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2007 and relative to all diabetes aged 45-75 years were extracted, HbA1c (\%), systolic and diastolic blood pressure (BP, mmHg), total cholesterol (TC), HDL-C, Triglycerides (TG), LDL-C (all mg/dl), and prescriptions of drugs used to treat diabetes, hypertension, and dyslipidemia were analyzed. The software for the extraction of all measurements was specifically developed by WeTeDa.s.r.l., which also cured the coding system to fulfill privacy protection. Data from first 60 patients with atheroembolic disease were included in the study. An adjusted logistic regression was used to evaluate potential predictors of non-target for any of the above CV risk factors.

Results: 4479 pts (515M, 495F), aged 64.7y (mean) selected inclusion criteria and were considered for analysis. In these pts, the most recorded parameters were: HbA1c (at least 1 value in 84.7% of pts); BP (92.9%); TC and TG (46.6%); HDL-C (44%). During the study period (18mo), HbA1c was measured 3.65 times, BP 3.84, lipid parameters 1.35. At the last available data written in the follow-up period (16mo); HbA1c > 7% (cancer) was achieved in 39.3% or pts (76.7% of whom insulin-treated). Non-adherence to insulin therapy, female gender, and ADK affiliation represented the main determinants of suboptimal HbA1c value. In statin-treated pts (unfortunately only 16.5%), target values for lipid parameters were defined in 89% for TC, 58% for HDL-C, 60% for TG. Non-statin-treatment (for TC), female gender (for HDL-C) and BMI (for TG) represented important predictors of failure to gain lipid target values. However, in all these parameters we always observed an improvement between the first and the last data entry, this reflecting an appreciable, although partially ineffective) therapeutic effort. Discussion: Our results show that achievement of the therapeutic target is still far to be reached in a large part of "real world" high-risk diabetic population. The CV risk is not only right now, as previously indicated, but may be also the consequence of inappropriate or suboptimal therapeutic strategies, indicating that this area will deserve a higher intensity of clinical governance.

Conclusions: This study demonstrates that in type 2 DM pts: (a) monitoring and optimal treatment of all CV risk factors is critical; (b) insulin still represents the most effective drug for optimal glyemic control; (c) complete lipid profile is undervalued and statins still remain under-prescribed. Concerted aimed strategies may detect the influence of different therapeutic strategies on global CV risk profile, and could be used for monitoring their efficacy in large and complex populations.

[17] p65hsc GENE EXPRESSION IS INCREASED IN ESRD PATIENTS ON HD MODALITIES: RELATIONSHIPS TO SYSTEMIC OXIDATIVE STRESS, INFLAMMATION, AND CARDIAC PARAMETERS

I. Bucučević, M. Zanetti, R. Barabarci, A. Bosotti, F. Bianco, G. Panacea, L. Castin, G. Guarinieri. University of Trieste, Italy

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Oxidative stress is involved in uremia-associated atherosclerosis, p65hsc, a novel mediator of oxidative stress, promotes endothelial dysfunction and vascular disease. No information is available on p65hsc gene expression in end-stage renal disease (ESRD) on hemodialysis (HD) and on its potential associations with plasma markers of oxidative stress as well as with clinical parameters. The objective of this study was to assess p65hsc gene expression from peripheral blood mononuclear cells (PBMCs) of ESRD patients on HD compared with control subjects and its potential associations with plasma TBARS, a quantitative oxidative stress as well as with inflammatory, anthropometric and clinical parameters. Total mRNA was extracted from PBMCs from twenty ESRD patients before their hemodialytic session and from twenty-one control subjects. p65hsc and TNFa mRNA levels were assessed using real time PCT. Plasma glucose, total, HDL and LDL cholesterol, triglycerides, albumin and CRP were also measured. Compared to control subjects, p65hsc and TNFa mRNA was significantly higher in the patients. In univariate analysis p65hsc mRNA was directly (R = 0.363, P = 0.006) associated with plasma TBARS in the patients. Among clinical parameters, p65hsc mRNA was directly correlated (R = 0.423, P = 0.059) correlated with the systolic blood pressure (R = 0.486, P = 0.025) in the ESRD on HD group. None of these associations were detected in control subjects. In contrast, a direct association between p65hsc and TNFa mRNA was demonstrated both in the patients (R = 0.499, P = 0.02) and in control subjects (R = 0.396, P = 0.014). In conclusion, p65hsc gene expression is upregulated during ESRD on HD and is associated with markers of oxidative stress and inflammation as well as with some cardiovascular risk factors. These data suggest that p65hsc contributes to systemic oxidative stress in uremia and therefore may be involved in vascular disease with ESRD.

[18] INFAMMATION AND REMODELING MARKERS IN AORTIC VALVE DISEASE: ROLE OF AIIMS, TIMPS, OPN AND GGT IN LESION DEGENERATION

S. Cappelli, A. Vianello, B. Rossi, M.C. Epistolo, A. Mazzone, M. Glauber, A. Perissinotti, S. Verrini, V. Fantini, M. Tegugno, G. Fantini, A. Strumia. Department of Human Pathology and Oncology, University of Siena, Siena, Italy

E-mail: cappelli601@unisi.it

Background: degenerative aortic valve disease, which leads to aortic valve insufficiency (AVI) is characterized by leaflet stiffening and calcification and presents the histological features of atherosclerotic lesions: inflammatory cells, lipid deposits, neovessels, calcified nodules and fibrotic tissue. Twenty-one control subjects. p65hsc and TNFa mRNA levels were assessed using real time PCT. Plasma glucose, total, HDL and LDL cholesterol, triglycerides, albumin and CRP were also measured. Compared to control subjects, p65hsc and TNFa mRNA was significantly higher in the patients. In univariate analysis p65hsc mRNA was directly (R = 0.363, P = 0.006) associated with plasma TBARS in the patients. Among clinical parameters, p65hsc mRNA was directly correlated (R = 0.423, P = 0.059) correlated with the systolic blood pressure (R = 0.486, P = 0.025) in the ESRD on HD group. None of these associations were detected in control subjects. In contrast, a direct association between p65hsc and TNFa mRNA was demonstrated both in the patients (R = 0.499, P = 0.02) and in control subjects (R = 0.396, P = 0.014). In conclusion, p65hsc gene expression is upregulated during ESRD on HD and is associated with markers of oxidative stress and inflammation as well as with some cardiovascular risk factors. These data suggest that p65hsc contributes to systemic oxidative stress in uremia and therefore may be involved in vascular disease with ESRD.

[19] LIGHT AND REGULAR CIGARETTE CONSUMERS AND ENDOThelial DYSFUNCTION

S. Castelnovo1, B. Frigerio1, M. Amato2, A. Ravanelli2, E. Tremoli1,2, R. Sirtori2, D. Baldessari1-2, 1Department of Pharmacological Sciences, University of Milan, and 2Cardiologico Monzino, IRCCS, Milan, Italy

E-mail: samuela.castelnovuno@unimi.it

Background: Several studies have shown that cigarette smoking has a negative effect on cardiovascular health and that it’s an independent determinant of flow mediated dilation (FMD), a well accepted indicator of endothelial function. The present work aimed to study how smoking may induce changes in the functional and morphological characteristics of the carotid artery.

Methods: We recruited 64 aortic valve disease patients (pts) undergoing surgery for treatment of aortic valve disease and were evaluated in all patients. All patients underwent the following examinations: sphygmography tests for plasma and tissue MPAs activity; ELISA for TIMPs, OPN and GGT determination; immunohistochemistry to localize proteins.

Results: Tissue calcification was predominant in the AS group in comparison to the AS group (82.50% vs 53.50%; P < 0.002). The tissue TIMP2/MPM2 ratio was more elevated in the AS group in comparison to the AS (P < 0.01). In the entire group of diseases (AS and AS), calcification was associated with inflammation and neovascularization (P < 0.0001) and was inversely correlated with tissue TIMP2/MPM2 ratio (P < 0.016). GGT was expressed by C68R and TRAP+ cells found around neovessels and calcified aortic valve. Plasma levels of GGT were not significantly different between groups, in the entire group of diseases, enzymatic activities of GGT and GGT were associated with high BMI levels (P < 0.04) in patients with dilated ventricular chambers (P < 0.05). GGT activity correlates with BMI levels (P < 0.026) and with diameter of the left ventricle (P < 0.03).

Conclusions: We confirm the close relationships between pathologic processes underlying both atherosclerosis and degenerative aortic valve disease. We report a physiopathologic gradient for GGT with its increase in the AS group and its decrease in the AS group.

[20] BALANCE BETWEEN CIRCULATING ENDOTHELIAL PROGENITOR CELLS (EPCs) AND MATURE CIRCULATING ENDOTHELIAL CELLS (ECs) IN RELATION TO THE SEVERITY OF PERIPHERAL ARTERIAL DISEASE

F. Cesari1, F. Sotti1, A.M. Gorl1, R. Coparole1, M. Di Mare3, G. Pratesi1, R. Puliti1, C. Pratesi1, R. Abbate2, G.F. Gensini1. 1Department of Medical and Surgical Critical Care, Thrombosis Centre, University of Florence, Florence, Italy, 2Ospedale Universitario Careggi, Florence; Central Laboratory, 3Ospedale Universitario Careggi, Florence; 4Unit of Vascular Surgery, Azienda Ospedaliero-Universitaria Careggi, Florence, Italy

E-mail: francesa.cesari@gmail.com

Introduction: The maintenance of endothelial health depends, not only on the local milieu, but also on circulating endothelial progenitor cells (EPCs) or mature circulating endothelial cells (ECs) that support the viability of vascular endothelium and promote revascularization of ischemic areas. On the other hand, circulating mature endothelial cells (ECs) are considered a marker of endothelial injury. Previous studies demonstrated reduced number of EPCs in peripheral arterial disease (PAD) patients, but few data are available on ECs.