P1.33 NEPHROPROTECTIVE AND ORGANOPROTECTIVE EFFICACY OF MOEXIPRIL IN PATIENTS WITH MILD TO MODERATE HYPERTENSION

M. Demidova¹, M. Leonova¹, Y. Belousov¹, A. Tarasov¹. ¹Russian State Medical University, Korolev, Moscow, Russian Federation

Objective: The aim of this study was to evaluate the nephroprotective and vasoprotective efficacy of Moexipril in patients with mild-to-moderate arterial hypertension.

Design and Methods: This was 18-week, prospective, open-labeled study in one center in 34 patients with mild to moderate hypertension. Initially all patients received 7.5 mg of Moexipril once daily, if it wasn't effective the dose was doubled (to 15 mg daily). Nephroprotection was studied by evaluation of microproteinuria: morning microalbuminuria (N < 15 mg/l) and β_2 -albuminuria (N < 0.37 mg/l) in urine. Vasoprotection was studied by evaluation of endothelial function: endothelium-dependent vasodilatation (EDV) in method with reactive hyperemia (>10%), endothelium-independent vasodilatation (EIDV) in method with nitroglycerine (>15%); calculated index of vasodilatation (IV): EIDV/ EDV (N = 1.5–1.9).

Results: EDV in patients after treatment was increased up to 12.3% (versus 10.1% before the treatment) (p > 0.001). EIDV after the treatment was increased up to 17.5% (versus 11.0% before the treatment) (p > 0.001). IV after the treatment was 1.42. The results of evaluation microproteinuria are in table 1.

Table 1.

Index	Before treatment	After treatment	Significance
Microalbuminuria (mg/l)	28.3±4.1	11.5±5.2	p > 0.001
β ₂ -albuminuria (mg/l)	1.2±0.1	0.24±0.2	p > 0.001

Conclusions: Moexipril has beneficial nephroprotective and vasoprotective effects in patients with arterial hypertension.

P1.34 WHICH PERIDIALYSIS BLOOD PRESSURE BEST PREDICTS INTERDIALYTIC LEVEL?

Y. Bataille¹, A. Saint-remy¹, E. Godon², B. Dubois¹, J-M. Krzesinski¹. ¹Centre Hospitalier Universitaire, Liege, Belgium, ²Centre Hospitalier Hutois, Huy, Belgium

Objective: ESRD patients have a high CV morbidity and mortality. Hypertension (HT) is frequent and has multifactorial origins. Hemodialysis (HD) treatment is a particular model of subacute blood volume variation inducing modifications of blood pressure (BP) and cardiac output. But what is the best BP parameter to consider to appreciate the BP load on the CV system?. The aim of the present study was to define which peridialysis BP can best predict the interdialytic BP profile.

Design and Method: Data were collected for 31 HD patients (M=16; F=15) dialyzed 3 times/week during 4 hours each session, mean age was 66 ± 14 years. Patients were submitted to a 48h monitoring of BP with a Spacelabs 90207 device placed 10 minutes before the first HD session and removed 10 minutes before the next session. Pre-dialysis and post-dialysis BP, the mean of pre and post-dialysis BP and interdialytic BP were recorded as well as weight gain between 2 consecutive dialysis. Interdialytic BP was expressed by the mean of all BP recorded between 4 h after the end of the first HD and 40h later before the following HD session.

Results: None of the parameters differed between gender. Compared to normotensive (NT) patients (n = 14), hypertensive (HT) patients (n = 17) had significantly higher: post-dialysis SBP(146 \pm 21 vs119 \pm 20) and DBP(84 \pm 12 vs 69±14), interdialytic SBP(140±14 vs125±15), 48h SBP load (54% vs 28%) and 24h, daytime and nighttime SBP. Rates of non dippers (nocturnal BP fall<10%) were equal during the two consecutive nights: 90% for SBP and 65% for DBP and did not differ between NT and HT. Reversed rhythm concerned 39% of patients for their SBP and 25% for their DBP. Analysis of reproducibility between the 2 days of ABPM indicated that, only among NT patients, the nighttime SBP was higher during the second night than the first one, but among HT patients, none of the parameters differed. In NT, interdialytic SBP was significantly correlated to pre- and post-dialysis SBP, but slightly better with their average (0.71;p < 0.001), In HT, only the average of pre- and post-dialysis SBP was correlated to interdialytic SBP(0.54;p=0.03). Correlations were quite higher between interdialytic DBP and pre and post-dialysis DBP and their average, both in NT and HT patients. Weight gain was not related to any peridialysis BP or to interdialytic BP or pulse pressure but only moderately to overall BP load (p = 0.03).

Conclusions: Peridialysis BP, especially the average of pre- and postdialysis BP, can contribute to the clinical management of interdialytic BP. This is especially true for DBP rather than for SBP and better prediction is observed in normotensives than in hypertensives HD patients. Weight gain does not appear as a predictor of large interdialytic SBP fluctuations.

P1.35 ISOLATED UNCONTROLLED HYPERTENSION AT HOME AND IN THE OFFICE AMONG TREATED HYPERTENSIVE PATIENTS FROM THE J-HOME STUDY

T. Obara¹, T. Ohkubo¹, J. Funahashi¹, M. Kikuya¹, K. Asayama¹, H. Metoki¹, T. Oikawa¹, J. Hashimoto¹, K. Totsune¹, Y. Imai¹. ¹Tohoku University Hospital, Sendai, Japan

Objective: To evaluate the current status of blood pressure (BP) control as measured at home and in the office and to clarify and compare the prevalence and characteristics of isolated uncontrolled hypertension as measured at home (home hypertension) and in the office (office hypertension).

Design: A cross-sectional study.

Method: 3400 patients with essential hypertension (mean age: 66 years; males: 45%) receiving antihypertensive treatment in primary care offices in Japan

Results: Overall, the mean home systolic BP (SBP)/diastolic BP (DBP) was 140/82 mmHg, and the mean office SBP/DBP was 143/81 mmHg. Of the 3400 subjects, 19% had controlled hypertension (home SBP/DBP < 135/85 mmHg and office SBP/DBP < 140/90 mmHg), 23% had home hypertension (home SBP/DBP ≥ 135/85 mmHg and office SBP/DBP < 140/90 mmHg), 15% had office hypertension (home SBP/DBP < 135/85 mmHg and office SBP/DBP ≥ 140/90 mmHg), and 43% had uncontrolled hypertension (home SBP/DBP ≥ 135/85 mmHg and office SBP/DBP ≥ 140/90 mmHg). Compared to controlled hypertension, factors associated with home hypertension included obesity, habitual drinking, relatively higher office SBP, and use of 2 or more prescribed antihypertensive drugs. Compared to uncontrolled hypertension, factors associated with fifice hypertension included younger age, female gender, lower body mass index, no family history of cerebrovascular disease, relatively lower office SBP, and use of one prescribed antihypertensive drug.

Conclusions: Physicians should consider home BP measurements in hypertensive patients who have factors that are associated with home hypertension or office hypertension.

P1.36 ETHNIC DIFFERENCES IN ARTERIAL STIFFNESS AND WAVE REFLECTIONS AFTER CIGARETTE SMOKING

D. Lemogoum¹, J.R. M'Buyamba-Kabangu², M. Leeman¹, L. Van Bortel³, J.P. Degaute¹, Ph. van de Borne¹. ¹Service of Cardiology Erasme Hospital, Brussels, Belgium, ²University of Kinshasa Hospital, Kinshasa, Zaire, ³Heymans Institute of Pharmacology, Ghent, Belgium

Objective: To test the hypothesis that cigarette smoking increases pulse wave velocity (PWV), a marker of arterial stiffness, and augmentation index (AI), a measure of wave reflection, in Blacks more than in Whites. Background: Smoking increases plasma nicotine. Nicotine releases catecholamines and alters arterial distensibility. Nicotine intake per cigarette is greater and serum cotinine levels, the proximate metabolite of nicotine, are higher in Blacks than in Whites.

Methods: We matched Black (n = 30) and White (n = 30) smokers for age and gender. We determined carotid-femoral PWV (PWV_{CF}) and carotid-radial PWV (PWV_{CR}) (ComplioR), Al was measured by applanation tonometry (SphygmocoR), blood pressure (BP), heart rate (HR) and cotinine levels before and after cigarette smoking. We also performed measurements in 16 participants after sham smoking.

Results: Smoking increased AI, PWV $_{CF}$ and PWV $_{CR}$ in the whole population (all p<0.05, n=60). Increases in AI and PWV were related to serum cotinine levels (all p<0.05). Smoking increased more serum cotinine (p=0.01) and mean BP (p=0.03), but raised HR to a lesser extend, in Blacks (+8±4 vs +13±6 bpm in Whites, mean±SD, p=0.01). Blacks increased largely AI adjusted for HR (+7.2±8 vs Whites: +4.4±8%, p=0.03), PWV $_{CF}$ (+1.1±0.2 vs Whites:+0.6±0.3 m/s, p<0.01) and PWV $_{CR}$ (+1.4±0.1 vs Whites: +0.7±0.4 m/s, p<0.01) normalized for mean BP. No changes were observed with sham smoking.

Conclusions: Smoking acutely increases PWV and AI in Blacks more than in Whites. Differences in nicotine metabolism and beta-adrenergic sensitivity could explain these findings.