S-C-15

Expression of Macrophage Migration Inhibitory Factor in Acute Myocardial Infarction

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Background: Myocardial remodeling after acute myocardial infarction (AMI) contributes to the development of cardiac failure and late mortality. Its pathogenic mechanism is not entirely understood. Macrophage Migration Inhibitory Factor (MIF) is a chemokine that involves in the regulation of inflammatory and immune responses. Therefore, we hypothesize that MIF is activated in the course of AMI which might have pathological importance in the myocardial remodeling, which has not been shown before.

Methods: An animal model of AMI was established by ligating the left anterior descending coronary arteries of the 10-week old male Sprague-Dawley rats. Thirty post-AMI were killed at 3 hr, 6 hr, 1 day, 3 days and 7days. Six sham-operated rats were used as controls. The expression of MIF was revealed and estimated by immunohistochemistry and western blot. The macrophage infiltration was also studied by immunohistochemical method using anti-ED1 antibody.

Results: Immunostaining of MIF in AMI heart showed that it was expressed mainly at the peri-infarct zone in the left ventricle, mainly from the myocytes. A lower of expression was found at the septum and right ventricle. The amount of staining was more abundant from 1 day to 7 days after AMI. Macrophage infiltration at the infarct area was found after 3 days and increased after 7 days of AMI, which confirmed the chemotactic effect of MIF on these cells.

Conclusion: We have demonstrated for the first time that after AMI, MIF expression is increased at the peri-infarcted zone. Macrophages infiltration are also involved in AMI, but after the onset of MIF expression. Therefore MIF may act as a modulator that regulates the macrophage functions, and thus influencing the process of myocardial remodeling.

S-CH-1

RANDOMISED PLACEBO-CONTROLLED STUDY OF THE EFFECT OF FOSINOPRIL ON LEFT VENTRICULAR MASS IN UNTREATED HYPERTENSIVE PATIENTS: FINAL RESULTS

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Objective: Left ventricular hypertrophy is a powerful predictor of cardiovascular morbidity and mortality. We tested the hypothesis that fosinopril, an angiotensin-converting enzyme inhibitor, reduces left ventricular mass in hypertensive patients.

Design and Methods: 54 patients (M:F, 34:20; age 48± 14 yrs) with untreated mild essential hypertension were randomised to treatment with oral fosinopril (10mg-20mg daily) or placebo for 12 weeks. No additional drugs were allowed. The primary outcome measure was the change in left ventricular mass index determined by echocardiography.

Results: Diastolic blood pressure changed from 97.2± 1.8 mmHg at baseline to 98.1±2.3 mmHg at the final visit in control patients and changed from 99.2±2.0 mmHg to 96.0±2.5 mmHg in patients treated with fosinopril ($p = 0.05$). Systolic blood pressure changed from 147.9±2.6 mmHg at baseline to 151.1±3.4 mmHg at the final visit in control patients and changed from 159.2±4.2 mmHg to 149.1±4.5 mmHg in patients treated with fosinopril ($p = 0.01$). The left ventricular mass index changed from 110.3±6.1 g/m^2 to 114.3±8.7 g/m^2 in control patients and changed from 133.4± 9.2 g/m^2 to 127.1± 11.0 g/m^2 in patients treated with fosinopril ($p = 0.03$). There was no significant change in the left ventricular systolic or diastolic function, nor were there any significant changes in plasma electrolytes and renal function.

Conclusion: Treatment with fosinopril as monotherapy for 12 weeks reduces left ventricular mass in hypertensive patients.