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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 ≥ 5), 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 serum albumin, AAC and PWV remained independent predictors of outcome. AAC and PWV are independent predictors of all-cause mortality and nonfatal CV events in dialysis patients. The risk associated with an increased PWV is less pronounced at higher levels of calcification. Assessment of AAC using a lateral abdominal x-ray and PWV is feasible in a clinical setting and both may contribute to an accurate CV risk estimation in this heterogeneous 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 CVD 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 CVD is largely attributable to increased distending pressure. Higher rates of vascular hypertrophy, subclinical atherosclerosis and incident CVD in DM are not attributable to coexistent hypertension in this population.

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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