Extracellular matrix remodeling attenuated after experimental postinfarct left ventricular aneurysm repair

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摘要

Abstract

BACKGROUND: Left ventricular aneurysm repair (LVAR) prevents congestive heart failure after myocardial infarction (MI). LV dilation after MI is related to postinfarct myocardial remodeling and leads to CHF. Because changes in matrix metalloproteinases (MMPs), tissue inhibitors of metalloproteinases (TIMPs), and the physical properties of collagens are involved in myocardial remodeling, the effect of postinfarct LVAR on these factors was tested. METHODS: Rats with surgically induced MI, which did or did not receive postinfarct LVAR, were compared with each other and with controls. TIMP messenger RNA and protein expression, MMP gelatin zymography activity, and collagen fibrosis were measured in heart tissue. RESULTS: A threefold difference in the infarction area ratio was observed between samples of LVAs and of repaired LVAs. Compared with rats without LVAR, rats with repaired LVAs exhibited a higher percentage fractional shortening and significantly lower LV end-systolic and end-diastolic diameters. These salutary effects on LV diameter after LVAR were accompanied by a reversal of myocardial remodeling activity. After MI, TIMP expression decreased, MMP activity increased, and collagen fibrosis increased. After LVAR, TIMP expression increased, and MMP activity and collagen fibrosis decreased. These markers of remodeling activity changed significantly and approached preinfarct levels after LVAR. CONCLUSIONS: This study demonstrated that postinfarct LVAR prevents further congestive heart failure by attenuating myocardial remodeling in the LV and is thus indicated both to prevent heart failure and to reduce excessive postinfarct myocardial remodeling.

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