3.1: AORTIC STIFFNESS IN MIDDLE AGED WOMEN IS HERITABLE AND RELATES TO BLOOD PRESSURE AND AORTIC CALCIFICATION: A TWIN STUDY


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3.1 AORTIC STIFFNESS IN MIDDLE AGED WOMEN IS HERITABLE AND RELATES TO BLOOD PRESSURE AND AORTIC CALCIFICATION: A TWIN STUDY

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Background: Pulse wave velocity (PWV), measure of aortic stiffness, is predictive of cardiovascular events. PWV is strongly related to age and blood pressure but its relation to other risk factors and presence of calcification is unclear. We sought to determine the association between PWV and cardiovascular risk factors, aortic calcification and heritability of PWV.

Methods: Subjects were 900 female twins (504 dyshygic, 396 monohygric), 53-63 years (Interquartile range), from TwinsUK cohort. PWV was determined over the carotid-femoral region using the Sphygmocor system. Age matched women (n = 40) with PWV in the 1st and 3rd tertiles of the PWV distribution (entire cohort) underwent computed tomography from the carotid to iliac bifurcation to determine calcification. Calcium content was scored using the Agaston method. Heritability of PWV was determined using structural equation modelling.

Results: In multivariate regression PWV was significantly correlated with age, mean arterial blood pressure (MAP) and heart rate (standardized regression coefficients, β = 0.41, 0.39 and 0.20 respectively, each P < 0.001). PWV was not significantly associated with LDL-cholesterol, HDL-cholesterol, smoking or body mass index. Aortic calcification was greater (median 450 vs. 63 units, P = 0.001) in the highest compared to lowest tertile of PWV and was independently associated with PWV in regression analysis (β = 0.48, P < 0.01). Heritability of PWV was 0.54 and when corrected for MAP and heart rate 0.51.

Conclusion: In women aortic stiffness is heritable and relates to age, blood pressure and aortic calcification but not to other conventional cardiovascular risk factors. Genes involved in aortic calcification may be important determinants of PWV.

3.2 IS AORTIC STIFFNESS READY FOR CLINICAL PRACTICE? RESULTS FROM THE ROTTERDAM STUDY

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Background: Vascular Ehlers-Danlos syndrome (vEDS) is a rare severe genetic disease which results from mutations in the gene encoding type III procollagen (COL3A1), characterized by vascular and/or hollow organ ruptures. No treatment is yet validated. We tested the ability of celiprolol, a beta1-adrenoceptor antagonist with a beta2-adrenoceptor agonist action, for preventing the complications of vEDS in a prospective, randomized, open, blinded endpoints trial.

Methods: Fifty three previously untreated vEDS patients were randomized to a 5-year treatment with either celiprolol (n = 25) or no treatment (n = 28). The two groups were matched for demographic, medical historic and clinical characteristics. Celiprolol was up-titrated from 100 to 400 mg by steps of 100 mg every 6 months. The primary end-point was an arterial event (rupture or dissection, fatal or not) occurring during follow-up. Secondary endpoints were intestinal or uterine rupture or major clinical events, related to vEDS, judged by the event committee.

Results: Mean duration of follow-up was 47 (± 15) months. The study was ended prematurely by the safety monitoring board since significant differences were reached between two groups. The primary endpoint was reached by 5 patients (20%) in the celiprolol group and by 14 patients (50%) in the control group (hazard ratio, 0.36; 95% CI, 0.15 to 0.88; P = 0.04). Primary