(control group, n=30). Tei-index, LV remodelling (LV weight/body weight ratio, interstitial fibrosis) was evaluated and cardiomyocyte apoptosis was detected with the TUNEL method. In addition, the expression levels of CTGF and nicotinamide adenine dinucleotide phosphate (NADPH) in myocardial were detected.

Results Infarct size was similar among the groups 30 d after MI Tei-index (Gs-Rb1 vs simple MI vs control: 0.47±0.03 vs 0.61±0.07 vs 0.39±0.05; p<0.05) and LV remodeling (LV weight/body weight ratio: 4.1±0.3 vs 4.3±0.2 vs 4.0±0.3 mg/g, p<0.05; interstitial fibrosis: 3.4±1.3 vs 3.7±1.6 vs 3.2±1.1%, p<0.05) was significantly improved by Gs-Rb1. Gs-Rb1 significantly inhibited cardiomyocyte apoptosis (3.2±0.4% vs 4.7±0.6%; p<0.05), cardiac NADPH oxidase activation (Gs-Rb1 vs simple MI vs control: 3.97±0.21 vs 4.54±0.22 vs 3.11±0.26 RLU/mg protein) and CTGF levels (0.77±0.25 vs 1.03±0.21, p<0.05) compared with simple MI group or control group.

Conclusions Ginsenosides-Rbl improves LV dysfunction and prevents LV remodeling early after MI, in which the effects of Gs-Rbl, inhibiting CTGF expression and NADPH oxidase activation, and cardiomyocyte apoptosis, may be the underlying mechanisms.

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GINSENOSIDES-RBL EXERTS BENEFICIAL EFFECTS ON LEFT VENTRICULAR DYSFUNCTION AND REMODELLING EARLY AFTER MYOCARDIAL INFARCTION

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Objectives The aim of this study was to investigate whether Ginsenosides-Rbl (Gs-Rb1) has improved left ventricular (LV) dysfunction and remodelling early after myocardial infarction (MI).

Methods Sixty male 8-week-old Wistar mice with extensive anterior MI were randomly divided into Gs-Rb1 group (20 mg/kg/day, n=30) and simple MI group (n=30), being intramyocardially administered, for 30 days starting on day 1 after surgery. Age-matched male rats served as sham-operation group