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INSULIN RESISTANCE AND PROLONGED ANTIHYPERTENSIVE THERAPY
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The purpose is to investigate the influence of the prolonged treatment by ACE inhibitor enalapril upon the blood immunoreactive insulin (IRI) levels and daily profile of blood pressure (DBPBP), hemodynamics parameters in non-diabetic hypertensives. Studies were performed on 62 hypertensives with congestive heart failure (CHF) II-III (NYHA), aged 43-75 and 10 healthy people (control). Basal hyperinsulinemia was observed in 23 (37.10%) cases (I group), 2hr postloading hyperinsulinemia - in 27 (43.55%) cases (II group) with IRI levels higher in 2.7 and 3.1 times vs control (p<0.01). The normoinsulinemia was observed in 12 (19.30%) cases (III group). Basal hyperinsulinemia (II group) was associated with the increase of values of unfavorable DBPBP night-peaker (43.50% vs 29.60% in II group) and eccentric hypertrophy of left ventricular with systolic dysfunction (60.90% vs 45.50% in II group). After one year of treatment with enalapril, basal and 2hr postloading hyperinsulinemia statistically significantly decreased in 39.13% cases in I group, only 2hr postloading hyperinsulinemia -in 37.03% cases in II group and without IRI levels changes - III group. The DBPBP normalization, regression of pathological remodeling of left ventricular, the significant improvement of clinical course of arterial hypertension and CHF were greater in patients of III group and in patients with normalized IRI levels. Thus, the prolonged blockade of the renin-angiotensin system with ACE inhibitor enalapril promotes the decrease of insulin resistance in hypertensive patients with CHF.

Key Words: Insulin resistance, Hypertension, Angiotensin-Converting-Enzyme Inhibitor

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URIC ACID LEVEL IS ONE OF THE PREDICTORS OF BP ELEVATION AND OBESITY
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Plasma uric acid level is well known as the predictor of renal injury and hypertension. In the present study, we tried to clarify whether plasma uric acid level is one of the predictors of hypertension, obesity or obesity-related hypertension in a longitudinal study of 5 years in a cohort. In 433 young, nonobese (BMI<26 kg/m2) normotensive Japanese men who were working in a factory, BMI, %fat accumulation (%FA), BP, pulse rate, plasma uric acid (UA), norepinephrine (NE), insulin, leptin (LEP), total cholesterol (Tch), triglyceride (TG), BUN and creatinine (Crn) were measured every 1 yr for 5 yrs. None had diabetes, medications for hyper-uric acidemia or hypertension during the study. BP elevation and weight gain (WG) for 5 yrs were defined as more than 10% increases in mean BP or BMI at entry. BP elevation and WG were noted in 13% and 17% in a cohort. WG induced BP elevation was 53% of subjects with WG. At entry, subjects with WG had higher levels of mean BP, pulse rate, UA and NE than subjects without WG (UA 5.6 mg/dl vs 4.1, P<0.01), although BMI, %FA, insulin, LEP, Tch, TG, BUN and Crn were similar. The absolute increases for 5 yrs in BMI, %FA, mean BP, UA, Tch, TG, plasma NE, insulin and LEP were greater in subjects with WG (UA 1.8 mg/dl vs 0.1, P<0.01). Subjects with BP elevation had higher levels of mean BP, pulse rate, UA, and plasma NE at entry (UA 6.1 mg/dl vs 4.1, P<0.01), although BMI, %FA, insulin, LEP, Tch, TG, BUN and Crn were similar. The absolute increases for 5 yrs in mean BP, BMI, %FA, plasma NE, UA and Crn were greater in subjects with BP elevation (UA 1.2 mg/dl vs 0.3, P<0.01). Subjects with WG induced BP elevation had higher level of UA at entry than subjects without WG induced BP elevation (UA 6.3 mg/dl vs 4.8, P<0.05). The absolute increases for 5 yrs in BMI, %FA, mean BP, pulse rate, UA, TG, NE, insulin and LEP were greater in subjects with WG induced BP elevation than subjects without significant BP elevation (UA 2.2 mg/dl vs 1.5, P<0.05). When analyzed the change in mean BP as a dependent factor in association with changes in BMI, plasma NE, insulin, LEP, UA, Tch and TG as determinant factors by multiple analysis, at 5 yrs, changes in BMI (P=0.007), plasma NE (P=0.019), and UA (P=0.007) were significant determinant factors in the changes in mean BP (R2=0.189, F=8.93, P=0.0001). In conclusion, these results demonstrated that plasma uric acid level is one of the predictors of weight gain, BP elevation and weight gain-induced BP elevation.

Key Words: Uric Acid, Weight Gain, Blood Pressure Elevation

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RENIN-ANGIOTENSIN SYSTEM POLYMORPHISMS INFLUENCE CARDIOVASCULAR RISK PROFILE OF OBESE ESSENTIAL HYPERTENSIVE PATIENTS
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Considering the role of RAS in the pathogenesis of obesity-hypertension, we studied possible contribution of RAS polymorphisms for defining cardiovascular risk profile of these patients.

We studied 70(41.58.6%) obese (Mean BMI:32.5±2.2) essential hypertensives never treated with ACEIs or ARBs with a mean age 58±8 y.o. 52(74.3%) patients were receiving other antihypertensive drugs. All patients were genotype for M235T polymorphism of AGT, the I/D of ACE and the a1166c of angiotensin II ATIR by PCR and restriction enzyme analysis. BP,HR,BMI were measured as well as determined fasten levels of glucose, insulin, uric acid, lipicid profile, creatinine, and 24 h microalbuminuria

Not statistically significant differences in BMI and rest parameters were found. Patients with the genotypes TT (AGT), DD (ACE) and cc(AGT) showed worse cardiovascular profile. These results indicate that RAS polymorphisms contribute to define C-V risk profile of the obese hypertensives.