INFLAMMATORY AND GLUCOSE METABOLISM PARAMETERS IN OBESE PATIENTS

Kikhara Atuman, Kanika Denis, 6th-year students
Scientific supervisor– senior lecturer N. V. Demichova
Sumy State University, department of family medicine with the course of endocrinology

Hyperinsulinaemia promotes smooth muscle proliferation in the vessel wall and stimulate production of plasminogen activator inhibitor. These adverse proliferative and thrombogenic actions, however, must be set against the vasculoprotective effects of insulin which include stimulation of endothelial nitro-oxide production, inflammation markers (interleukin (IL)-6, tumor necrosis factor-α - TNF-α), such that net effects on atherogenesis are hard to quantify. Epidemiological studies have been contradictory, and take together suggest that hyperinsulinaemia is only weakly predictive of accelerated atherogenesis without necessarily implying a causal relationship. Insulin resistance correlates better with coronary artery disease, and in the Insulin Resistance Atherosclerosis Study had an independent effect on carotid intimal medial wall thickness that persisted following adjustment for smoking, lipid levels, hypertension, diabetes and gender. In this study we investigated inflammatory and glucose metabolism parameters in morbidly obese (BMI>40kg/m2) subjects.

Materials and methods: We studied 17 (5 men, 12 women, mean age 37,1+/-8,4, BMI 44,14+/-3,9 kg/m2) normotensive obese subjects without prior diabetes diagnosis. 13 age-matched healthy non-overweight subjects served as controls. All subjects underwent oral 75 g glucose tolerance test (OGTT) with plasma insulin measurements. Fasting plasma subclinical inflammation markers (IL-6; TNF- α). Intima-media thickness (IMT) of caroid artery was measured as well.

Results: During OGTT, as compared to the controls, obese subjects had higher glucose level at 60 min (141±38 vs 103±36 mg/dl; $p<0.03$) and 120 min (121±33 vs 90±28 mg/dl; $p<0.03$) as well as fasting insulin level (28±25 vs 10±8 μIU/mL; $p<0.04$) and insulin level at 60 min (115±63 vs 33±21 μIU/mL; $p<0.001$). Moreover, in the obese subjects HOMA-IR (6.2±5.4 vs 2.34±1.86, $p<0.03$), TNF-α (24.1±11.0 vs. 13.3±6.9 ng/ml), IL-6 (6.82±4.1 vs 3.85±1.4 pg/ml; $p<0.001$) were greater than in the controls. IMT was higher in the study group (0.69±0.12 vs 0.59±0.1 mm, $p<0.04$). There were no differences between both groups in regard to plasma lipid profile, systolic and diastolic blood pressure.

Conclusion: Non-diabetes morbidly obese subjects already present with mild post-challenge hyperglycemia and hyperinsulinemia as well as subclinical inflammation. The results of our study suggest that vascular risk associated with obesity precedes diabetes development. This finding might have important implications for adopting prevention measures of cardiovascular disease in morbidly obese individuals.