| 72 | Friday, June 12 |
| 74 | Saturday, June 13 |
| 173 | Sunday, June 14 |
| 263 | Monday, June 15 |
17.00 - 18.30 Orange Hall 1

Parallel Oral Session 9A

INFLAMMATION AND IMMUNITY

Chairpersons:
S. Carugo (Milan, ITALY)
Y. Sirenko (Kiev, UKRAINE)

17.00 • HYPERURICEMIA IS AN INDEPENDENT DETERMINANT OF ARTERIAL STIFFNESS
C. Antza, S. Papakatsika, G. Kotronis, K. Mikoudi, S. Stabouli, V. Kotsis
Thessaloniki, GREECE

17.10 • SODIUM SENSITIVE HYPERTENSION: CAN IT BE ASSESSED BY MEASURING URIC ACID LEVELS?
S. Radenkovic, G. Kocic, D. Stojanovic, B. Mijajlovic, D. Valjevic, J. Radovic, N. Janic
Niš, SERBIA

17.20 • OSTEOPONTIN AND OSTEOPROTEGERIN ACTIVATE MONOCYTES INTO ANTI-INFLAMMATORY PROPERTIES IN THE
PATIENTS WITH HYPERTENSION-RELATED VASCULAR CALCIFICATION
Q. Ge, C. Ruan, P. Gao, Y. Mo, D. Zhu, J. Wang
Shanghai, CHINA

17.30 • CARDIOVASCULAR RISK FACTOR PROFILE IN AN ITALIAN COHORT OF PATIENTS WITH RHEUMATOID ARTHRITIS:
RESULTS OF A THREE YEARS FOLLOW-UP
Milan, ITALY

17.40 • SYMPATHETIC NERVOUS SYSTEM DRIVES RENAL INFLAMMATION BY ALPHA(2A)-ADRENORECEPTORS
H. Hoch 1, L. Hering 1, S. Crowley 2, J. Zhang 2, G. Yang 1, L.C. Rump 1, O. Vonend 3, J. Stegbauer 1
1 Düsseldorf, GERMANY, 2 Durham, NC, USA, 3 Wiesbaden, GERMANY

17.50 • IMPACT OF METABOLIC, HEMODYNAMIC AND INFLAMMATORY FACTORS ON TARGET ORGAN DAMAGE IN
HEALTHY SUBJECTS
M. Blicher 1, R. Kruger 2, T. Hansen 3, M.H. Olsen 1
1 Odense, DENMARK, 2 Potchefstroom, SOUTH AFRICA, 3 Gentofte, DENMARK

18.00 • CARDIOVASCULAR TARGET ORGAN DAMAGE IN PREMENOPAUSAL SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS
AND IN CONTROLS. ARE THERE ANY DIFFERENCES?
A. Paini, L. Andreoli, M. Salvetti, F. Dall’Ara, S. Piantoni, C. Donini, C. Agabiti Rosei, F. Bertacchini, D. Stassaldi, E. Agabiti Rosei,
A. Tincani, M.L. Muiesan
Brescia, ITALY

18.10 • INTERLEUKINS 33 AND 1B SERUM LEVELS ARE CONNECTED TO COMMON CAROTID ARTERIES REMODELING IN
HYPERTENSIVE PATIENTS WITH OBESITY
O. Honchar, I. Ashchukina, O. Kovachova
Kharkiv, UKRAINE

18.20 • PROTECTION AGAINST COMPLEMENT ACTIVITY IS REDUCED IN ARTERIAL HYPERTENSION
F. Castagna 1, J. Wang 1, M. Emri 1, G. Wang 1, S. Jelic 1, G. Parati 1
1 New York, NY, USA, 1 Milan, ITALY
HYPERURICEMIA IS AN INDEPENDENT DETERMINANT OF ARTERIAL STIFFNESS

C. Antza, S. Papakatsika, K. Kotronis, K. Mikoudi, S. Stabouli, V. Kotisis. Hypertension Center, 3rd Department of Medicine, Papanicolaou Hospital, Aristotle University of Thessaloniki, Thessaloniki, GREECE

Objective: The aim of the study was to identify determinants of arterial stiffness in patients with increased uric acid levels.

Design and method: 280 consecutive subjects (51.4% male) aged 52.98 ± 22.9 years were included in the study. Subjects were never treated before for hypertension or uric acid. A physician measured office BP three times in each subject using a mercury sphygmomanometer. All subjects underwent 24h-ABPM on a usual working day. Pulse wave velocity (PWV) was measured after 15 min of rest in the supine position. The subject was not speaking or sleeping in a quiet, semi-darkened, temperature-controlled laboratory. Participants had been advised to refrain from eating, smoking and drinking caffeine beverages and alcohol before measurement. PWV was calculated as the transit time of the arterial pulse along the carotid-femoral distance divided with the distance measured directly.

Results: Carotid-femoral PWV was independently associated (ANCOVA analysis) with age (B = 0.13, P < 0.001), 24h average SBP (B = 0.07, P < 0.05) and uric acid (B = 0.72, P < 0.001), but not with office BP values, e-GFR, lipid levels, gender and BMI. Carotid-femoral PWV was found 8.215 ± 0.41 m/sec (SE) in patients with normal uric acid values and 10.252 ± 0.91 m/sec (SE) in patients with hyperuricemia after adjustment for age, gender, office BP, 24h SBP, 24h pulse pressure, e-GFR, fasting serum cholesterol, triglycerides and BMI. The difference in carotid-femoral PWV between normal uric acid subjects and hyperuricemic patients was 2.037 ± 1.008 m/sec (SE). This difference was statistically significant at the 0.05 level after Bonferroni’s adjustment for multiple comparisons.

Conclusions: Arterial stiffness was found increased in patients with hyperuricemia suggesting a role for increased uric acid in the pathophysiology of large arteries atherosclerosis independent of age, gender, obesity, blood pressure levels and kidney function.

SODIUM SENSITIVE HYPERTENSION: CAN IT BE ASSESSED BY MEASURING URIC ACID LEVELS?

S. Radenkovic 1, G. Kocic 2, D. Stojanovic 1, B. Milojkovic 3, D. Velickovic 3, J. Radovic 1, N. Jancic 1, 3 Institute of Pathophysiology, Medical Faculty, University of Nis, Nis, SERBIA, 2 Institute of Biochemistry, Medical Faculty, University of Nis, Nis, SERBIA, 5 Medical Faculty, University of Nis, Nis, SERBIA

Objective: It was already documented, by many investigators, that hyperuricemia presents an important factor in the development of essential arterial hypertension. The goal of this study was to examine correlation between serum uric acid levels in patients with essential arterial hypertension and index of sodium sensitivity, as the main parameter of salt-sensitive hypertension.

Design and method: The investigation included 236 participants of both sexes. Clinical group included 178 of participants, mean age 59 ± 18.2 years, with at least 5 years of hypertension history and preserved kidney function. They were divided into 2 subgroups according to the serum uric acid levels. Control group involved 58 healthy volunteers, who were age and sex matched with the clinic group. The levels of serum uric acid were measured spectrophotometrically. Sodium sensitivity index was assessed as the main parameter of salt sensitive hypertension. It was calculated as the difference in 24 hours sodium excretion between period of sodium rich diet (250 mmol/24 hours) and sodium lean diet (50 mmol/24 hours), divided by mean arterial pressure, measured twice respectively.

Results: First clinical subgroup had 95 patients, with normal uric acid serum values (256 ± 35 μmol/l), and the second subgroup had 83 patients, with significant increase of uric acid serum values (572 ± 49 μmol/l; P < 0.01). Sodium sensitivity index in the first subgroup had normal values (0.026 ± 0.005), and in a second subgroup was significantly higher (0.078 ± 0.02; p < 0.01). We found a high positive correlation (r = 0.721, p < 0.01) between an increase in serum uric acid level and salt-sensitivity index in patients.

Conclusions: Hyperuricemia and salt-sensitivity index correlate highly, therefore serum uric acid levels may be used as diagnostic parameters of salt-sensitive arterial hypertension in the population of patients with essential hypertension.
Design and method: 34 patients with SLE clinically stable (SLEDAI Score 2.5 +/- 1.5) (mean age 32 ± 7 years, range 19–44) and 34 controls matched for sex, age, body mass index (BMI), clinic blood pressure (BP) and antihypertensive treatment (if present), underwent: 24 hours BP monitoring, echocardiography with tissue Doppler analysis (TDI) for the evaluation of left ventricular (LV) structure and function, carotid ultrasound for intima-media thickness (IMT) and carotid distensibility measurement, and pulse wave velocity measurement for aortic stiffness (PWV).

Results: By definition no difference was observed for age, sex, BMI and clinic BP values and a similar Framingham risk score was observed between SLE and controls (1.3 ± 2.7 vs 1.5 ± 2.3%, p<0.05). No significant differences were observed for all echocardiographic parameters except LV longitudinal systolic function (5m), an early index of LV systolic dysfunction (see Table). Carotid IMT and distensibility, as well as PWV and the prevalence of an abnormal aortic stiffness were both similar in the two groups. At the logistic analysis, PWV was independently associated with LV mass in controls and with the steroid weekly dose in SLE patients.

Conclusions: In patients with SLE and low activity index of the disease we did not observe significant vascular alterations compared to controls with similar cardiovascular risk. The early LV systolic impairment observed in this group of patients needs confirmation in larger cohorts.

### 9A.09 PROTECTION AGAINST COMPLEMENT ACTIVITY IS REDUCED IN ARTERIAL HYPERTENSION

F. Castagna 1, J. Wang 1, M. Emit 1, G. Wang 1, S. Jelic 2, G. Parati 1, 2, 3, S. Luca Hospital, IRCCS Istituto Auxologico Italiano, Dept. of Cardiovascular, Neural and Metabolic Sciences, Milan, ITALY

Objective: Different elements contribute to arterial hypertension (HTN) etiology. Among these, endothelial dysfunction and vascular inflammation are now considered important co-factors. We hypothesized that distinctive molecular pathways of endothelial activation are present in HTN patients.

Design and method: 6 HTN patients, free of any other condition that may have affected the vascular endothelium, (mean[SD] age 40[11], 67% female) and 13 healthy controls (age 39[10], 37% female) participated. Endothelial cells (ECs) were harvested from a superficial forearm vein through 20-gauge angiocatheter by inserting 3 endovascular wires sequentially under sterile conditions. ECs were then washed from wires and fixed on slides. Each harvesting yielded 2000–5000 ECs.

Results: We identified reduced expression of CD59, a plasma membrane-bound protein that prevents the final assembly of the terminal complement complex (TCC), in HTN patients compared to controls (0.2551 vs 0.5790, p = 0.05). In vitro experiments confirmed an increased complement deposition/activity in CD59-knockout endothelial cells vs controls. (C5b-9 positivity[SD] 23.3[4.8]% vs 8.3[2.8]%, p < 0.05)

Conclusions: Protein expression is similar among arterial and venous endothelial cells, but venous ECs are not subject to the direct effect of elevated blood pressure, since they are located in low-pressure districts barely influenced by arterial blood pressure. Therefore, an increased arterial blood pressure cannot be the cause of this protein dysregulation. On the contrary, we suggest that the presence of reduced CD59 expression may be a co-factor in the development of arterial hypertension and the increase in vascular risk.