rotic plaques, (bilateral 1272; right 76; left 148) corresponding with an almost 5% higher prevalence of unilateral plaques left than right (P < 0.0001). Mean carotid wall thickness was also higher on the left side compared to the right with respectively 2.8 ± 1.3 mm and 3.1 ± 1.2 mm (P < 0.0001), whereas degree of stenosis did not differ. Plaques located in the right carotid artery had significantly more frequent calcification (42% vs 34%) as predominant component, suggesting a more stable phenotype, whereas on the left side, IPH (7% vs 10%), indicating a more vulnerable phenotype, and fibrous tissue (43% vs 47%) were significantly more prevalent. LRNC was equally distributed. Subjects with an atherosclerotic plaque on the left side have therefore an OR for IPH, fibrous tissue and calcification of 1.5, [0.9-1.4], 1.2, [1.1-1.4] and 0.7, [0.5-0.8], respectively.

Conclusion: Although atherosclerosis is a systemic disease, we found an unusual distribution of carotid atherosclerotic plaque size and composition in the general population. IPH and fibrous tissue were more frequent observed in the left carotid artery, whereas calcification was more often present in the right. Our findings suggest that the prediction of cerebrovascular disease to the left side may be explained by the vulnerable phenotype of plaques in the left carotid artery.

P2414 | BEDSIDE

Purpose: We previously validated a gene expression score (GES) comprising age, sex, and 23 genes for determining obstructive coronary artery disease (CAD, ≥50% stenosis) likelihood, measured by quantitative coronary angiography in an invasive angiography population. The GES validity in patients upstream in the referral path and the relationship with CT-angiography (CTA) parameters are unknown.

Methods: Non-diabetic patients referred for myocardial perfusion imaging (MPI) were enrolled; before MPI, blood samples were obtained for GES. If clinically indicated, patients had invasive angiography; all others had research CTA to obtain anatomical information. CTA scans were evaluated qualitatively by 2 independent readers and a protocol validated calcium scores (CAC, Apolipoprotein) were also evaluated;

Results: A total of 431 patients had a GES score; MPI result, and either CT or invasive angiograms with 63 obstructive CAD cases. For GES, ROC AUC was 0.79, and specificity values of adropin levels were 90.7% and 70.9% respectively (cut off 2.73). In conclusion, lower adropin levels were associated with CAD. Adropin is an independent risk factor for CAD.

P2414 | BEDSIDE
Deficiency of a new protein associated with cardiac syndrome X; called adropin by A. Celik, M. Malin, M.A. Kobat, A. Baydas. Elazig Education and Research Hospital, Elazig, Turkey

The pathophysiology of Cardiac Syndrome X (CSX) is still unclear, but most patients with CSX have endothelial dysfunction. It has been shown that adropin uniquely affects the regulation of endothelial function. The purpose of the study was to evaluate the role of adropin in CSX. 86 consecutive Cardiac Syndrome X-diagnosed patients and 86 age-sex matched healthy subjects were enrolled into the study. Serum adropin (using an ELISA method) were measured in each subject. The baseline characteristic properties of subjects patients showed no significant differences between the two groups with respect to sex distribution, age, fasting glucose, serum creatinine, total cholesterol, LDL-C, HDL-C, triglyceride and mean platelet volume (p < 0.05 for all). The history rate of diabetes mellitus and hypertension were significantly higher in the CSX than the control group (respectively, 34.8% vs 15.1%, p < 0.001; 50.0% vs 46.5% vs 29.1%, p < 0.01 for hypertension). The adropin levels were significantly lower in CSX patients than healthy subjects (1.7 ± 0.8 mg/mL and 3.4 ± 1.8 mg/mL, respectively; p < 0.001). The BMI values of CSX patients were significantly higher than control subjects (28.1 ± 2.4 kg/m² and 26.0 ± 3.7 kg/m², respectively; p < 0.001). Plasma nitrite/nitrate levels were lower in patients with CSX than control subjects (15.9 ± 1.6 μmol/L vs 25.4 ± 2.8 μmol/L, respectively; p < 0.001) and they have a significantly positive correlation with plasma adropin levels (r = 0.463, p < 0.01).

In the multiple linear regression analysis, nitrite/nitrate levels, BMI, and adropin were found to be independent risk factors for CSX (Table 1). A ROC curve is used to identify the ability of adropin levels to predict the cardiac syndrome X. The area under the ROC curve was 0.854 for adropin levels (P = 0.0001). The sensitivity and specificity of adropin levels were 90.0% and 79.0% respectively (cut off value 2.73). In conclusion, serum adropin levels were associated with CSX. Adropin is an independent risk factor for CSX.

Table 1. The linear regression analysis of factors predicting CSX

<table>
<thead>
<tr>
<th>Nitrite/nitrate</th>
<th>BMI</th>
<th>Adropin</th>
</tr>
</thead>
<tbody>
<tr>
<td>-0.836</td>
<td>0.004</td>
<td>-0.194</td>
</tr>
<tr>
<td>-0.001</td>
<td></td>
<td>0.005</td>
</tr>
</tbody>
</table>

P2415 | BEDSIDE
A novel lamin a/c mutation in a Dutch family with premature atherosclerosis by A.W. Weterings1, I.A.W. Van Rijsingen2, A.S. Tromp3, A.H. Zwinderman4, R.H. Lekanne Deprez2, M.M.A.M. Mannens5, M.A. Van Den Bergh Weerman6, A.C. Van Der Wal7, S.J. Pinto7. 1Academic Medical Center, Department of Vascular Medicine, Amsterdam, Netherlands; 2Academic Medical Center, University of Amsterdam, Department of Cardiology, Amsterdam, Netherlands; 3Academic Medical Center, Department of Clinical Genetics, Amsterdam, Netherlands; 4Academic Medical Center, Department of Clinical Epidemiology Biostatistics & Bioinformatics, Amsterdam, Netherlands; 5Academic Medical Center, Department of Pathology, Amsterdam, Netherlands

Objective: We report a novel lamin A/C (LMNA) mutation, c.667G→A, in a family with extensive atherosclerosis, diabetes mellitus and steatosis hepatitis.

Methods: In silico analysis (using Alamut version 2.2), co-segregation analysis, electron microscopy, extensive phenotypic evaluation and literature comparison were used to determine the significance of this mutation.

Results: The father of three siblings died at the age of 45 years. The three siblings and the brother and sister of the father were referred to the cardiovascular genetics department, because of the premature atherosclerosis and dysmorphic characteristics observed in the father at autopsy. Clinical evaluation revealed atherosclerosis, insulin resistance and hypertension in the proband and dyslipidaemia and hepatic steatosis in all the patients with the mutation.

Conclusion: Based on the facts that in silico analysis predicts a possibly pathogenic mutation, the mutation co-segregates with the disease, only fibroblasts from mutation carriers show nuclear blebbing and a similar phenotype was reported to be due to certain missense mutations in LMNA we conclude that we deal with a pathogenic mutation. We conclude that the phenotype is similar to Dunnigan-type familial partial lipodystrophy.