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PROTECTIVE EFFECTS OF OPTIMAL PRESCRIPTION OF JIASHEN ON MYOCARDIAL INFARCTION WITH THE SUPPRESSION OF RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM IN RATS

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Objectives We tested the hypothesis that optimal prescription of Jiashen (OPJSH), a traditional Chinese medicine prescription, attenuates renin-angiotensin-aldosterone system (RAAS) to protect cardiac function and reduce myocardial infarct size (IS) after myocardial infarction.

Methods Male Sprague-Dawley rats (9–10 weeks) were subjected to sham-MI or MI by ligating the left anterior descending coronary artery for 1 week. The rats were divided into five groups: sham, MI, OPJSH (3 g/kg/day), OPJSH (6 g/kg/day), and losartan (an AT1 antagonist, 10 mg/kg/day). The vehicle, OPJSH, or losartan was given by oral gavage once a day after MI. Both IS and cardiac function were determined using TTC staining and Echocardiography at 1 week after MI, respectively. The levels of angiotensin II (ANG II) and aldosterone (ALD) in the non-infarcted area of the left ventricle were assayed using ELISA at 1 week after MI.

Results OPJSH (3 or 6 g/kg/day) administered after MI reduced IS compared with MI group (39±9%, 33±13% vs 55±8%, $p<0.05$) with the greater effect at a dose of 6 g/kg/day. Administration of losartan also reduced IS compared with MI group (39±6% vs 55 ±8%, $p<0.05$). Compared with MI group, administration of OPJSH (3 or 6 g/kg/day) improved cardiac function as evidence by partially preventing the increases in LVEDD (0.87±0.15 vs 0.72 ±0.13 or 0.65±0.13 cm, $p<0.05$) and LVESD (0.72±0.15 vs 0.55 ±0.16 or 0.45±0.16 cm, $p<0.05$), and the decreases in LVEF (39.0 ±8.1% vs 53.6±20.1% or 69.4±15.6%, $p<0.05$) and LVFS (16.3 ±3.8% vs 25.6±12.8% or 36.5±13.9%, $p<0.05$), the greater effect was achieved at a dose of 6 g/kg/day. Losartan treatment also improved cardiac function compared with MI group as shown by the normalisation of LVEDD (0.49±0.08 vs 0.87±0.15 cm, $p<0.05$) and LVESD (0.30±0.06 vs 0.72±0.15 cm, $p<0.05$), and attenuating the decreases in LVEF (75.4±4.9% vs 39.0±8.06%, $p<0.05$) and LVFS (38.6±4.2% vs 16.3±3.8%, $p<0.05$). Additionally, administration of OPJSH (3 or 6 g/kg/day) attenuated the increases in myocardial levels of ANG II and ALD compared with MI group (ANG II: 1.77±0.02 or 1.77±0.04 vs 2.01±0.06 ng/mg protein, $p<0.05$; ALD: 1353±52 or 1356±34 vs 1571±52 pg/mg protein, $p<0.05$). Losartan treatment also inhibited the increases in myocardial level of ALD compared with MI group (1264±51 vs 1571±52 pg/mg protein, $p<0.05$).

Conclusions Our data demonstrated that in agreement with losartan-induced cardioprotection, OPJSH given after MI reduced myocardial IS and improved cardiac function that was associated the decreases in myocardial levels of ANG II and ALD. The results indicate that OPJSH protects against MI possibly via attenuating the RAAS. The results suggest that OPJSH may have a beneficial potential for the prevention and treatment of MI.