1.3: PROGNOSTIC VALUE OF CAROTID-FEMORAL PULSE WAVE VELOCITY FOR CARDIOVASCULAR EVENTS: AN IPD META-ANALYSIS OF PROSPECTIVE OBSERVATIONAL DATA FROM 14 STUDIES INCLUDING 16,358 SUBJECTS


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Oral Presentation Abstracts

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1.1 PROGNOSTIC VALUE OF COMBINED ASSESSMENT OF AORTIC STIFFNESS AND CALCIFICATION IN DIALYSIS PATIENTS: OUTCOME DATA OF THE CALCIFICATION OUTCOME IN RENAL DISEASE (CORD) STUDY

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Radiographic calcification and arterial stiffness each individually have been shown to predict outcome in dialysis patients. However, it remains unknown whether combined assessment of these markers of cardiovascular (CV) damage also provides additional predictive value.

Scoring of abdominal aortic calcification (AAC) using plain lateral abdominal x-ray and measurement of carotid-femoral pulse wave velocity (PWV) were performed in a cohort of 1084 prevalent dialysis patients recruited from 47 European dialysis centers. After a follow-up of 2 years, 234 deaths and 91 nonfatal CV events were recorded. Compared with the lowest tertile of AAC, the risk of an event was increased by a factor 3.7 in patients with a score of 5 to 15 (middle tertile), and by a factor 8.6 in patients with scores of 16 to 24. Additionally, each 1-m/s increase in PWV was associated with a 15% higher risk. At higher AAC (scores >24), the effect of PWV was attenuated because of a negative PWV x AAC interaction (hazard ratio [HR]: 0.895 and 0.865 for middle and upper AAC tertiles). In Cox-regression analysis accounting for age, diabetes, and creatinine, arterial hypertrophy and atherosclerosis were significantly greater in the two DM groups and not increased by coexistent hypertension. Although hypertension significantly altered arterial structure and function compared to normal, differences were eliminated by consideration of systolic pressure. Among 2441 participants without CV at baseline, events occurred in 10.1% of normals, 17.8% with hypertension alone, 25.5% with DM alone, and 29.3% with both. Rates were significantly higher in the 2 DM groups and not increased by coexistent hypertension. Adjusted hazards ratios were 1.69 (p<0.001) for hypertension alone, 3.16 (p<0.001) for DM alone, and 3.85 (p<0.001) for both (p<0.001 for trend).

Conclusions: Both hypertension and DM cause increased subclinical and clinical CV. The impact of hypertension on CV is largely attributable to increased distending pressure. Higher rates of vascular hypertrophy, subclinical atherosclerosis and incident CV in DM are not attributable to coexistent hypertension in this population.

1.2 DIFFERENTIAL IMPACTS OF HYPERTENSION AND TYPE 2 DIABETES MELLITUS ON ARTERIAL DISEASE AND CARDIOVASCULAR OUTCOMES: THE STRONG HEART STUDY

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Background: Both hypertension and diabetes mellitus (DM) increase risk for subclinical and clinical cardiovascular disease (CVD). The relative extents to which hypertension and DM induce subclinical CVD have not been examined, but recent Framingham data suggest that clinical CVD risk in DM is largely attributable to coexistent hypertension.

Methods: We examined subclinical arterial disease (carotid artery hypertrophy, atherosclerosis, stiffness) and incident CVD in a population-based cohort with high rates of DM and CVD.

Results: 2887 participants were divided into 4 groups: normal (n=799), hypertension alone (n=647), DM alone (n=494), and both hypertension and DM (n=947). In multivariable models adjusting for other CVD risk factors and creatinine, arterial hypertrophy and atherosclerosis were significantly greater in the two DM groups and not increased by coexistent hypertension. Although hypertension significantly altered arterial structure and function compared to normal, differences were eliminated by consideration of systolic pressure. Among 2441 participants without CV at baseline, events occurred in 10.1% of normals, 17.8% with hypertension alone, 25.5% with DM alone, and 29.3% with both. Rates were significantly higher in the 2 DM groups and not increased by coexistent hypertension. Adjusted hazards ratios were 1.69 (p<0.001) for hypertension alone, 3.16 (p<0.001) for DM alone, and 3.85 (p<0.001) for both (p<0.001 for trend).

Conclusions: Both hypertension and DM cause increased subclinical and clinical CVD. The impact of hypertension on CV is largely attributable to increased distending pressure. Higher rates of vascular hypertrophy, subclinical atherosclerosis and incident CV in DM are not attributable to coexistent hypertension in this population.
We have undertaken an individual participant data (IPD) meta-analysis of carotid-femoral pulse wave velocity (cf-PWV) with all-cause mortality, CHD, stroke and combined CVD events using data from 14 studies (2 unpublished). Unlike a previous report, which only used published data, we were able to undertake standardised analyses with and without adjustment for cardiovascular risk factors and test, a priori, for potential interactions between cf-PWV and age group, gender, diabetic or hypertensive status on the various outcomes. We calculated discrimination statistics for models with and without cf-PWV, specifically focussing on individuals at intermediate (25-75th percentile) risk of CVD after adjustment for conventional Framingham risk factors. Fourteen studies provided data on 16,358 subjects with 170 combined CVD events. We derived within study 2-scores of log transformed cf-PWV (pooled SD = 3.3 m/s). Risk of all outcomes was associated with increased cf-PWV (Table 1) and was linear across the range of cf-PWV values with no evidence of interaction except for age group (see Figure, p-value for trend = 0.0095). The additional benefit of measuring cf-PWV to reclassify intermediate risk individuals was assessed using the net reclassification index. 18.6% (p < 0.001) and 22.4% (p < 0.001) were appropriately reclassified into higher or lower quartiles of risk for CHD and stroke outcomes respectively. These findings highlight the added value of cf-PWV as an independent predictor, over and above existing risk factors, in intermediate risk groups and for younger subjects. Assessment of PWV should better identify high risk populations that may benefit from more aggressive risk factor management.

Table 1: Cox Proportional Hazards Models for cf-PWV as Predictor of an Outcome Event During the Follow-Up Period.

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Model 1*</th>
<th>p-value</th>
<th>Model 2*</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>All cause mortality</td>
<td>1.21(1.16, 1.27)</td>
<td>&lt;0.001</td>
<td>1.16(1.11, 1.23)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CHD</td>
<td>1.33(1.19, 1.48)</td>
<td>&lt;0.001</td>
<td>1.22(1.09, 1.36)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CVD</td>
<td>1.42(1.27, 1.59)</td>
<td>&lt;0.001</td>
<td>1.28(1.16, 1.41)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke</td>
<td>1.52(1.30, 1.78)</td>
<td>&lt;0.001</td>
<td>1.25(1.14, 1.39)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*Hazard ratio per 1 SD of log-transformed cf-PWV adjusted for age and sex. 
As model 1, further adjustment for Framingham risk factors (systolic blood pressure, cholesterol, HDL-cholesterol, smoking status, baseline diabetes and baseline hypertension, where available).

Objective: Chronic kidney disease (CKD) is characterized by high cardiovascular risk. Subclinical damage to large arteries has been largely described in CKD and is mostly characterized by an increase in arterial stiffness and an outward remodeling of the carotid artery. However, the predictive value of arterial remodeling and stiffening for cardiovascular events and mortality is still debated in pre-dialysed CKD (stage 2-5).

Methods: 180 patients (mean age 59.6 ± 14 years) with CKD (mean mGFR 32 mL/min/1.73m²) were included in this longitudinal study. Patients underwent a yearly check-up including arterial evaluation (carotid-femoral pulse wave velocity (SphygmoCor®), carotid thickness, diameter and stiffness (Art-Lab system®) and GFR measurement with the 51Cr-EDTA clearance.

Results: During an average follow-up of 49 ± 16 months, 36 fatal or nonfatal cardiovascular events occurred. In COX regression analyses, PWV was significantly associated with fatal and nonfatal cardiovascular events (risk ratio for 1 SD 1.46 [1.04-2.04], P = 0.02) independently of age, body mass index, proteinuria, measured glomerular filtration rate and mean blood pressure. By contrast carotid intima-media thickness and circumferential wall stress were not significantly associated with fatal and non fatal cardiovascular events.