Myostatin expression in ventricular myocardium in a rat model of volume-overload heart failure

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摘要

Abstract

BACKGROUND: Mechanical stress increases myocardial myostatin expression. However, the expression of myostatin in chronic heart failure resulting from volume-overload and after treatment with beta-blockers is little known. The authors hypothesize that myostatin plays a role in the failing myocardium because of volume-overload. MATERIALS AND METHODS: Aorto-caval shunt was created over a 4-week period in adult Sprague-Dawley rats to induce volume-overload heart failure. RESULTS: Heart weight and body weight ratio significantly increased after shunting. The left ventricular end-diastolic dimension also significantly increased. Treatment with carvedilol in the shunt group reversed the increase in heart weight and ventricular dimension to the baseline values. Myocardial and skeletal myostatin proteins were up-regulated in the shunt group. The mRNA of myocardial myostatin also increased in the shunt group. Treatment with carvedilol reversed both protein and mRNA of myocardial myostatin to the baseline values. Treatment with N-acetylcysteine and doxazosin partially decreased myostatin mRNA and protein expression as compared with the shunt group. Carvedilol normalized the increased immunohistochemical labelling of myocardial myostatin in the shunt group. CONCLUSION: Myocardial myostatin mRNA and protein expression were up-regulated in the rat model of volume-overload heart failure. Treatment with carvedilol is associated with a limitation of increased myostatin expression in the failing ventricular myocardium.