Correlation of Heat Production of Culprit Atherosclerotic Lesion With Soluble Cell Adhesion Molecules

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Cell adhesion molecules are critical markers of the inflammatory process, which is involved in the pathogenesis of coronary artery disease (CAD). Previous ex vivo and in vivo studies have shown that thermal heterogeneity within human atherosclerotic plaques. The purpose of the present study was to measure the luminal surface temperature in patients with CAD and to correlate it with the soluble cell adhesion molecules in order to evaluate the role of inflammation in heat production in acute coronary syndromes. Methods: In the study we included 25 patients (pts) (12 with myocardial infarction (MI) and 13 with unstable angina (UA)) and 10 sex- and age-matched controls without CAD. In all pts plasma levels of soluble inter-cellular adhesion molecule-1 (ICAM-1) and vascular cell adhesion molecule (VCAM-1) were measured. A thermography catheter developed in our institution was used, in order to measure intracoronary temperature. A thermistor probe with a temperature accuracy of 0.05 °C, was attached at the distal end of a long 3F polyurethane shaft. Thus, we measured the median temperature differences at the site of the lesion from the core temperature (TD).

Results: The median temperature differences at the site of the lesion from the core temperature (TD) were increased in patients with MI (0.59 ± 0.19 °C) and UA (0.37 ± 0.16 °C) (p<0.01). Levels of VCAM-1 and ICAM-1 concentrations were increased in pts with CAD compared with the control group (VCAM-1: 571.2 ± 169.7 ng/ml vs. 406.1 ± 131.2 ng/ml, p = 0.01). Also, a correlation with ICAM-1 was also observed without however reaching statistical significance. Conclusion: An aggressive inflammatory response occurring in acute coronary syndromes results in increased local heat production. This suggests that, reaching temperature measurement of culprit lesions may be used in future studies to evaluate the effect of anti-inflammatory regimens on the atherosclerotic plaque stabilization.

Endogenous Endothelin-1 Reduces the Postischemic Functional Recovery of Prolonged Hypoperfused Myocardium via the Endothelin-A Receptor

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Background: The release of endothelin-1 (ET-1) from the damaged endothelium may play a role in the inflammation and maintenance of myocardial ischemia. This study examines the ETA-receptor mediated role of endogenous endothelin on postischemic myocardial function after prolonged hypoperfusion.

Methods: In an isolated rat heart model for short-term hibernation the left ventricular functional recovery after 3h hypoperfusion (15% of preischemic coronary flow) followed by 2h reperfusion was determined (isovolumetric steady state hemodynamics: coronary flow, left ventricular pressure (LVP), dP/dtmax; maximal isotropic response to calcium stimulation: max LVP). The effect of ET conversion inhibition using L-NAME, contraction to 30 mM PGF2α was increased in LCX (4.63±0.28 g vs. 2.41±0.26 g, P<0.001). Endothelin-dependent relaxation to 100 ng PDE4 was lower in LAD than in LCX (5.8±0.5% vs. 13.6±3.0%, P<0.01). Reduction of the endothelin-dependent relaxation was significantly greater in the LAD than in the LAD (77.0±0.04% vs. 59.0±0.03%, P<0.01). Endothelin-dependent relaxation to 100 nM sodium nitroprusside (SNP) was similar in LCX and LAD, however, both arteries were significantly more sensitive to the same dose of SNP after NO blockade with L-NAME (26.8±4.5% vs. 78.7±2.8% in LAD, p<0.001 & 25.4±3.4% to 71.2±3.9% in LAD, p<0.001). Conclusions: The pig coronary artery showed adaptive responses 8 weeks after amiodarone constrictor placement, which tends to abrogate myocardial ischemia via decreasing vascular tone. This adaptation may in part involve changes in nitric oxide pathways since the decreased contraction and increased relaxation responses of the affected coronary arteries were partially inhibited by L-NAME.

Myocardial Infarction and Ischemia