Arterial wall stiffness in patients with abdominal aortic aneurysms and concomitant coronary artery disease

I. Ikonomidis[1], P. Rafouli-Stergiou[2], N. Katsiki[3], N. Kadoglou[2], S. Vlachos[2], J. Thymis[2], J. Parissis[2], K. Moulakakis[4], J. Kakisis[4]

[1]*National & Kapodistrian University of Athens, Athens, Greece;* [2]*Attikon University Hospital, 2nd Cardiology Department, Athens, Greece;* [3]*Ahepa General Hospital of Aristotle University, First Department of Internal Medicine, Division of Endocrinology and Metabolism, Diabetes Center, Me, Thessaloniki, Greece;* [4]*Attikon University Hospital, Department of Vascular Surgery, Medical School,National and Kapodistrian University of Athens,Greece, Athens, Greece*

Introduction: Pulse wave velocity (PWV) is a valid, clinically feasible marker of arterial stiffening, and a strong predictor of outcomes. In patients with abdominal aortic aneurysms (AAA) alterations in arterial wall composition and inflammation were proposed as possible contributors to increased arterial stiffness.

Purpose: The present study aimed to compare aortic elastic properties in patients with abdominal aortic aneurysms (AAA), with or without coronary artery disease (CAD), as well as healthy individuals.

Methods: A total of 130 patients with AAA, eligible for interventional repair, and 30 healthy individuals, comprising the control group (HC), were evaluated. Study groups did not differ in anthropometrical characteristics and CV risk factors. Presence of CAD was identified by coronary angiography. Aortic PWV (aPWV) was measured using the Arteriograph method.

Results: aPWV was found considerably higher in AAA patients compared to HC group (11.5 ± 2.9 vs. 7.3 ± 1.6 m/s, $p<0.001$), after adjustment for age, sex and mean arterial pressure (MAP). Interestingly, among patients with AAA, those with concomitant CAD (n=41) had higher aPWV than those without CAD (12.5 ± 2.9 vs. 11.0 ± 3.0 m/s, $p=0.03$), after adjustment for age, sex and MAP. In receiver-operator-curve (ROC) analysis, the area under the curve (AUC) of aPWV for the prediction of CAD presence in the AAA study group was 0.72 [95% confidence interval (CI): 0.55–0.84, $p=0.03$]. According to this, the best cut-off was a value of aPWV above 12.8 m/s (78% sensitivity and 73% specificity). This cut-off was identified as a significant predictor of CAD presence in the AAA study population according to univariate logistic regression analysis (OR=2.51, 95% CI: 1.79–5.19, $p=0.02$). After adjustment for age, sex, dyslipidemia, smoking and MAP in multivariate logistic regression analysis, the cut-off aPWV level remained independently associated with the presence of CAD (OR=1.64, 95% CI: 1.19–4.08, $p=0.03$).

Conclusion: The co-existence of CAD and AAA is characterized by a greater arterial stiffness. This finding supports a role of measuring arterial stiffness markers when evaluating AAA patients' cardiovascular risk and selecting endovascular stents with more favorable elastic properties. Further studies are needed to explore whether aPWV values could be used to as a screening tool to detect CAD or to guide treatment in AAA patients.