P1.50: IS AORTIC PULSE WAVE VELOCITY A STRONGER MARKER OF CARDIOVASCULAR RISK THAN CALCULATED RISK SCORES IN YOUNGER WOMEN?

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Fat mass index (kg/m²) of the Europe-wide 'Heartscore' initiative. BMI, waist & bio-impedance year CV risk score was estimated using the Joint British Guidelines, similar to 35.1-37.2 underwent anthropometric, biochemical and aPWV measures. Younger women are considered to have low cardiovascular (CV) risk at this age better than CV scores alone (which omit obesity indices). Predictive scoring systems aim to assess overall CV risk from calculated 10 year absolute CV risk.

Conclusion: Measurement of PWV may significantly change CV stratification in addition to echocardiography and to detection of albuminuria, but not after CUS; our results confirm that evaluation of different forms of TOD is not after CUS; our results confirm that evaluation of different forms of TOD is effective therapy for patients with (resistant) hypertension. However, the exact mechanism leading to blood pressure reduction is not fully elucidated.

Conclusion: In young women, even without overt diabetes or hypertension, adjusting for other factors, current adiposity and calculated CV risk were independently related to aPWV. This suggests aPWV reflects the 'load' of CV risk at this age better than CV scores alone (which omit obesity indices).

Conclusion: In this large animal model of arterial hemodynamics assessed in a large animal model, Cardiotrophin-1 (CT-1) is a cytokine belonging to the interleukin-6 superfamily that exhibits trophic and survival properties in a number of cell types. CT-1 expression has been recently been identified within the media of atherosclerotic arteries, but its role in the vessel remains unknown. The aim of this study is to characterize CT-1 actions and regulation in vascular smooth muscle cells (VSMC). Primary rat aorta VSMC were stimulated with CT-1 (10^-10^-M) for up to 48 hours, without and with antibiotics against CT-1 receptors. Moreover, the effects of aldosterone (10^-8-10^-6M) and angiotensin II (10^-8-10^-7M) on CT-1 expression were evaluated. Cell proliferation was determined by MTT assay. The expression of CT-1, collagen type I, and fibronectin was quantified by Western blot. Matrix metalloproteinases (MMPs) activities were assessed by gelatin and casein zymographies. A 48-hour treatment with CT-1 induced VSMC proliferation in a dose-dependent manner (p<0.01). A 24-hour incubation with CT-1 led to an increased expression of collagen type I (p<0.01) and fibronectin (p<0.05), with a parallel and dose-dependent increase in active MMP-2 (p<0.01), MMP-3 (p<0.05) and MMP-9 (p<0.01). All of these effects being reversed in the presence of antibiotics against CT-1 receptors. VSMC spontaneously expressed CT-1, both aldosterone and angiotensin II enhanced (p<0.01) CT-1 expression in these cells in a dose- and time-dependent manner. CT-1 induces proliferation and a secretory phenotype in VSMC. Upregulation of CT-1 expression by angiotensin II and aldosterone in VSMC suggests a mediator role for this cytokine in alterations of these cells caused by the RAAS in vascular diseases.