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## STUDY OF ARTERIAL STIFFNESS PARAMETERS AND ENDOTHELIAL VASOMOTOR FUNCTION IN PATIENTS WITH JOINT HYPERMOBILITY SYNDROME

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**Introduction.** Arterial stiffness plays a central role in hemodynamic dysfunction characterized by excess pulsatility; specifically, it leads to heart failure, cerebrovascular disease, and chronic kidney disease [1]. Joint hypermobility syndrome (JHS) is based on genetic disorders of collagen synthesis, which develop tissue weakness and fragility, and can lead to various clinical consequences that resonate far beyond the musculoskeletal system. Detecting of arterial stiffness and other early indicators of cardiovascular disease is a reasonable first-level approach to medical management of people with heritable disorders of connective tissue who are at risk of cardiovascular events [2].

**Aim of the study.** The purpose of this research was to study of arterial stiffness parameters and endothelial vasomotor function in patients with JHS.

**Materials and methods.** Study population included 105 adults with JHS (90 women and 15 men aged 22 [21; 23] years) and 57 age-, gender-, height-, weight-, body mass index and blood pressure matched healthy controls. The Brighton criteria

was used to assess JHS. The Cardio-Ankle-Vascular Index (CAVI) is a marker of arterial stiffness from the origin of the aorta to the ankle and is independent of changes in blood pressure during measurement. CAVI was measured by the VaSeraVS-1500-N device. Pulse wave velocity is the velocity of propagation of the pulse wave along the arterial tree and is calculated by dividing the distance between two set points by the transit time needed for the wave to cover this distance and is increased by a reduction in the intrinsic elasticity of the arterial wall. Carotid-radial pulse wave velocity (PWVcr) was measured noninvasively by rheovasograph “Impecard-M”. The forearm blood flow (FBF) was measured during reactive hyperemia to test endothelium-dependent vasodilatation by rheovasograph “Impecard-M”. Statistical analysis was performed by Statistica 10.0.

**Results and discussion.** PWVcr was higher in patients with JHS to compare with healthy controls (7,4 [6,1; 8,4] vs 4,3 [3,9; 6,9],  $p < 0.001$ , respectively). Abnormal PWVcr value more than 7.0 m/s was in 60 patients with JHS and in 14 persons of control group ( $FET = < 0.0001$ ). The CAVI (6,0 [5,6; 6,4] for JHS vs 5,9 [5,6; 6,3],  $p = 0,058$  for healthy controls) and FBF (18,2 [5,6; 31,4] vs 24,8 [12,0; 36,7],  $p = 0,13$  respectively) were comparable. However, abnormal CAVI values (more than 6.6 (for women) and 6.8 (for men) were obtained among 21 patients with JHS and 4 healthy persons ( $FET = 0.039$ ), abnormal values of FBF (less than 12%) were obtained among 44 patients with JHS and 13 controls ( $FET = 0.01$ ).

**Conclusion.** Patients with JHS showed abnormal endothelial function and decreased arterial elasticity in comparison with healthy controls. Further investigations are needed in order to assess the prognostic value of these parameters for cardiovascular outcomes in patients with JHS.

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