A20.E192
JACC March 9, 2010
Volume 55, issue 10A

CARDIAC FUNCTION AND HEART FAILURE

GALECTIN-1 DEFICIENT MICE HAVE ABNORMAL BASELINE CARDIAC FUNCTION AND WORSE REMODELING AFTER ACUTE MYOCARDIAL INFARCTION

ACC Poster Contributions
Georgia World Congress Center, Hall B5
Sunday, March 14, 2010, 9:30 a.m.-10:30 a.m.

Session Title: Myocardial Function/Heart Failure---Basic/Molecular--Diverse Signaling Pathways
Abstract Category: Myocardial Function/Heart Failure---Basic/Molecular
Presentation Number: 1015-69

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Background: Acute myocardial infarction (AMI) induces an inflammatory response leading to heart failure (HF). Galectin-1, a beta-galactoside binding lectin, is an immunomodulator that maintains T cell homeostasis and enhances production of interleukin-10. The aim of this study was to evaluate the role of Galectin-1 in normal cardiac function and post-AMI ventricular remodeling in mice.

Methods: Male mice with deletion of the gene encoding Galectin-1 (Lgals1, Gal-1 KO mice) or age and sex matched mice with similar genetic background (WT) underwent experimental AMI by surgical ligation of a coronary artery (n=6 for each group). Transthoracic echocardiogram was performed at baseline (before surgery) and 7 days after surgery.

Results: Gal-1 KO mice had a 20% larger left ventricular (LV) end-diastolic diameter (EDD), a 6% greater LV end-systolic diameter (ESD) and a 20% lower LV fractional shortening (FS), without any difference in body weight. Seven days after AMI both groups showed cardiac enlargement and a decrease in cardiac function with Gal-1 KO mice showing a significantly greater increase in LVEDD than WT (see figure).

Conclusion: Galectin-1 is an innate immune modulator that plays a protective role in normal cardiac homeostasis and in post-infarction remodeling. Modulation of the immune response using exogenous Galectin-1 may be a viable strategy to prevent HF after AMI.