MYOCARDIAL ISCHEMIA AND INFARCTION

MAST CELL COULD REGULATE THE SURVIVAL OF CARDIOMYOCYTES IN A MYOCARDIAL INFARCTION RAT MODEL

ACC Poster Contributions
Georgia World Congress Center, Hall B5
Monday, March 15, 2010, 9:30 a.m.-10:30 a.m.

Session Title: Mechanisms of Ischemic Injury and Protection
Abstract Category: Myocardial Ischemia/Infarction--Basic
Presentation Number: 1157-292

Authors: Youngkeun Ahn, Jin Sook Kwon, Yong Sook Kim, Ae Sin Cho, Mun Hwa Hong, Myung Ho Jeong, Seung Uk Lee, Jeong Gwan Cho, Jong Chun Park, Jung Chaee Kang, Department of Cardiology, Chonnam National University Hospital, Gwangju, South Korea, Gwangju Christian Hospital, Gwangju, South Korea

Background: Recent study demonstrated that mast cells have various effects on the progression after acute myocardial infarction (MI). At acute phase, MI is involved with inflammation. This inflammation is regulated by neutrophil, monocyte, macrophage, and mast cell. We elucidated the effects of mast cell in a MI rat model.

Methods: To evaluate the effects of mast cell, we isolated the cell from rat peritoneal cavity and prepared mast cell granule (MCG) from purified mast cell by stimulation with compound 48/80. In vitro assay, we treated MCG to rat neonatal cardiomyocyte (rCMC) and exposed the cells into hypoxic condition (N2:95 %, CO2:5 %) and after 6 hours it returned to normoxic condition.

Results: In Propidium Iodide (PI) & Anexin assay, MCG treatment decreased death (necrosis and apoptosis) of rCMC. Survival cell ratio was 86.3±3.5 % in normoxic condition, 65.2±3.2 % in hypoxic condition, and 73.3±7.1 % in hypoxic condition with MCG treatment. At the same cells with immunofluorescence staining, by FACS analysis and western blot analysis, hypoxic condition decreased troponin-I expression to 60 % level of normoxic condition. However, MCG treatment returned expression of troponin-I to the level of normoxic condition. And MCG treatment increased the expression of phosphorylation of Akt, ERK, and c-kit receptor. In vivo study, in a MI rat model, MCG injection into the infarct area improved left ventricular (LV) function and increased capillary density 3 times more than that of control rats (phosphate buffered saline injection) (p<0.05 in each).

Conclusions: The role of MCG on the regulation of angiogenesis and cell survival could result in improved LV function after MI.