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Heart Failure and Cardiomyopathies

DIPEPTIDYL PEPTIDASE-4 INHIBITOR ATTENUATES CARDIAC DYSFUNCTION AND ADVERSE REMODELING AFTER MYOCARDIAL INFARCTION

Poster Contributions
Poster Hall B1
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Session Title: Pathophysiology of Cardiac Remodeling
Abstract Category: 13. Heart Failure and Cardiomyopathies: Basic
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Background: Adverse cardiac remodeling after myocardial infarction (MI) leads to progressive heart failure (HF). Since diabetes increases risks of MI and HF, roles of antidiabetic drugs on the heart have been extensively investigated. DPP-4 inhibitor is a new antidiabetic drug that exerts cardioprotection. However, its role on cardiac function and remodeling in chronic MI is unclear. We hypothesized that DPP-4 inhibitor reduces adverse cardiac remodeling and improves cardiac function in chronic MI rats.

Methods: Male Wistar rats (n=36) with chronic MI induced by LAD ligation were divided into 6 groups to receive vehicle, vildagliptin (3 mg/kg/d), metformin (30 mg/kg/d), enalapril (10 mg/kg/d), combined metformin and enalapril, or combined vildagliptin and enalapril for 8 weeks. Plasma MDA, heart rate variability (HRV), pathological and biochemical studies for cardiac remodeling, cardiac function, and apoptosis were determined.

Results: Chronic MI rats had increased oxidative stress, depressed HRV, adverse cardiac remodeling indicated by cardiac fibrosis in peri-infarct area, and LV dysfunction. Treatment with vildagliptin and enalapril significantly decreased oxidative stress and fibrosis, and improved HRV and %FS (Figure). However, these benefits were not found in metformin group.

Conclusion: Vildagliptin, but not metformin, exerts similar cardioprotective effects as enalapril in attenuating oxidative stress and cardiac remodeling, and improving cardiac function in chronic MI rats.