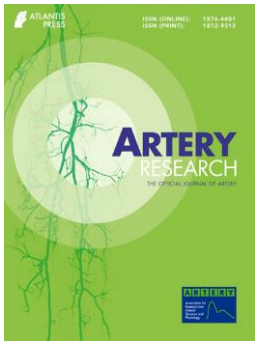

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

P4.08: AUGMENTATION INDEX AND AORTIC DISEASE IN MARFAN-LIKE SYNDROMES

K. Mortensen, J. Baulmann, M. Rybcynski, M. Reppel, H. Schunkert, Y. von Kodolitsch

To cite this article: K. Mortensen, J. Baulmann, M. Rybcynski, M. Reppel, H. Schunkert, Y. von Kodolitsch (2010) P4.08: AUGMENTATION INDEX AND AORTIC DISEASE IN MARFAN-LIKE SYNDROMES, Artery Research 4:4, 163–163, DOI: https://doi.org/10.1016/j.artres.2010.10.060

To link to this article: https://doi.org/10.1016/j.artres.2010.10.060

Published online: 21 December 2019

139 subjects (31 normotensives-NT, 38 hypertensives-HT, 24 diabetic normotensives-DMNT, and 46 diabetic hypertensives-DMHT) were enrolled. Applanation tonometry (Sphygmocor) was used to measure aortic (carotid to femoral) and peripheral (carotid to radial) pulse wave velocity (aPWV and pPWV respectively). Common carotid stiffness (CS) was determined using the real-time computerized echo-tracking system "Carotid Studio".

pPWV was similar in the four groups. aPWV significantly increased from NT (7.3±1.1m/s) to HT (8.2±1.4 m/s) and DMNT (8.3±1.3 m/s), reaching the highest value in the DMHT group (10.8±1.9 m/s). CS was similar in NT (6.1±0.8m/s) and DMNT (6.4±1.1 m/s), and significantly increased in HT and DMHT (6.7±1.2m/s, 7.4±1.2m/s).

In a model adjusted for age, gender and diabetes, the presence of hypertension carried a 5.6-fold (confidence interval-95% 1.9-16.5) increased risk of having aPWV above the median value of 8.5 m/s, and a 4.5-fold (1.7-11.9) increased risk of having CS (above median value of 6.6 m/s). The presence of diabetes carried a 7.2-fold (2.8-18.9) increased risk of having increased aPWV, while the analysis was not significant for carotid stiffness (OR 2.1, 0.9-4.7).

In conclusion, increased aPWV is present in both diabetic and hypertensive patients, and the combination of the two risk factors induces an even greater aortic stiffening. While hypertension is characterized by vascular stiffening at both the aortic and carotid level, carotid stiffening in diabetic patients appears to be mostly dependent on the concomitant presence of hypertension.

P4.08
AUGMENTATION INDEX AND AORTIC DISEASE IN MARFAN-LIKE SYNDROMES

K. Mortensen [1], J. Baulmann [1], M. Rybcynski [2], M. Reppel [1], H. Schunkert [1], Y. von Kodolitsch [2]
[1]*University of Luebeck, Luebeck, Germany*
[2]*University of Hamburg, Hamburg, Germany*

The augmentation index (AIx@HR75) and central pulse pressure (CPP) can be measured noninvasively with applanation tonometry (APT). In this observational study, we investigated the relationship between AIx@HR75, CPP and aortic disease in Marfan-like syndromes.

Methods: We performed APT in 78 consecutive patients in whom classic Marfan syndrome (MFS) had been excluded (46 men and 32 women aged 34 ±13 years). These patients comprised 9 persons with MFS-like habitus, 6 with a bicuspid aortic valve (BAV), 5 with MASS phenotype, 3 with vascular type of Ehlers–Danlos syndrome (EDS), 3 with familial thoracic aortic aneurysm, 2 with Loeys–Dietz syndrome (LDS), 1with mitral valve prolapse syndrome, 1 with familial ectopia lentis,and 48 persons with Marfan-like features but no defined syndrome. During 20 ± 18 months after APT, we observed progression of aortic diameters in 15 patients, and aortic surgery or aortic dissection in 3 individuals.

Results: All 11 patients with Marfan-like syndromes and progression of aortic disease exhibited AIx@HR75 ≥11%, including 8 individuals with aortic diameters ≤95th percentile of normal at baseline. Similarly, all 7 individuals without any defined syndrome but progression of aortic diameters exhibited AIx@HR75 >11%, including 6 individuals with aortic diameters ≤95th percentile at the time of APT. Aortic disease did not evolve at AIx@HR75 <11%. CPP is also related to disease progression.

Conclusions: Aortic disease evolution relates to AIx@HR75 and CPP in Marfanlike syndromes. Larger studies will be required to establish APT for prediction of aortic disease evolution in Marfan-like syndromes.

P4.09
THE INCREASED TRANSMURAL PRESSURE CHANGES A PATTERN OF DILATION REACTION OF MAGISTRAL ARTERIES

D. Matisone, V. Dzerve, I. Kukulis
Research Institute of Cardiology, University of Latvia, Riga, Latvia

Objective: In our previous investigations it was shown that dilatation of magistral arteries (MA) after arterial occlusion (AO) occurs only after the restoration of free blood flow and manifests as an increment of blood flow in the beginning of reactive hyperemia (RH). The aim of this study was to examine whether the increased transmural pressure ($P_{transm.}$) can modulate dilatation reaction of MA after AO.

Design and methods: The investigation was performed on 12 healthy subjects aged between 28-40 yrs. Dilation of MA was appraised from changes in blood flow (I) and haemodynamic resistance (R) in the forearm during RH in two different situations - after 7 min. AO and after 7 min. AO with previous venous congestion (VC). Forearm I was determined by venous occlusion plethysmography. R was calculated from data on I and mean arterial pressure. Systemic arterial pressure was determined auscultatory.

Results: VC before AO of 7 min. abolished the increment of blood flow during RH - maximal I and minimal R was observed just after restoration of free blood flow and normalisation rate of I and R was more rapid than that after 7 min. AO. Maximal I was greater after 7 min. AO with previous VC than that after 7 min. AO (42±1,4 and 33±1,6 ml/100cm^3/min., correspondingly) and minimal R was smaller after 7 min. AO with previous VC than that after 7 min. AO (2,25±0,1 and 2,7±0,2 PRU_{100}, correspondingly).

Conclusion: The increase of $P_{transm.}$ changes a patern of dilatation reaction of forearm magistral arteries.

P4.10
INFLAMMATION IS NOT RELATED TO ABDOMINAL AORTIC ANEURYSM DIAMETER

M. Elkhawad [1], J. R. Boyle [2], I. B. Wilkinson [1], J. H. F. Rudd [1]
[1]*University of Cambridge, Cambridge, United Kingdom*
[2]*Cambridge University NHS Hospitals, Cambridge, United Kingdom*

Background: Chronic inflammation is a hallmark characteristic in the pathophysiology of abdominal aortic aneurysms (AAA). In this study we sought to prospectively investigate whether inflammation was related to aortic diameter. Given that diameter has been shown to be the best predictor of AAA expansion rate, we hypothesised that larger aneurysms would have the greatest degree of inflammation.

Methods: 34 patients with AAA were imaged using fluoro-deoxyglucose positron emission tomography (FDG-PET) with CT co-registration. Uptake of FDG, which directly reflects metabolic activity and is shown to correlate with macrophage activity, was quantified as a tissue to background ratio (TBR). A TBR was calculated for the entire aorta and for the ascending, descending, abdominal and aneurysmal segments. TBR values were correlated with AAA diameter as measured on CT images. Inflammatory biomarkers, including hsCRP were also measured.

Results: The mean aneurysm diameter was 44mm (SD+/-9mm). There was no correlation between aortic diameter and TBR within the aneurymal sac ($r=0.021$, $p=0.93$). The AAA diameter also did not correlate with abdominal aortic TBR ($r=0.144$, $p=0.54$) or the total aortic TBR ($r=0.058$, $p=0.844$). There was a modest but statistically non-significant correlation between AAA diameter and hsCRP ($r=0.424$, $p= 0.12$).

Conclusions: There was no relationship between FDG uptake, a surrogate of inflammation, and AAA diameter. However, this may indicate that inflammation is a potential predictor of AAA growth rate independently of AAA diameter.

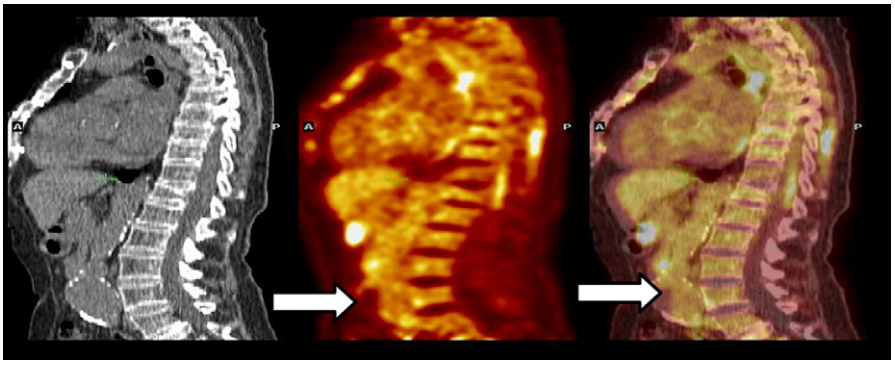

Figure 1 Image of CT, PET and co-registered image showing inflamed AAA.