decrease up-regulation of LDLR, VLDLR and CD36 either induced by HG or by HG+cytokine cocktail. At 48h, HG was only able to up-regulate LDLR mRNA expression. The cytokine cocktail was able to induce a significant increase in CD36 [17.1fold (NG vs HG)] and LDLR [3.1fold (NG)/2.6fold (HG)] no effect was observed on VLDLR mRNA expression. troglitazone, mevinolin, LY were having no effect on VLDLR, LDLR regulation. troglitazone was the only one to reduced cytokine cocktail-induced CD36 up-regulation. In conclusion, PPARγ agonist was the only pharmacological agent able to induce an early and a late regulation of CD36 mRNA. Statin and protein kinase CbII inhibitor were only able to early regulate CD36, LDLR and VLDLR.

W01.14 WAIST/HIP RATIO, GLYCOSILATED HAEMOGLOBIN AND HIGH BLOOD PRESSURE ARE THE MOST DETERMINING FACTORS OF CAROTID INTIMA-MEDIA THICKNESS

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Introduction: Carotid intima-media-thickness (IMT) has been introduced as an early marker of atherosclerotic disease. Furthermore, a high prevalence of increased carotid IMT has been found in the hypertensive population.

Objectives: 1. To determine the prevalence of increased IMT in high risk patients.

2. To evaluate its impact on anthropometric, metabolic and haemodynamic parameters.

3. To correlate IMT and endothelial dysfunction.

Material and Methods: Study population: n=64 patients, 32m and 32f, aged 21-87 years

Hypertension: JNC-7.

Syndrome X: ATP-III.

IMT: HP Doppler-Ultrasound.

Vascular risk: Framingham-PROCAM tables.

Anthropometric parameters: BMI (kg/m^2), waist-circumference (cm), waist/hip ratio.

Metabolic parameters: HbA1c (HPLC) Insulin-Resistance (HOMA), Fasting glucose, HDL, Cholesterol, and Triglycerides (HITACHI-autoanalyzer); LDL (Friedewald formula).

Endothelial function: PAI-1 (ng/ml) by Menarini-ELISA.

ABPM: Spacelabs 90207.

Statistical analysis: T-Student, Chi-Square, multivariate analysis.

Results: Theprevalence of IMT >= 0.12 was 59.3%. 86,1% of such patients fulfilled the Syndrome X criteria and 71% the hypertension criteria.

2. Patients with high IMT showed greater age (p=0.0001) and CVR [Framingham (p=0.0001); PROCAM (p=0.000)]

3. In the multivariate analysis the significant parameters were: HbA1c (p=0.005), waist/hip ratio (p=0.009) and presence of hypertension (p=0.049).

4. A linear correlation exists IMT/PAI: p=0.026.

Conclusions: 1. The independent factors associated with increased IMT were: waist/hip ratio, HbA1c and hypertension.

2. An statistically significant correlation exists between IMT and PAI-1, that could explain the high risk of atherosclerotic disease in these patients.

W01.15 INTERLEUKIN INFLAMMATORY PHENOTYPES IN PREMATURE CORONARY ATHEROSCLEROSIS

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Patients with premature coronary syndromes impose an enormous challenge to the long-term management of atherosclerosis. Though the inflammatory markers available are sensible, but lack specificity and are not permanent.

We analysed the polymorphisms of two pleiotropic inflammatory mediators, interleukin IL-8 (-251 A/T), and IL-6 (-174 G/C) promoters. PCR-SSP techniques rendered hyper, medium and hypoproductive phenotypes. We studied 393 patients (P), after a premature coronary syndrome were compared with 276 aged, healthy controls (C); both had similar AA-AT/TT and GG-GC/CC distributions.

Results: Patients were 44 years old and controls 69; (p<0.000). With the exception of smoking (P81% vs. c52%; p<0.000), dyslipidemia (P63% vs. c37%; p<0.05), and family history of premature CHD (P37%vs. c14%; p<0.000) major risk factors were more prevalent among controls. Patient study showed males were 4 years younger (F47 vs. M43; p<0.048). Gender differences included higher prevalence of diabetics (M74%vs. F25%; p<0.028), and smokers (M86%vs. F14%; p<0.000) among males. Multivessel disease related to dyslipidemia (86%; p<0.007), metabolic syndrome (35%; p<0.041) and hyperproductive IL-8 phenotype (75%; p<0.033) in males. Recurrence related to smoking (p<0.045), diabetes (p<0.05) and PCI (p<0.041). Hyperproductive IL-6 correlated with metabolic syndrome (p<0.001) and IL-8 with multivessel disease (p<0.011). Hyperproductive IL-8 phenotype (p<0.010) explained 73.4% of recurrence in a logistic regression model built with risk factors, gender, clinical features and both phenotypes.

Conclusions: Despite an unusual model with a considerable age gap between patients and controls, patients had a higher prevalence of some major risk factors. Hyperproductive interleukin-8 phenotype is a suitable and permanent marker of recurrence that may contribute to characterise individual variability.

W01.16 DISTINCT YET COMPLIMENTARY ROLES OF IIbIIIa INHIBITORS AND HEPARIN DURING PLATELET ACTIVATION: IMPLICATIONS FOR RESTENOSIS FOLLOWING PCI

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Introduction To better understand the mechanism behind the beneficial effects of heparin and IIbIIIa blockers when given together during percutaneous coronary interventions (PCI), we studied the effect of unfractionated heparin (UFH) versus low molecular weight heparin (LMWH) in combination with IIbIIIa blockers, on platelet aggregation, P-selectin expression, PDGF secretion and secondary coronary artery smooth muscle cell proliferation (cSMC). Persistent platelet activation despite adjunctive IIbIIIa therapy may lead to release of mediators that promote acute thrombosis and restenosis after PCI.

Methods Washed platelets were stimulated with thrombin in the presence or absence of UFH, LMWH (Enoxaparin) and/or a GpIIbIIIa blocker (Abciximab, Eptifibatide and Tirofiban).

Results Although IIbIIIa antagonists effectively blocked aggregation, UFH and LMWH were more effective at blocking activation. UFH was significantly better than LMWH at inhibiting P-selectin expression (P=0.001) and PDGF release (P=0.012), and UFH alone blocked the ability of thrombin activated platelets to promote cSMC proliferation (P < 0.0001).

Conclusion UFH produces significantly greater inhibition of platelet activation and smooth muscle proliferation than LMWH, at least in vitro, thus cautioning against the adoption of LMWH as the standard during PCI before clinical trials are completed.

W01.17 MATRIX METALLOPROTEINASE 2 AND 9 EVALUATION IN PATIENTS WITH OR WITHOUT DIABETES DURING ACUTE CORONARY SYNDROME AND AFTER THE ACUTE EVENT

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Objective: Matrix metalloproteinases (MMPs) are critical for vascular remodelling by regulating degradation of the extracellular matrix. We evaluated MP2 and MMP9 levels in diabetic or nondiabetic patients with acute coronary syndrome (ACS) and after 3 months.

Methods: We detected by ELISA serum MMP2 and MMP9 levels in 40 patients aged (mean±SD) 68±9 years with ACS, evaluated at diagnosis and after a 3-months follow-up. Of these, 27 patients aged 67±11 years resulted nondiabetics, and 13 patients aged 76±5 years resulted diabetics.

Results: MMP2 and MMP9 levels were not significantly different in both groups during ACS; MMP2 levels were significantly higher (p< 0.0001) in nondiabetic patients and significantly higher (p=0.002) in diabetic patients after 3 months compared to the period of the acute event, respectively. MMP9 levels were not significantly increased (p= ns) in nondiabetic patients and significantly higher (p=0.046) in diabetic patients after 3 months compared to the period of the acute event, respectively.