patients with non-dipper patterns, interleukin-1β was significantly higher, than in dipper patients (2.21 [1.62–2.77] vs 2.26 [2.0–3.11], p = 0.03, Mann-Whitney).

Conclusions: Non-dipper pattern of blood pressure circadian rhythm is associated with increased inflammatory markers in hypertensive patients as well as in normotensive subjects.

CORRELATION BETWEEN VASCULAR INFLAMMATION MARKERS, DIASTOLIC DYSFUNCTION AND CARDIOVASCULAR RISK IN PATIENTS WITH TAKAYASU ARTERITIS

Sebastiano Cicco1, Vanessa Desantis2, Antonio Vaccà3, Gerardo Cazzato4, Antonino G. Solomando1, Cecilia Susca5, Gabriele Brossolo5, Cristiana Caterna1, Aurelia Lamazzuori6, Ilaria Saltarello5, Maria Antonia Frassanito5, Giuseppe Ingravallo5, Leonardo Resta5, Giuseppe Baniéri5, Monica Montagnani5, Roberto Ria5, 1Department of Biomedical Sciences and Human Oncology, Unit of Internal Medicine, University of Bari, Bari, ITALY, 2Department of Biomedical Sciences and Human Oncology, Pharmacology Section, University of Bari, Bari, ITALY, 3Division of Internal Medicine, Department of Medicine, Building 8, University of Udine, I-33100 Udine, ITALY, 4Section of Pathology, Department of Emergency and Organ Transplantation (DETO), University of Bari, Bari, ITALY, 5Department of Admission and Emergency Medicine and Surgery, S. Maria degli Angeli Hospital, ASL Bari, Putignano, ITALY, 6Department of Biomedical Sciences and Human Oncology (DIMO), General Pathology Unit, University of Bari, Bari, ITALY

Objective: Takayasu arteritis (TAK) increases vascular stiffness and arterial resistence. Abnormal immune response is a crucial factor in the pathogenesis of TA. Here, we investigated i) vascular and cardiac ultrasonography parameters as increased cardiovascular risk in TA patients, compared to atherosclerotic patients; ii) Treg and Th17 cells frequency in TA-refractory patients treated with infliximab

Design and method: Clinical, instrumental and biochemical data in patients with active TAK were compared in a case control study were compared to age- and sex-matched atherosclerotic patients. In a subpopulation of TA patients, Treg/Th17 cells percentage was measured before (T0) and after 18 months (T18) of infliximab treatment. Echocardiogram, supraaortic Doppler ultrasound, and lymphocytogram were carried out in all patients. Histological and immunohistochemical analysis were performed to correlate vessel wall patho-morphology and clinical/laboratory results.

Results: TA patients displayed increased aortic valve dysfunction and diastolic dysfunction compared to atherosclerosis. Moderate-to-severe aortic regurgitation correlates with the highest serum levels of uric acid in TAK patients. A significant increase in aortic stiffness was associated with peripheral T lymphocyte levels. Increase in CD3+CD4+ and CD8+ infiltration and High-mobility group box 1 (HMG1) was significantly higher in TAK group. CD15+ neutrophils were significantly higher in TAK, suggesting an association with inflammation-related vascular damage. Flow cytometric Tregs percentage was significantly reduced in TAK patients. Interestingly, in patients treated with infliximab, this value significantly increased at T18 compared to T0. Concomitantly, the frequency of CD3+CD4+IL-17+ cells behaved in the opposite way: the higher number of Th17 cells was observed in TAK patients. Interestingly, in patients treated with infliximab, this value significantly increased at T18 compared to T0.

Conclusions: These observations strengthen the clinical efficacy of infliximab in TAK patients, supporting the idea that biologic therapy may achieve a better control of TAK progression and help to stabilize the Treg/Th17 score toward values similar to those found in atherosclerotic patients

MICRO- AND MACROVASCULAR ALTERATIONS IN RHEUMATOID ARTHRITIS COMPARED TO TYPE 2 DIABETES MELLITUS: EVIDENCE OF SIMILAR DEGREE OF ORGAN DAMAGE

Panagiota Amyfani, Barbara Nikolaidou, Elena Gavrilaki, Areti Triantafyllou, Antonios Lazaridis, Panagiotis Dolgyras, Antonia Dimitriadou, Stella Douma, Eleni Gavriilaki, 3rd Department of Internal Medicine, Papageorgiou General Hospital, Thessaloniki, GREECE

Objective: Rheumatoid arthritis (RA) is characterized by excess cardiovascular risk attributed to the chronic inflammatory load in combination with the accumulation of traditional cardiovascular risk factors. RA is being currently perceived as a cardiovascular risk factor equivalent to type 2 diabetes mellitus (DM2), although recent relevant studies have provided conflicting results. The aim of the present study was to compare markers of micro- and macrovascular dysfunction between patients with relatively well-controlled RA and patients with DM2.

Design and method: Patients with RA who presented low levels of systemic inflammation and patients with DM2, who were free from cardiovascular comorbidities including hypertension, were studied. A control group that comprised of healthy volunteers was additionally included. All participants underwent application tonometry (Sphygmocor device) to assess a carotid-femoral pulse wave velocity (PWV), a augmentation index corrected for 75 bpm (AIx@75), central systolic/diastolic blood pressure (cSBP/cDBP), central blood pressure (cMBP), carotid intima-media thickness, SEVR, subendocardial viability ratio.

Results: We studied 31 patients with RA at remission or low disease activity, 32 patients with DM2 and 69 controls matched for age and office blood pressure. Significant differences were observed in several outcomes of the study between patients and controls; by contrast, none of the studied parameters (PWV, AIx@75, cSBP/cDBP, cMBP, carotid intima-media thickness, SEVr) significantly differed between patients with RA and patients with DM2 (Table 1).

Conclusions: Patients with RA present impaired markers of micro- and macrovascular dysfunction, even in the absence of cardiovascular comorbidities including hypertension and whilst at remission or low disease activity. Moreover, the degree of divergent micro- and macrovascular alterations in this well-characterized group of RA patients appears comparable to that associated with DM2. These findings are line with the widely held perception that RA should be regarded as a novel cardiovascular risk factor equivalent to DM2.

ASTRAGALOSIDE IV INHIBITS H2O2-INDUCED APOPTOSIS IN H9C2 CELLS BY ATTENUATING OXIDATIVE STRESS AND MITOCHONDRIAL DAMAGE

Miaomiao Qi, Qiongying Wang, Runmin Sun, Litian Jiang, Yu Lan Zhuang University Second Hospital, Lanzhou, CHINA

Objective: To investigate the effects and mechanisms of As-IV on H2O2-induced apoptosis in H9C2 cells.

Design and method: H9C2 cells were divided into three groups, including normal control group (cultured without intervention), H2O2 group (cultured with 200μM H2O2 for 2 hours), As-IV group (cultured with 100μM/L As-IV for 1 hour). For cells treated with H2O2, group (pretreated with 100μM/L As-IV for 1 hour, then incubated with H2O2 for 2 hours). FITC/PI and DCFH-DA were stained to examine levels of apoptosis and ROS receptively. Levels of SOD and MDA were measured. Stained with JC-1 probe to observe the change of mitochondrial membrane potential (MMP). Western-blot was detected to analyze the expression of Drp-1.

Results: 1. The apoptosis rate of H2O2 group ((13.22 ± 3.17) vs 40.55 ± 13.74%), P < 0.05) was significantly higher than that of control group, which was lower in As-IV group (3.43 ± 4.3 vs 40.55 ± 13.74%), P > 0.05. 2. The ROS levels were higher in H2O2 group than control group(73.53 ± 5.33 vs 39.02 ± 7.90%), P < 0.05, which were significant reduced in As-IV with H2O2 group(57.6 ± 4.63 vs 73.53 ± 5.33%), P < 0.05) Meanwhile, the activity of MDA was increased and the activity of SOD was decreased in H2O2 group compared with that in control group(10.97 ± 0.56 vs 4.85 ± 0.55) nmol/mL, P < 0.05, (11.92 ± 2.34 ± 3.519 ± 7.287.32)μL, P < 0.05). Compared with H2O2 group, As-IV with H2O2 group significantly decrease the activity of MDA and increase the activity of SOD.(6.85 ± 0.79 vs 19.10 ± 0.07) %, P > 0.05). Compared with H2O2 group, MDA was significantly increased and the expression of Drp1 was higher in H2O2 group than that in control group((0.31 ± 0.1 vs 1.52 ± 0.25) %, P < 0.05). Compared with H2O2 group, MDA was significantly increased and the expression of Drp1 was upregulated((0.65 ± 0.38 vs 0.31 ± 0.01) %, P < 0.05), (0.72 ± 0.005 vs 0.86 ± 0.0003) %, P < 0.05).

Conclusions: As-IV exerts protective effects on H2O2-induced apoptosis by attenuating oxidative stress and mitochondrial damage.