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BOOK OF ABSTRACTS

Content

Oral Presentations

Poster Presentations

Last Minute Poster Presentations

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Significance of the body weight in endothelial dysfunction changes and systemic inflammation progression in coronary artery disease patients

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Endothelial dysfunction is regarded as a key event in the development and progression of atherosclerosis. It is suggested that dysfunction of the endothelium in large and medium-sized arteries plays a central role in atherogenesis. A recent study has documented the presence of an endothelial dysfunction in small resistance vessels of obese subjects, but it has not yet been ascertained whether an endothelial dysfunction of a large conduit artery is detectable in uncomplicated obesity and, in particular, whether abdominal fat plays a role in determining the degree of this early sign of vascular involvement.

Our aims was to investigate the influence of body weight values on the main markers of endothelial dysfunction (ED) and systemic inflammation (C-reactive protein (CRP), interleukin-6 (IL-6)) in patients with coronary artery disease (CAD).

Material and methods: The study involved 58 patients with coronary artery disease in the form of stable exertional angina II functional class, mean age – 54.5 ± 3.2 years, 25 of them women. Depending on body mass index (BMI) identified 2 groups: Group 1 – 26 patients with a BMI of 28.4 ± 1.3 kg/m 2 group – 32 patients with a BMI of 33.6 ± 2.5 kg / m².

The following anthropometric variables were evaluated in all subjects: weight, height and BMI. Serum total cholesterol, triglycerides and HDL cholesterol were measured. LDL cholesterol was calculated by using Friedwald formula. Using high resolution vascular ultrasound, we measured brachial artery responses to reactive hyperemia (with increased flow causing endothelium-dependent dilatation) and sublingual nitroglycerin (causing endothelium-independent dilatation). Were analyzed indicators of endothelium-dependent vasodilatation ((EDVD), an increase of ≥ 10% of the original diameter during reactive hyperemia), endothelium-independent vasodilatation ((ENVD), ≥20% increase in the sample with nitroglycerin) Also we analyzed results of quantitative determination of C-reactive protein (CRP), interleukin-6 (IL-6).

Results: At rest, the diameter of the brachial artery was 0.48 ± 0.02 cm patients of group 1 and 0.59 ± 0.02 cm patients in group 2. When EDVD reactive hyperemia increased by 15% in patients of group 1 and remained virtually unchanged in the 2nd group (respectively: 0.55 ± 0.02 cm; $p < 0.05$; 0.59 ± 0,03 cm; $p > 0.05$). The diameter of the brachial artery after 5 min after administration of nitroglycerin in patients in Group 1 increased by 19.4% and in the 2nd only 9.7% (respectively: 0.56 ± 0,02 cm ($p < 0.05$) ; 0.01 cm ± 0.63 ($p > 0.05$). Indicators of IL-6 had no statistical difference in the two groups (respectively: 6.85 ± 0.27 pg / ml; 6.91 ± 0.33 pg / ml control 6.54 ± 0.18 pg / ml). The level of CRP slightly increased in patients of group 1 was significantly increased in patients in group 2 (respectively: 2.68 ± 0.16 mg / ml ($p > 0.05$); 4.82 ± 0.14mg / ml ($p < 0.05$); control 2.12 ± 0.06 mg / ml).

Conclusions: In patients with stable exertional angina II functional class with increasing BMI occurs inhibition of endothelial function and activation of systemic inflammation, manifested by decrease EDVD and ENVD and rising CRP, which adversely affects on the course of coronary artery disease and induces the development of vascular complications.

This risk appears to stem from multiple abnormalities in adipose tissue function leading to a chronic inflammatory state and to dysregulation of the endocrine and paracrine actions of adipocyte-derived factors. These, in turn, disrupt vascular homeostasis by causing an imbalance between the NO pathway and the endothelin 1 system, with impaired insulin-stimulated endothelium-dependent vasodilation.

Keywords: endothelial dysfunction, systemic inflammation, coronary artery disease, endothelium-dependent vasodilatation, endothelium-independent vasodilatation