Loss of Myocardial Function and Redox Balance - Relationship Between Survival Pathway Activation and Ventricular Remodeling Mechanisms Post-AMI

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Background: Events occurring subsequent to acute myocardial infarction (AMI) are determinants of the cardiac damage extent later on. The role of redox balance (RB) in the post-ischemic cardiac tissue may be critical in this process. Objectives: To assess cardiac function and its correlation with RB 48 hours post-AMI. Methods: Male Wistar rats, 8-week-old (n=6/group), weighing 229±24g, were randomized in two groups: Sham (S) and AMI. AMI was produced via ligation of the left coronary artery. Cardiac function parameters were evaluated 48h later. Oxidative profile was studied by measuring enzymes expression of superoxide dismutase (SOD), catalase (CAT), peroxiredoxine 6 (Prx-6), reduced (GSH) and oxidized (GSSG) glutathione ratio (GSH/GSSG) and hydrogen peroxide (H2O2) concentration (nmol/mg protein).

Results: Ejection fraction (EF) was lower in the infarct group: AMI (51±5%) vs. S (77±6%) (p=0.0001). H2O2 was diminished: AMI (0.022 ± 0.005) vs. S (0.032 ± 0.008) (p=0.024). We found a correlation between GSH/GSSG and EF (r=0.79; p=0.009).

Conclusion: The loss of myocardial function and impaired RB may be associated with the activation of mechanisms that trigger the process of ventricular remodeling. In this study, the low H2O2 noted may act as a 'sensor' that could be regulated by Prx-6 for survival pathway activation within this timeframe following AMI.