Myocardial Pre-Conditioning-The Role of Inflammatory Regulation in Myocardial Protection

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Background: Preinfraction angina has been shown to reduce infract size in experimental models and lead to a more favorable clinical outcome in post-MI patients. The mechanism of protection conferred by preinfarction angina is unknown. We explored the potential role of inflammatory regulation pathways in preinfraction angina-induced myocardial protection in patients who develop acute MI.
Objectives: To investigate the hypothesis that circulating regulatory T (Treg) cells are involved in ischemic preconditioning in AMI patients with angina pectoris occuring during three months prior to the cardiac event.
Methods: Our study consisted of patients enrolled in the TAPAS registry trial who had Acute MI and underwent cardiac catheterization in the 'Tel aviv Sourasky' medical center. ECG characteristics upon administration (ST-segment, Q-leades), CPK peak levels, LV dilatation and segmental analysis, LVEF in day 1 and day 30 (by echocardiogram evaluation) were used as a measure of myocardial damage and infract extent. Treg-cell levels were evaluated by flow cytometry (Treg cells identified as CD4+CD25highCD127low) and mRNA expression of FoxP3.
Results: No difference was observed in Treg-cell levels comparing preconditioned versus non-preconditioned patients in a pilot study (n=26) which included 8 patients in the IPC group and 18 control patients.
Conclusion: Based on our preliminary data, Treg levels do not seem to be involved in ischemic preconditioning in the model described, however, additional patients are required to reach a final conclusion (patients recruitment is ongoing).