P167: ACUTE EXERCISE EFFECTS ON VASCULAR AND AUTONOMIC FUNCTION IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE

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To link to this article: https://doi.org/10.1016/j.artres.2018.10.220

Published online: 7 December 2019
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AORTIC PULSATILITY, AND NOT MEAN ARTERIAL PRESSURE, IS AN INDEPENDENT DETERMINANT OF LEFT MAIN CORONARY ARTERY DISEASE
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Introduction: Left Main Coronary Artery (LMCA) disease is prognostically the most important coronary lesion. LMCA differs from the other coronaries in having high elastin content. Aortic Pulsatility (AP) is an independent predictor of cardiovascular events in CAD. We hypothesized that pulsatile stress may be an independent determinant of disease in the LMCA.

Methods: This was a prospective cohort study in patients undergoing coronary angiography between the years 2011 and 2016 (n = 4633, 25% female) at King Abdul Aziz Cardiac Center, Riyadh, Saudi Arabia. We excluded patients with acute myocardial infarction, cardiogenic shock and significant valvular disease. Aortic systolic and diastolic blood pressures (BP) were measured in the ascending aorta. Mean Arterial Pressure (MAP) by direct integration of the BP curve and Pulse pressure (PP) as PP/MAP. CAD was defined as >50% stenosis in any major vessel.

Results: Six percent of the population had LMCA disease (mean age 60 ± 11 years, 25% female). LMCA disease was associated with higher PP (p < 0.001) compared with similar MAP (94 ± 16 vs. 94.5 ± 14, p = 0.92) in patients with non-LMCA disease. AP was significantly higher (0.72 ± 0.30) in LMCA disease compared with; 3-vd (0.63 ± 0.32); 2-vd (0.61 ± 0.28), 1-vd (0.58 ± 0.31) and non-obstructive CAD (0.52 ± 0.26) (p < 0.0001). In a stepwise regression model, MAP was an independent predictor of LMCA disease (R²=0.68, P<0.0001) even when adjusted for potential confounders, including MAP, age and gender.

Conclusions: LMCA disease is independently associated with high AP. Considering aortic pulsatile stress to be an independent cardiovascular prognosticator, stiffness of the LMCA may play an important role in plaque formation, hitherto ignored.

References

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Purpose: To examine the acute effect of maximal exercise effort on pulse wave velocity (PWV) and heart rate variability (HRV) in patients with CAD with a range of functional capacity levels, and the association between these parameters 1,2,3.

Methods: Thirty-six patients with CAD (62 ± 10 y) ranging in very-poor (5.22 ± 0.83METs; n = 18; VPFIT-CAD) to poor (6.50 ± 1.35METs; n = 18; PFIT-CAD) functional capacity, and 18 age-sex-matched healthy controls (8.53 ± 1.84METs; FFIT-CON) had their aortic and peripheral PWV, and HRV assessed prior to, and at 10 min and 30 min following a maximal cycle-ergometer test.

Results: Aortic- and peripheral-PWV did not differ between groups (p > 0.05) at baseline. Aortic-PWV was significantly increased at 10 min (0.63 – 0.98 m.s⁻¹) following exercise in all groups, but only remained so at 30 min in PFIT-CAD. Lower IMP-PWV decreased in VPFIT-CAD and FFIT-CAD to 10min (0.48; 0.51 m.s⁻¹) and remained so at 30 min (0.51; 0.45 m.s⁻¹), but not in PFIT-CAD. Still, no interaction effects were observed (p = 0.864). RMSSD was lower in PFIT-CAD compared to FFIT-CON (6.55, p = 0.009). RMSSD decreased at 10min following exercise in PFIT-CAD (5.26, p = 0.005) and FFIT-CAD (8.86, p < 0.001) but only remained so at 30min in PFIT-CAD (3.27, p = 0.47; p-interaction = 0.001). A significant correlation between changes in aortic-PWV and RMSSD assessed from prior to 10min recovery was observed in VPFIT-CAD (r = 0.44, p = 0.034).

Conclusions: Patients with CAD have similar arterial response to maximal exercise compared to their higher fit healthy peers. However, HRV following exercise is apparently compromised in CAD patients. The reduction in aortic PWV is parallel to the changes in HRV in patients with CAD with very-poor functional levels.