Aortic flow propagation velocity in the assessment of arterial stiffness

Arteryel sertliğin değerlendirmesinde aort akım yayılma hızı

Arterial stiffness is currently regarded as an independent predictor of cardiovascular morbidity and mortality. There is a close relationship between arterial stiffness and target organ damage in hypertensive patients who have been recently diagnosed as hypertensive and who are not on any medications but are instead treated with lifestyle modifications (1). In recent years, several noninvasive methods for the calculation of arterial stiffness have come to light, such as the ambulatory arterial stiffness index (AASI), the home arterial stiffness index (HASI), the central and peripheral augmentation index (CAIx and PAIx) or the pulse wave velocity (PWV), which despite the advent of other techniques remains the clinical gold standard in this area. Alterations in the vascular wall are mediated by the development of atherosclerosis and can be explained by the relationship between activity of the autonomic nervous system and inflammation (2, 3) in addition to the coexistence of other vascular risk factors.

In the article "Association of aortic flow propagation velocity with ankle-brachial index in patients with hypertension: an observational study", published in this issue of the Anatolian Journal of Cardiology, Güntekin et al. (4) evaluate a new method of measuring arterial stiffness, which has been previously shown to be a predictor of coronary artery disease (CAD) (5), and its correlation with peripheral arterial disease measured by the ankle-brachial index (ABI). The results were obtained in newly diagnosed and never treated patients with hypertension that were free from cardiovascular disease and some risk factors. Body mass index and systolic blood pressure were the cardiovascular risk factors which values were significantly higher in the hypertensive patients group than in the control group. With respect to subclinical organ damage, ABI was minor in the patients group than in the control one (1.08±0.07 and 1.14±0.07 respectively, p=0.0001) with no pathological values either group. The values of the measurements obtained by ultrasound were also significant between the two groups: deceleration time (DT, msec) 217.10±38.6 versus 184.02±32.2, p=0.0004; isovolumetric relaxation time (IVRT, msec) 105.70±18.1 versus 95.50±19.4, p=0.0098 and aortic flow propagation velocity (AVP, cm/sec) 54.97±9.3 versus 69.17±10.8, p=0.0006. The study confirms the existence of a relationship between aortic propagation velocity and the ABI, despite the absence of pathology in the latter. In addition, as the authors highlight at limitations section, future studies should be needed with a greater patient's sample.

Endothelial dysfunction is considered the first stage in the development of atherosclerosis. Thickening and stiffening of the arterial wall leads to increased arterial resistance, which may be reflected by a decrease in flow propagation velocity within the arterial lumen (6). Color M-mode propagation velocity measured along the origin of the descending thoracic aorta may reflect atherosclerosis when is decreased. AVP may be used in patients referred for noninvasive cardiovascular examination, to improve cardiovascular risk estimation, and for a better selection of high-risk candidates for additional exams and for the institution of preventive measures (5). In the same way, the value of exercise ECG is complemented by the color M-mode-derived AVP in the evaluation of CAD and in those patients with a positive exercise ECG test may prevent unnecessary invasive diagnostic approach for an intermediate likelihood of coronary ischemia (6). Given the above, this noninvasive technique is a not complicated method. Its availability enables it to be used in patients where it is necessary to stratify vascular risk and improve the selection of those who need to be made primary prevention. Also in middle risk patients and those candidates for more advanced studies in this line. Furthermore, future studies should be conducted to correlate the AVP with others non- vascular target organ damage and assess their contribution to the improvement in cardiovascular risk stratification.

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