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RELEASE OF GRANULOCYTE COLONY-STIMULATING FACTOR FOR STEM CELL MOBILIZATION INCREASES IMMEDIATELY AFTER INFARCT-RELATED ARTERY REPERFUSION IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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BACKGROUND/AIMS: Myocardial infarction results from myocardial cell hypoxia involving the epicardial coronary microcirculation, collateral vessel formation and angiogenic factors. Angiogenesis is controlled by angiogenic cytokines such as vascular endothelial growth factor (VEGF), stem cell factor (SCF), granulocyte colony-stimulating factor (GCSF), and stromal cell-derived factor-1 α (SDF-1α). However, the precise mechanism of these cytokines in acute myocardial infarction and the effects of coronary reperfusion on the circulating levels of angiogenic factors are still unknown.

METHODS & RESULTS: First we developed a mouse femoral artery angioplasty model study involving two groups. The first group received total ligation of the right lower limb. The second, reperfusion group was subjected to tissue ischemia lasting 90 seconds without ligation. Levels of VEGF, SDF-1α, SCF, G-CSF, and the numbers of circulating endothelial progenitor cells (EPCs) were found to be significantly increased in the group receiving total ligation only. The reperfusion group was found to have significantly higher levels of SDF-1α, SCF and G-CSF and higher circulating EPC numbers at the point of peak concentrations. Elevation of EPC numbers coincided with the increase in G-CSF levels, suggesting a correlation between G-CSF and progenitor cell mobilization. Secondly, we designed a human acute myocardial infarction and percutaneous coronary intervention therapy observation study. Here, compared with the unsuccessful reperfusion group, the SDF-1α, SCF, G-CSF, and circulating EPCs significantly increased immediately after reperfusion of the involved artery.

DISCUSSION/CONCLUSION: Angiogenic factors and circulating EPC numbers increase in the acute stage of mouse femoral artery ligation and human myocardial infarction. Our findings suggest that G-CSF plays a critical role in EPC mobilization after reperfusion therapy.

Key words: Myocardial infarction, Cytokines, Reperfusion