Neuro-Ophthalmology

CHANGES OF SCLERAL LAMINA CRIBROSA THICKNESS AS A RISK FACTOR IN THE DEVELOPMENT OF RETINAL NEURODEGENERATION IN DIABETES MELLITUS

Maryna Karliychuk[1], Pavlo Bezditko[2], Serhii Pinchuk[3]
[1]*Department of Ophthalmology, Bukovinian State Medical University, Ukraine*
[2]*Department of Ophthalmology, Kharkiv National Medical University, Ukraine*
[3]*Department of Cataract and Glaucoma Surgery, Center of eye microsurgery «Vash Zir», Ukraine*

PURPOSE: to analyze the state of scleral lamina cribrosa (LC) in type 2 diabetes mellitus (DM) and the corresponding morphometric changes in retinal ganglion cell complex (GCC).

METHODS. 575 patients (1150 eyes) with type 2 DM and 50 healthy persons (50 eyes) aged 55,9±7,8 years were examined. LC thickness was measured with SD optical coherent tomography using LC_Thickness_programm.m and main_low_noise_filters_programm.m, based on adaptive compensation algorithm for eliminating the high-level noise in deep layers of optic nerve and improving the visualization of LC posterior border. 5 indicators of GCC state were analyzed: average thickness, average thickness in superior and inferior segments, focal loss volume (FLV), global loss volume (GLV).

RESULTS. Analysis of LC thickness made it possible to distinguish the following groups: in 1st group (78.9% of eyes of diabetic patients) a mild thickening of LC (700 μm) was observed; in 17.6% (2nd group) a moderate thickening (700-900 μm), and in 3.8% (3rd group) – significant thickening (900 μm) was observed. Average GLV in 2nd group was 2.9 times higher, in 3rd group – 5.3 times higher than in healthy individuals (3,51±2,73 %) (p0,001). FLV of retinal GCC in 2nd and 3rd groups was 13.2 and 16.4 times respectively higher than that of healthy individuals (p0,001). The highest index of FLV was observed in 3rd group (5.9 times higher than that of 1st group, p0,001).

CONCLUSIONS. Revealed morphometric changes of GCC depend on state of scleral LC, changes in thickness of which can be considered as pathogenetic factor in development of retinal neurodegeneration in DM.