Commentary

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Endothelial cells are the source of several inflammatory cytokines, both pro- and anti-inflammatory, which are playing a role of angioprotective agents preventing atherosclerosis. Moreover, endothelial dysfunction is an onset of intravascular inflammation. In patients with metabolic syndrome, a visceral fat tissue is an additional factor increasing on-going inflammation. Adipose tissue produces several bioactive mediators - adipocytokines, which in turn influence insulin resistance, diabetes, body-weight homeostasis and inflammation. They control inflammatory and oxidation stress, lipoprotein-associated phospholipase A2 activity and in consequence increase dynamics of atherosclerosis via plaque rupture as well as heart and vessel complications. Adipocytokines have also been shown as a risk factor for reinfarction. In consequence, it is the second pathway of on-going inflammation. Changes in life style, weight reduction and tobacco use cessation are the possible ways of metabolic syndrome improvement resulting in adipocytokine inhibition, including HMG-CoA inhibitors known as proinflammatory cytokines inhibitors.

Seino Y, Hiose Y (2009) showed that adiponectin and leptin could be a long-way predictor of metabolic syndrome. Jacobs M (2009) makes a similar remark indicating direct connection between ischaemic heart disease and metabolic syndrome via subclinical inflammation.

The reviewed paper took up an important problem of concentration of adipocytokines contained in fat tissue of patients qualified for CABG surgery, i.e. patients with highly advanced atherosclerotic changes in blood vessels.

Authors have demonstrated that concentrations of leptin, adiponectin, resistin and apelin in a group of surgically treated patients with CAD and in healthy subjects are different. In a group of CAD patients, serum leptin concentration was significantly higher and serum adiponectin and apelin concentrations were significantly lower in comparison with healthy subjects (see also Bastard JP, Maachi M, Lagathu C. EurCyt Network 2006; 17: 4-12).

Here a question arises what the primary reason of these cytokine disturbances is. Is it an atherosclerosis degree presented by carotid artery intima-media thickness in CAD or is it metabolic syndrome presented in all examined patients?

The authors found that apelin could play a possible role in blood pressure regulation. This finding is quite interesting, it is a novelty, but requires further examinations.

The final question applies to the field of interest of cardiac surgeon, who usually concentrates on a fluent surgery more than on cytokine secretion. The information that adipokine secretion impairs insulin signaling, promotes endothelial dysfunction, vascular remodeling and foam cell formation in the arterial wall could help predict potential cardiovascular complications. However, the authors do not provide a clear-cut connection between cytokine concentration and cardiovascular complications that can have a bearing on a surgical procedure or post-surgical patient condition. It is a serious defect of this work.