

LOSS OF MYOCARDIAL FUNCTION AND REDOX BALANCE - RELATIONSHIP BETWEEN SURVIVAL PATHWAY ACTIVATION AND VENTRICULAR REMODELING MECHANISMS POST-AMI

ANGELA MARIA VICENTE TAVARES; ARAUJO, ASR; DALL ALBA, R; TAFFAREL, AC; NICOLAIDIS, G; BELLÓ-KLEIN, A; ROHDE, LE; CLAUSELL, N

Background: Events occurring subsequent to acute myocardial infarction (AMI) are partially determinants of the cardiac damage extent later on. The role of redox balance in the post-ischemic cardiac tissue may be critical in this process. **Objectives:** To assess cardiac function and its correlation with redox balance in cardiac tissue 48 hours post-experimental AMI. **Methods:** Male Wistar rats, 8-week-old (n=6/group), weighing 229±24g, were randomized in two groups: Sham-operated (S) and AMI. AMI was produced in rats via ligation of the left coronary artery. Cardiac function parameters were evaluated by echocardiography 48h later. Oxidative profile was studied by measuring antioxidant enzymes expression of superoxide dismutase (SOD), catalase (CAT) and peroxiredoxine 6 (Prx-6). Oxidative damage was quantified by protein oxidation (carbonyls), lipid peroxidation (chemiluminescence - CL), reduced (GSH) and oxidized (GSSG) glutathione ratio and hydrogen peroxide (H₂O₂) concentration (nmol/mg protein) by spectrophotometer. **Results:** Ejection fraction (EF) was lower in the infarct group: AMI (51±5%) vs. S (77±6%) (p=0.0001). H₂O₂ was diminished 48 hours post-AMI: AMI (0.022 ± 0.005) vs. S (0.032 ± 0.008) (p=0.024). Prx-6 was increased in infarcted group: AMI (163.5 ± 4.7) vs. S (144.5 ± 2.4). We found a correlation between reduced/oxidized glutathione ratio (GSH/GSSG) and EF (r=0.79; p=0.009) at 48 hours post-MI. CL, carbonyls, SOD, and CAT were not different between groups. **Conclusion:** These data suggest that the loss of myocardial function and impaired redox balance may be associated with the activation of mechanisms that trigger the process of ventricular remodeling in heart failure. In this study, the low H₂O₂ concentrations noted may act as a 'sensor' that could be regulated by Prx-6 for survival pathway activation within this timeframe following AMI.