

$p < 0.05$), protein (immunohistochemistry) (2.30 ± 0.52 vs 3.45 ± 0.50), $p < 0.05$) and protein (FCM) ($36.66 \pm 5.50\%$ vs $61.56 \pm 11.23\%$), $p < 0.05$) in NIZ decreased significantly in MI group. Compared with MI group, the LVMI (2.27 ± 0.08 vs 2.62 ± 0.16), $p < 0.05$) decreased significantly in Ato group, but was higher than those in Sham group ($p < 0.05$); the expressions of FoxO3a mRNA (0.47 ± 0.05 vs 0.29 ± 0.05), $p < 0.05$), protein (immunohistochemistry) (2.91 ± 0.49 vs 2.30 ± 0.52), $p < 0.05$) and protein (FCM) ($50.64 \pm 9.69\%$ vs $36.66 \pm 5.50\%$), $p < 0.05$) were significantly increased in Ato group, but were lower than those in Sham group ($p < 0.05$).

Conclusion Atorvastatin has a protective effect on ameliorating ventricular remodelling in rats induced by MI. The mechanisms of statins antiventricular remodelling could be associated with its effects of up-regulating FoxO3a.

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EFFECTS OF ATORVASTATIN ON FOXO3A EXPRESSION IN POST-MYOCARDIAL INFARCTION RATS

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Objective To assess the effects of atorvastatin on ventricular remodelling in rats after myocardial infarction and to investigate the alternation of the expression of FoxO3a in nuclear of myocardial cells.

Method Twenty-four hours after myocardial infarction by left anterior descending coronary artery ligation, the survival rats were randomly divided into myocardial infarction group (MI, $n=8$), atorvastatin 10 mg/(kg d) treatment group (Ato, $n=8$). Sham-operated animals underwent identical surgery except for the coronary artery ligation (Sham, $n=10$). After four weeks, the effects of atorvastatin on myocardial fibrosis were evaluated by detecting changes of left ventricular mass index (LVMI), and the expressions of FoxO3a in non-infarction zone (NIZ) by immunohistochemistry staining, RT-PCR. At the same time, the level of non-phosphorylation FoxO3a was measured by flow cytometry (FCM). The data were analysed by SAS 9.1 software.

Result Comparing with Sham group, the LVMI (2.62 ± 0.16 vs 1.80 ± 0.13), $p < 0.05$) was increased significantly in MI group; the expressions of FoxO3a mRNA (0.29 ± 0.05 vs 0.57 ± 0.06),