Parameters of BMI and glucose in patients with ischemic heart disease and metabolic syndrome.

Key Words: Arterial hypertension, Body Mass Index, Glucose, Ischemic Heart Disease, Lipid Metabolism, Metabolic Syndrome

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**THE ROLE OF CD40-LIGAND AND ADHESION MOLECULES IN ACUTE CORONARY SYNDROMES**

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**Background:** Soluble CD40-ligand plays a key role in immune response, while vascular cell adhesion molecule (VCAM-1) and intercellular adhesion molecule-1 (sICAM-1) mediate the leukocytes adhesion to endothelium. We investigated whether the expression of CD40 and the soluble forms of VCAM-1 and ICAM-1 are affected during the acute phase of unstable angina (UA) and myocardial infarction (MI).

**Methods:** This study enrolled 26 patients with stable angina (SA), 23 patients with UA, 19 patients with MI and 13 healthy controls. Blood sampling was performed by admission to the hospital for UA and MI, and after at least 3 months of stable condition in SA. Serum levels of sVCAM-1, sICAM-1 and CD40-L were determined with ELISA.

**Results:** Serum levels of CD40-L were significantly lower in healthy controls (4.8 ± 1.0 ng/ml) compared to SA (7.9 ± 0.95 ng/ml, p < 0.05), UA (7.83 ± 0.8 ng/ml, p < 0.05) and MI (7.21 ± 1.65 ng/ml, p < 0.05), while no difference was observed between patients with SA, UA or MI. Similarly, serum levels of sVCAM-1 were also significantly lower in healthy controls (360 ± 87 ng/ml) compared to SA (906 ± 118 ng/ml, p < 0.05), UA (735 ± 97 ng/ml, p < 0.05) and MI (752 ± 89 ng/ml, p < 0.05), while no difference was observed between patients with SA, UA or MI. However, no significant difference was observed in sICAM-1 levels between patients with stable angina (SA), UA or MI. Finally, no significant difference was observed in sICAM-1 levels between controls and patients with SA, UA or MI (306 ± 31 ng/ml, 338 ± 13 ng/ml, 331 ± 19 ng/ml and 362 ± 22.7 ng/ml respectively, p = ns for all versus controls).

**Conclusions:** CD-40 and sVCAM-1 are elevated in both stable and unstable coronary syndromes. Thus, these molecules may not be involved in the pathogenesis of plaque instability or rupture observed during unstable coronary syndromes.

Key Words: Coronary Artery Disease, Infections

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**CARVEDILOL REGULATED ACTIVITY OF NITRIC OXIDE SYNTHASE ISOFORMS IN ATHEROSCLEROTIC RABBITS INDUCED BY CHOLESTEROL AND BALLOON INJURY**

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We have reported that carvedilol, a β-blocker with antioxidant activity, improved endothelial dysfunction in atherosclerotic rabbits induced by cholesterol and balloon injury (Jun Pu, et al. Atherosclerosis Suppl. 2003; 4:78). The present study examined the effect of carvedilol on activity of endothelial NO synthase (eNOS) and inducible NO synthase (iNOS) in this animal model. Six rabbits received atherogenic diet alone, and six rabbits received atherogenic diet supplemented with carvedilol (10 mg/kg/d). After treated for 1 week, they underwent balloon injury and remained on their respective diets for the further 10 weeks. Six sham untreated rabbits received standard diets as the normal control. At the end of the experiment, total cholesterol in serum, nitrate and nitrite (NOx) levels in plasma, and eNOS and iNOS activity in aortic segments were determined. Superoxide levels in aortic segments were measured by dihydroethidium staining using laser-scanning fluorescent confocal microscopy. Serum levels of total cholesterol, plasma levels of NOx, iNOS activity and dihydroethidium staining for superoxide in aortic segments were markedly increased whereas aortic eNOS activity was reduced in atherosclerotic control compared with normal control rabbits (P < 0.01). Chronically administrated carvedilol had no significant effect on serum lipid levels (P = NS). However, carvedilol significantly reduced (P < 0.05) plasma NOx levels, significantly (P < 0.05) increased eNOS activity and significantly (P < 0.05) decreased iNOS activity and dihydroethidium staining for superoxide in aortic segments in atherosclerotic rabbits. These findings suggest that carvedilol enhances the activity of eNOS and decreases activity of iNOS without significantly altering serum lipids, possibly via an antioxidative protection. These beneficial effects might contribute to its vasculoprotective and antiatherosclerotic effects.

Key Words: Atherosclerosis, Beta-Blocker, NO Synthase

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**P-463**

**THE IMPACT OF HEPATITIS A VIRUS (HAV), CYTOMEGALOVIRUS (CMV), HERPES SIMPLEX VIRUS (HSV), HELICOBACTER PYLORI (HP) AND C-REACTIVE PROTEIN (CRP), ON ENDOTHELIAL FUNCTION AND CLINICAL STABILITY IN PATIENTS WITH CORONARY ARTERY DISEASE**

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**Background:** Inflammation plays a major role in the development of acute coronary syndromes. However, the role of viral infections remains unclear. We examined the role of hepatitis A virus (HAV), cytomegalovirus (CMV), herpes simplex virus (HSV), helicobacter pylori (HP), C-reactive protein (CRP), on endothelial function and clinical stability in patients with coronary artery disease.

**Methods:** The study population consisted of 97 patients: 27 patients with stable angina, 40 patients with unstable angina and 25 controls with angiographically normal coronaries. Forearm blood flow was measured using venous occlusion strain-gauge plethysmography. Endothelium dependent (EDD) and endothelium independent (EID) dilation were expressed as the % change of flow from baseline to the maximum flow during reactive hyperemia or after sublingual nitroglycerin administration respectively. IgG and IgM antibodies against HAV, CMV, HSV and HP as well as C-reactive protein levels (CRP) were determined by admission.

**Results:** EDD was higher in healthy subjects (78.0 ± 9.8%) compared to patients with unstable angina (57.3 ± 4.5%, p < 0.05) or stable angina (69.3 ± 5.4%, p < 0.05), while patients with stable angina appeared to have higher EDD than patients with unstable angina (p < 0.05). CRP levels were significantly lower in patients with stable angina (6.3 ± 1.05 mg/dl) compared to patients with unstable angina (19.9 ± 3.1 mg/dl, p < 0.05). Healthy controls had significantly lower CRP compared to all the other groups (1.05 ± 0.3 mg/dl, p < 0.01 vs all). The frequency of IgG-HAV was significantly lower in controls (9/12 or 75%) compared to patients with stable (26/27 or 96.6%, p < 0.05) or unstable angina (40/40 or 100%, p < 0.05). EID, IgG and IgM for CMV, HSV and HP did not differ between groups.

**Conclusions:** Patients with stable angina had higher EDD and lower levels of CRP and anti-HAV IgG, compared to unstable syndromes. This finding suggests that enhanced inflammatory process with the involvement of viral infection of hepatitis A may participate in endothelial dysfunction and clinical instability in patients with coronary artery disease.

Key Words: Acute Coronary Events, Inflammation