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Groups	Before treatment			After treatment		
	Glucose, mmol/l	Insulin, mU/ml	HOMA	Glucose, mmol/l	Insulin, mU/ml	HOMA
fosinopril	5,42 ± 0,18	8,8 ± 0,83	2,35 ± 0,25	5,35 ± 0,18	8,01 ± 0,69	2,08 ± 0,2
telmisartan	5,5 ± 0,14	9,4 ± 0,92	2,37 ± 0,25	5,34 ± 0,14	6,53 ± 2,98*	1,62 ± 0,18*
aliskiren	6.50 ± 0.38	10.36 ± 2.51	3.39 ± 1.04	6.09 ± 0.5	8.09 ± 2.33*	2.39 ± 0.94*

Results: The blood pressure control was absolutely equivalent in all groups. Main results of changes of metabolic parameters are presented in the table. At the beginning of investigation the levels of serum glucose, insulin, HOMA index before and during OGTT in all groups were comparable. At end of the study we observed the tendency to improvement of OGTT data and lipid level in patients treated by fosinopril (HOMA changed from 2,35 ± 0,25 to 2,08 ± 0,2, $p > 0,05$, TG and HDL cholesterol did not change) and significantly improvement in patients treated by telmisartan (HOMA changed from 2,37 ± 0,25 to 1,62 ± 0,18, $p < 0,05$, TG changed from 1,75 ± 0,15 to 1,34 ± 0,13 mmol/l, HDL cholesterol changed from 1,18 ± 0,03 to 1,26 ± 0,03 mmol/l, $p < 0,05$). Significant improvement in patients treated by aliskiren (HOMA changed from 3.39 ± 1.04 to 2.39 ± 0.94, $p < 0,05$, TG and HDL cholesterol did not change). In comparison between aliskiren, telmisartan and fosinopril groups at the end of study aliskiren and telmisartan more decreased HOMA, fasting insulin level than fosinopril.

Conclusion: The 6-month therapy in patients with AH and MS by fosinopril did not change HOMA, glucose, insulin, triglycerides levels. Therapy by telmisartan significantly improved HOMA, fasting insulin, TG, HDL level. Therapy by aliskiren significantly improved HOMA, fasting insulin and did not change TG, HDL level.

PP.9.228 INCREASED INFLAMMATORY MARKERS IN HYPERTENSIVE PATIENTS IN THE CONTEXT OF THE METABOLIC SYNDROME

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Objective: Studying laboratory parameters of hypertensive patients in the context of the metabolic syndrome (MS) could provide significant insights into the etiology of the condition, known to be complex and multifactorial. The purpose of this study was to evaluate the values of fibrinogen and hsCRP in hypertensive patients with metabolic syndrome compared to hypertensive patients without atherogenic dyslipidemia.

Material and Method: Forty-eight hypertensive patients with metabolic syndrome (mean age 57 ± 3.67 years) and forty-four hypertensive patients without atherogenic dyslipidemia (mean age 54 ± 4.80 years) were included in the study. MS was defined by the National Cholesterol Education Program Adult Treatment Panel III guidelines. In all patients, the plasma total cholesterol, triglycerides, HDL-cholesterol, LDL-cholesterol, hsCRP and fibrinogen levels were evaluated. Anthropometric characteristics and blood pressure were also recorded. The statistically analysis was done using Pearson's test and Student's t-test. $p < 0.05$ was considered statistically significant.

Results: Significant differences between groups were found for the following: BMI, waist, abdominal circumference, waist/hip ratio, total cholesterol, triglycerides, LDL-cholesterol (all $p < 0.001$) and HDL-cholesterol ($p = 0.008$). We found significant higher levels of fibrinogen (3.30 ± 0.34 vs 3.15 ± 0.29 g/L, $p = 0.03$) and hsCRP (4.24 ± 1.25 vs 3.45 ± 1.36 mg/L, $p = 0.008$) in hypertensive patients with metabolic syndrome compared to hypertensive patients without atherogenic dyslipidemia.

Conclusions: Our study reveals that inflammatory markers (hsCRP, fibrinogen) that are not routinely measured are elevated in hypertensive patients with metabolic syndrome and emphasize the value of these laboratory parameters in these patients.

PP.9.229 CLINICAL EFFICACY OF THE FIRST RENIN INHIBITOR - ALISKIREN IN PATIENTS WITH HYPERTENSION AND METABOLIC SYNDROME

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Aim: To estimate antihypertensive efficacy, cardiometabolic effects, influence on level of microalbuminuria (MAU) and vascular stiffness of the first renin inhibitor aliskiren in patients with hypertension, 1-2 stages and metabolic syndrome.

Materials and Methods: The study included 33 patients with hypertension, stages 1-2: average age was 41,2 ± 0,9 years, 16 men and 17 women, weight was 95,18 ± 4,84 kg, body mass index was 33,13 ± 1,31 kg/m², waist circumference was 106,38 ± 3,51 cm. All patients had arterial hypertension I-II degrees, duration of arterial hypertension was on average 4,8 ± 3,2 years. Previous therapy was changed to aliskiren in a starting dose 150 mg. If doses of the previous preparations had been maximum, the starting dose of aliskiren was 300 mg a day. The study lasted 24 weeks. All patients underwent clinical examination, including the anthropometrical measurement, 24-hour blood pressure monitoring (BPM), fasting glucose, glucose during oral glucose tolerance test, total cholesterol (TC), low density lipoproteins (LDL), high density lipoproteins (HDL), triglycerides (TG), level of MAU and rates of vascular stiffness (right/left pulse wave velocity - R/L-PWV, cardio ankle vascular index - CAVI1/L-CAVI1, right- augmentation index - R-AI).

Results: At the moment of the research termination there were 70% of patients on the monotherapy of aliskiren, 60% of which received aliskiren in a dose of 150 mg, 300 mg of aliskiren received 40% of patients. On the combined therapy there were 30% of patients, the majority of them received a combination of aliskiren and hydrochlorothiazide. Target levels of BP both systolic, and diastolic were achieved in 80% of patients. As a result the systolic BP significantly decreased from 137,38 ± 2,3 to 126,57 ± 1,9 mm Hg ($p < 0,01$), diastolic BP decreased from 84,90 ± 1,99 to 78,14 ± 1,25 mmHg ($p < 0,05$). Postprandial glucose level decreased from 7,22 ± 0,36 to 6,20 ± 0,22 mmol/L ($p < 0,05$). Levels of lipids significantly have not changed. The level of MAU decreased from 70,2 ± 21,7 to 41,3 ± 13,6 mg/l ($p < 0,05$). According to the data of volume sphygmography the rigidity of arteries decreased. R/L-PWV, which was initially above normal values has significantly decreased from 14,21 ± 0,45 m/s to 12,98 ± 0,23 m/s ($p < 0,05$). R-AI decreased from 1,13 ± 0,04 to 1,01 ± 0,01 ($p < 0,05$). That refers to the improvement of structural and functional abilities of a vascular wall.

Conclusions: The results of aliskiren therapy in patients with hypertension and metabolic syndrome showed antihypertensive effect with significantly decreased of postprandial glucose level, decreased the level of MAU and improvement of structurally and functional abilities of a vascular wall.

PP.9.230 LEVELS OF LEPTIN, ADIPONECTIN, INFLAMMATORY FACTORS AND ANTHROPOMETRIC PARAMETERS IN PATIENTS WITH ABDOMINAL OBESITY AND ARTERIAL HYPERTENSION

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Objective: to compare metabolic status and anthropometric parameters in normotensive and hypertensive patients with abdominal obesity (AO).

Methods: 120 patients with AO (IDF, 2005) age 43, 2 ± 0,8 yrs, body mass index (BMI) 32,1 ± 1,9 kg/m² were examined. 50 patients were hypertensive (BP ≥ 140/and, or ≥ 90 mm Hg). Body mass (BM), waist circumference (WC), BMI, body fat (BF,%) and lean body mass (LBM, kg) were measured. Lipid profile (total cholesterol, HDL-cholesterol, LDL-cholesterol, TG) was obtained. Fasting glucose and insulin, HOMA-IR were defined. The levels of leptin, adiponectin, TNF-Δ and CRP were measured.

Results: BMI, WC, BM, were significantly higher in hypertensive patients with AO (105,0 ± 1,4 cm and 100,2 ± 1,3 cm, $p = 0,01$, 96,4 ± 2,1 kg and 90,3 ± 1,7 kg, $p = 0,03$, 33,9 ± 0,6 kg/m² and 31,1 ± 0,5 kg/m², $p = 0,0001$, respectively). There were no differences in lipid profile, levels of fasting glucose, insulin and HOMA IR between normotensive and hypertensive patients. The levels of leptin, CRP and TNF-Δ were higher (66,1 ± 4,7 ng/ml and 60,1 ± 5,1 ng/ml, $p = 0,002$, 9,1 ± 1,7 mg/l and 6,1 ± 0,9 mg/l, $p = 0,04$, 52,6 ± 9,3 pg/ml and 33,0 ± 3,5 pg/ml, $p = 0,04$, respectively) as well as adiponeckin was lower (17,2 ± 2,0 μg/ml