Down Regulation of Apoptosis after Left Ventricular Aneurysm Repair

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摘要

Abstract

Background. Apoptosis is a common feature of the cardiomyopathic process contriuting to progressive decline in ventricular function after transmural myocardial infarction. We hypothesized that left ventricular aneurysm repair (LVAR) down-regulates apoptotic potential of cardiomyocytes in the surviving myocardium. Methods. In the rat infarct model, LV aneurysms were repaired by pursestring suture 2 weeks after coronary artery ligation. Cardiac function and myocardial infarction size were assessed by echocardiography and transverse heart sections, respectively, before sacrifice 12 weeks later. Cardiomyocytes and TdT-mediated dUTP terminal nick-end labeling (TUNEL) assays of apoptotic nuclei were analyzed adjacent to and remote from the aneurysm (8 in infarction group, 7 in aneurysm group, and 11 in repair group). Biochemical samples for immunoblot were also obtained from surviving myocardium. Results. A statistically significant increase in apoptotic rate was seen in both adjacent and remote areas ($p < 0.01$) after aneurysm formation. After LVAR, heart function was improved, and TUNEL assays also show significant decrease when compared with aneurysm group. But significant decreases were noted only in activated caspase-9 and increases in Bcl-2 in immunoblot analysis when comparing repair group with aneurysm group. Conclusions. Down-regulation of apoptosis accounts for the change in the long-term benefit after LVAR. To prevent heart failure, LVAR is indicated when it is large enough, and the infarction area should be excluded as much as possible