI.
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Keywords: Collateral circulation; Arteriogenesis; Myocardial infarction; Monocyte chemoattractant protein-1

1. Introduction

The formation of coronary collaterals is an adaptive response of the coronary vascular system to arterial occlusion. This process is involved in restoring coronary blood flow and salvaging the myocardium at ischemic regions. Previous studies have shown that the presence of collaterals may limit the size of an infarct, preserve the viability, and prevent ventricular aneurysm formation during an episode of acute coronary occlusion [1–4]. During the early phase of acute myocardial infarction (AMI), patients will show marked angiographic heterogeneity in collateral formation that is independent of the status of coronary artery occlusion [5]. However, the mechanism underlying these large differences

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between individual patients in the extent and adequacy of collateralization remains unclear despite of our increased understanding of the cellular and molecular processes involved in collateral development.

The sudden occlusion of an epicardial coronary artery creates a pressure gradient between the arteries proximal to the occlusion and distal from the occlusion, and this increases the blood flow through the preexisting arterioles. This shear stress activates the endothelium of the preexisting arterioles and it stimulates the production of various growth factors and cytokines, including monocyte chemoattractant protein-1 (MCP-1), vascular endothelial growth factor (VEGF), and etc [6–12]. This interplay of cells, growth factors and cytokines results in arteriogenesis, which is a process of opening and maturation of preexisting small arterioles; this is thought to play a major role in the formation of angiographically visible collaterals in patients with AMI and who do not undergo reperfusion therapy.

Among the various growth factors and cytokines, we hypothesized that MCP-1 activation would more potently contribute to the development of angiographically visible coronary collaterals during the early phase of AMI than any other growth factors and cytokines. Thus, we compared the plasma levels of MCP-1, VEGF, endostatin and stromal cell-derived factor-1 (SDF-1) between the patients with angiographically visible collaterals and those patients without collaterals.

2. Methods and materials

2.1. Patient selection

Among the patients with AMI at Kangnam St. Mary's hospital who did not receive reperfusion therapy within 24 h after the onset of chest pain, 40 patients who underwent coronary angiography (CAG) from 1 to 7 days after admission (mean duration: 3.6±2.2 days) were selected and they were then divided into 2 groups: group 1 had an angiographically demonstrated absence of collateral circulation (score 0, $n=20$) and group 2 had well-developed (score 2, $n=20$) collateral circulation. AMI was diagnosed based on chest pain that persisted for 30 min, elevation of the serum creatine kinase-MB fraction (CK-MB) to more than twice the upper limit of normal, and elevation of the serum troponin I level above the upper limit of normal according to the local quantitative or qualitative assays. ST elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI) were included as AMI.

The criteria for exclusion from the study were the following: 1) patients with AMI who received thrombolysis or primary percutaneous coronary intervention within 24 h after the onset of chest pain; 2) patients who suffered with recent MI or old MI that happened more than 7 days after the onset of chest pain; 3) patients with collateral formation due to a non-culprit lesion, as seen on the CAG; 4) patients who previously underwent CABG.

2.2. Blood sampling and coronary angiography

Immediately before coronary angiography, a blood sample was obtained from each patient through the introducer sheath that was placed in the femoral artery, and this sample was aliquoted in a 10 ml sterile tube (anticoagulant: EDTA) and then processed within 30 min.

Standard angiography, with ≥4 views of the left coronary system and 2 views of the right coronary artery, was used for interpretation. The collateral scoring system we used was modified from the previously described Thrombolysis In Myocardial Infarction Scoring System [2]. The ranking from 0 to 2 was based on the presence of collateral vessels and opacification of the recipient vessel. A grade of 0 was given for no visible collaterals, a grade of 1 was given for visible collaterals, but there was no filling of the recipient epicardial vessels, and a grade of 2 was given for filling (partial or complete) of the recipient epicardial vessels by the collaterals. A separate angiographer, who was blinded to the initial reading, reviewed the angiograms. For the cases of disagreement, a third angiographer, who was blinded to the initial two readings, served as an arbitrator.

2.3. Measurement of growth factors

The whole blood samples were centrifuged at 3000 rpm for 10 min at room temperature. The plasma supernatant was removed, frozen in liquid nitrogen and then stored at −80 °C in aliquots that were used for the growth factor level assays. Standard enzyme-linked immunosorbent assay kits (R&D Systems, Inc., USA) were used to determine the plasma growth factor levels of VEGF, endostatin, MCP-1 and SDF-1.

2.4. Data collection and statistical analysis

The clinical data was obtained from a comprehensive review of each patient's medical record and with using the established criteria for hypertension, diabetes mellitus, hyperlipidemia and myocardial infarction. Current smoking was defined as the active use of tobacco products at the time of enrollment into the study. Analysis between groups for statistically significant differences in the categorical data was performed using the chi-square test. The continuous variables are presented as mean±standard deviation (SD), and they were compared by Student's T test. A p value below 0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics and the cardiac enzymes

Table 1 summarizes the patient's profiles and the cardiac enzymes by the group. The mean age and the proportion of patients with angina or prior MI were higher in the group with well-developed collateral circulation, but the differences

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