of diabetes: 10.03±4.91 years and duration of hypertension: 11.32±5.38 years). Patients were assigned to antidiabetic and antihypertensive treatment aiming to attain pre-defined level of glycemic (HbAlc < 7%) and blood pressure control (<130/85 mmHg). Nephropathy progression parameters, average annual increment of microalbuminuria (Δ UAE) and deterioration of glomerular filtration rate (Δ GFR), were prospectively followed-up for a mean period of 6.28 (0.88) years. Statistical analysis was performed using the STATISTICA 4.5 program (StatSoft, Tulsa, OK, USA). The significance of differences was evaluated using the Kruskal-Wallis test. A value of p<0.05 was accepted as statistically significant.

Results: A total of 62 (49.2%) patients achieved good glycemic control while tight blood pressure control was achieved and maintained in 76 (60.3%) patients. Choice of antidiabetic treatment did not significantly influence the quality of glycemic control (P=0.4233), while choice of antihypertensive drugs significantly influenced the quality of achieved blood pressure control (P=0.0357), speaking in favor of ACEI.

Choice of antidiabetic treatment did not significantly influence the rate of microalbuminuria progression as well as the rate of deterioration of glomerular filtration both in patients with good (P=0.641 and P=0.8377, respectively) and those with poor glycemic control (P=0.305 and P=0.256, respectively). However, choice of antihypertensive treatment significantly influenced the rate of microalbuminuria progression (P=0.011) as well as the rate of deterioration of glomerular filtration (P=0.0013), but only in those with tight blood pressure control.

Conclusion: Independently from the quality of achieved glycemic control, choice of the antidiabetic treatment did not significantly influence the rate of nephropathy progression in hypertensive type 2 diabetic patients. However, choice of antihypertensive treatment significantly influenced the rate of progression of microalbuminuria and the rate of deterioration of glomerular filtration but only in those patients in whom good control of blood pressure was achieved.

P 8: Peripheral insulin resistance influence the presence of hypertension in overweight type 2 diabetic patients

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Backgrounds and Aims: Increased obesity is closely related to insulin resistance (IR) promoting the development of type 2 diabetes (T2D) and cardiovascular disease. IR represents a well-established risk factor for development of hypertension, but the significance of the associations between the IR and obesity in the development of hypertension in overweight patients with type 2 diabetes (T2D) still remains unclear. We analyzed the levels of insulin resistance in: overweight T2D patients with hypertension (group A, n=48, 30±35 BMI ≥25kg/m2), overweight T2D patients without hypertension (group B, n=43, 30±35 BMI ≥25kg/m2) and overweight nondiabetics without hypertension (group C, n=40, 30±35 BMI ≥25kg/m2).

Materials and Methods: Patients were aged 40-70 years, matched by gender, duration of diabetes, with optimal metabolic control (HbAlc=6.5±0.6%). Hypertension was defined as systolic BP ≥140 and diastolic BP ≥90mmHg measured by sphygmomanometer, or by established use of antihypertensive drugs. Assessment of IR were done with two complementary indexes of insulin sensitivity: (1) oral glucose insulin sensitivity (OGIS) index derived from 75 g oral glucose tolerance test and (2) homeostasis model assessment of insulin resistance (HOMA-IR) determined from fasting glucose and plasma insulin (PI) levels. PI and levels were measured by RIA method.

Results: We have found significantly higher PI levels in group A (A: 38.05+/−4.24; B: 29.33+/−3.13; C:21.34+/−2.23μIU/ml, A vs B; A vs C and B vs C p<0.05). Simultaneously, there was no significant difference in the level of HOMA-IR among diabetics (A: 9.43+/−2.69; B: 7.52+/−2.08; C: 5.73+/−1.83; A vs B p=NS; A vs C and B vs C p<0.05), reflecting hepatic insulin sensitivity. However, lowest OGIS were found in group A, being significantly lower in group A in comparison to group B (A: 287.67+/−35.42; B: 359.22+/−32.0; C: 494.29+/−38.81; A vs B p<0.05; A vs C p<0.01 and B vs C p<0.05) as measurement of peripheral IR. We have not found significant correlation between PI and presence of hypertension (r=0.177, p=NS). Also, there was no significant correlation between HOMA-IR and presence of hypertension among diabetics (r=0.218, p=NS). However, presence of hypertension among diabetics negatively correlate with OGIS index (r=-0.384, p<0.01), and in the model of linear regression analysis level of OGIS predicting the presence of hypertension irrespectively of the obesity (β= -0.334, p=0.07).

Conclusion: Our results imply that increased peripheral insulin resistance compared to hepatic insulin resistance stronger influence the presence of hypertension in overweight type 2 diabetic patients. Moreover, this association was not depending of obesity.

P 9: Taurine protects against impairment of mitochondrial function and oxidative stress in the heart of rats with fructose-induced insulin resistance

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Background and aims: Taurine (2-aminoethanesulfonic acid) is a free amino acid found in substantial amounts in all mammalian tissues. Recently, data is accumulating that show the effectiveness of T against diabetes mellitus, insulin resistance and its complications, including retinopathy, nephropathy, neuropathy, atherosclerosis and cardiomyopathy, independent of hypoglycemic effect. Numerous experimental and several clinical studies demonstrated that T helps the cardiovascular system through a variety of mechanisms including modulation of intracellular calcium concentration, antagonism of angiotensin II action, membrane-stabilizing, antioxidant, and lipid-lowering effects. The aim of the study was to assess the effects of T on mitochondrial respiratory chain activity and lipid peroxidation in the heart of prediabetic rats fed a high-fructose diet.

Materials and Methods: Male Wistar rats were divided into three groups: the control group fed on a regular diet (C, n=8), the high fructose-fed group (F, n=8), which had free access to 250 g/L solutions of fructose for 8 weeks and the fructose-fed group treated with taurine (F+T, n=8) for 8 weeks (100 mg/kg/day per os). Mitochondria were isolated by differential centrifugation from the hearts of fed rats. Oxygen consumption rate was measured polarographically at 37°C using a Clark-type oxygen electrode with either glutamate/malate or succinate as energy substrates of complex I or II, respectively. Levels of lipid hydroperoxides, reduced glutathione (GSH) and glutathione reductase (GR) activity were determined in mitochondrial preparations. Result: We found reduction of NAD-dependent substrate oxidation rate in metabolic state 3 in the heart mitochondria of rats with fructose-induced insulin resistance by 30% in comparison with intact control (p<0.02). However, FAD-dependent substrate oxidation rate was not changed in all experimental groups. Administration of T normalised the rate of glutamate/ malate oxidation in the state 3 (F+T:79.04±3.97 vs F:66.1±5.00; C:82.60±7.48 natomO/min.mg of protein, p<0.05) and the values of respiration control (F+T:5.38±0.32 vs F:4.63±0.25 ; C:5.8±0.35, p<0.02). Improvement of mitochondrial function in the