07.01: OUTWARD HYPERTROPHIC REMODELING AND INCREASED CAROTID ARTERY WALL STIFFNESS IN PATIENTS WITH RUPTURED INTRACRANIAL ANEURYSMS


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due to an increase in aortic stiffening (pulse wave velocity, $r = 0.77, p < 0.001$). Conclusions: Reflected wave can be followed travelling-back from the proximal aorta into the coronary arteries. These reflected waves augment coronary systolic blood flow. With increasing age the degree of augmentation of systolic coronary blood flow is increased.

Results: The numerical calculations yielded a DP of 11.7mmHg, which was in excellent agreement with the value of 10.5mmHg measured in vivo in the same patient (with pressure guide-wires) and with values measured in a silicon hydraulic bench model of the same geometry. A parameter study demonstrated a rapid increase in DP beyond 60% stenosis. In the post-stenotic dilatation zone, secondary flow patterns with recirculation were observed. Conclusion: These promising results demonstrate the feasibility and utility of patient-specific computer simulations in the diagnosis of individual patients, although further steps will be necessary to include pulsatile blood flow, distensible walls and patient-specific boundary conditions.

07.03 BETUIN-A IS INDEPENDENTLY ASSOCIATED WITH PROGRESSIVE AORTIC STIFFNESS IN PATIENTS WITH CHRONIC KIDNEY DISEASE
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Background: There is a disproportionate burden of vascular disease in patients with chronic kidney disease (CKD). Both aortic stiffness, as measured by carotid-femoral pulse wave velocity (C-F PWV), and deficiency in inhibitors of vascular calcification, such as Fetuin-A, have been implicated in the higher rates of cardiovascular mortality observed in this population. We sought to determine whether Fetuin-A concentration was inversely associated with progressive aortic stiffness.

Methods: 54 stable outpatients enrolled in a prospective cohort study of cardiovascular risk in CKD stages 3 and 4 underwent measurement of C-F PWV using Compilor under standardized conditions at baseline and 12 months. Baseline plasma Fetuin-A concentration was determined using the Biovendor ELISA kit.

Results: The population was aged 68.0±10.4 years, 80% male, 11% diabetic with a mean eGFR of 32.0±11.5. Baseline Fetuin-A did not correlate with patient age, eGFR, mean arterial blood pressure, albumin, calcium-phosphate product, parathyroid hormone or CRP. Baseline Fetuin-A was inversely correlated with the change in PWV over 1 year ($r = -0.52, p < 0.001$). After adjustment for change in mean arterial pressure between visits, age, eGFR and presence of diabetes the correlation was maintained ($r = -0.54, p < 0.001$). Using stepwise multiple linear regression with a model including age, change in eGFR, parathyroid hormone, CRP and diabetic status, Fetuin-A was the only independent predictor of change in aortic stiffness adjusted for change in MAP ($r$-coefficient -0.61, $p < 0.001$; $R^2$ total 0.36).

Conclusion: In a cohort of patients with CKD stages 3 and 4 there is an independent negative association between Fetuin-A and progressive aortic stiffness.