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Heart Failure

MECHANICAL UNLOADING WITH LEFT VENTRICULAR ASSIST DEVICE AFTER ACUTE MYOCARDIAL INFARCTION PREVENTS ADVERSE CARDIAC REMODELING

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Background: Mechanical stress plays a critical role during cardiac remodeling post myocardial infarction (MI). Left ventricular assist device (LVAD) can provide circulatory support with profound volume and pressure unloading. The objective of the present study is to demonstrate that mechanical unloading with LVAD after acute MI can prevent cardiac remodeling.

Methods: Adult Dorsett hybrid sheep were unloaded with a LVAD (Impella pump) for 2 weeks after acute MI and analyzed for 10 following weeks (n=8). Eight sheep with MI only and four sham sheep were used as controls. Regional diastolic and systolic strains of the left ventricle (LV) were measured by sonomicrometry array localization. The infarction size was measured at necropsy and digitalized as a ratio of infarcted scar area to the LV free wall. Apoptosis, hypertrophy, and ultrustructural damage were investigated in non-ischemic adjacent zone and remote zone to MI. Proteins involved in the mitochondrial apoptotic pathway (Calcineurin/BAD) and the pro-hypertrophic raf/MEK/ERK pathway were studied by western blotting.

Results: Impella pump provided 43.6±13.2% of cardiac output in the unloaded animals. LVAD unloading alleviated LV dilation and improved EF values compared to the MI group (End diastolic volume: 81.9±15.2 vs. 102.4±27.4 ml; Ejection fraction: 46.4±5.2% vs. 39.3±4.2%, $p<0.05$) at 12 weeks. Infarct size was reduced by mechanical unloading (26.1±1.1% vs. 19.7±1.8%, $p<0.05$). The regional myocardial strain (stretch) was reduced and the regional systolic strain (contractility) was conserved by LVAD unloading. Apoptotic myocytes increased and the Calcineurin/BAD apoptotic pathway was activated in the adjacent zone in the MI group but not in the LVAD group. Regional hypertrophy, evaluated by myocyte area on HE staining and on the single isolated myocytes, was also alleviated by mechanical unloading. Myocyte size increased and the raf/MEK/ERK pathway was activated only in the adjacent zone in the MI group. Ultrastructural damages after MI were also reduced by mechanical unloading.

Conclusion: Mechanical unloading by LVAD after acute MI prevents adverse remodeling, protects cardiac function, and reduces infarct size.