
DIABETIC RETINAL RETINAL NEURODEGENERATION

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Abstract

According to the International Diabetes Federation, there are 537 million patients with diabetes in the world [1]. In patients suffering from diabetes mellitus (DM), the risk of developing complications of the underlying disease is quite high, including the risk of developing diabetic retinopathy (DR). Diabetic retinopathy is not only a common complication of diabetes, but it is also recognized as the most severe, as it often leads to irreversible vision loss. In 2021, there were 103 million people worldwide suffering from some form of diabetic retinopathy, representing 22.3% of the total number of patients with diabetes (Teo et al., 2021).

Modern research methods have made it possible to consider DR not only as a vascular complication of diabetes, but also as a neurodegenerative process. At the same time, modern research methods make it possible to register retinal neurodegeneration much earlier than it is possible to detect such vascular signs of DR as microaneurysms and retinal hemorrhages [2, 3]. The most common signs of neurodegeneration detected by optical coherence tomography (OCT) were the death of ganglion cells and a decrease in retinal thickness [4, 5]. Also, according to electroretinography (ERG), signs of glial activation were detected.

Purpose of the study:

To analyze morphofunctional changes in the organ of vision in patients with retinal neurodegeneration and absence of signs of DR.

Material and research methods

This paper presents the results of an examination of 11 patients with diabetes and signs of retinal neurodegeneration. All patients underwent routine ophthalmological examinations, including visometry with keratorefractometry and determination of best-corrected visual acuity, non-contact tonometry, static perimetry on a computer autoperimeter APS-6000BER, biomicroscopy of the



anterior segment of the eye and biomicrophthalmoscopy with high-diopter lenses (60 and 78D). Optical coherence tomography was performed on a DRI OCT Triton tomograph, which features both multimodal fundus visualization and tomography using Swept technology Source. In addition, patients underwent routine laboratory tests: blood glycemic levels, biochemical blood tests, lipid profiles, etc. In addition, consultations were held with related specialists: endocrinologist, neurologist, otolaryngologist, nephrologist, etc. Magnetic resonance imaging of the brain was performed. The patient gave voluntary informed consent to participate in the examination and publication of the results of her examination in the specialized press.

Research results

We examined both eyes of 11 patients with type 2 diabetes aged from 42 to 57 years. The average age of the patients was 51.4 ± 6.3 years. The duration of diabetes ranged from 2 to 12 years, with an average of 6.4 ± 4.1 years. For the treatment of type 2 diabetes, patients received oral hypoglycemic drugs, in no case. Upon admission, patients presented the following complaints: 3 patients (13.6%) noted decreased vision at night, 4 patients (18.2%) noted glare, and 1 patient (4.5%) periodically noted color distortions; in other cases, patients did not complain (13.6%). Indicators of maximum correctable visual acuity averaged 0.94 ± 0.03 . Vision correction was carried out taking into account the automatic refractometry indicator. In 7 patients (63.6%) emmetropia was recorded in both eyes, in 3 patients (27.3%) mild hyperopia was recorded, and in 1 patient (9.1%) mild myopia was recorded. Intraocular pressure in all cases did not exceed normal values and averaged 16.3 ± 0.4 mm Hg. Art.

No pathological changes in the anterior segment (cornea, conjunctiva, anterior chamber) were found in all observations. In 8 patients (72.7%) minor lens opacities were detected; in the remaining patients, no lens opacities were detected. The opacities were probably due to a long history of type 2 diabetes and poor glycemic control. At the same time, according to ultrasound biomicroscopy, lens thickening sufficient to displace the iridolenticular diaphragm was not recorded in any patient.

During ophthalmobiomicroscopy, microaneurysms and single hemorrhages were found in the fundus of the eye in 4 patients (36.4%), and the vessels in the macular area were corkscrew-shaped. Nonproliferative diabetic retinopathy was reported

in these patients. In the remaining 7 patients (63.6%), no signs of diabetic retinopathy were recorded. A diagnosis was made: retinal angiopathy or background retinopathy. The most significant changes were recorded on OCT. The total thickness map of the inner and outer layers was compiled based on the Retina protocol Map when studying nine zones: fovea (diameter 1 mm), parafovea (diameter 3 mm), perifovea (diameter 5 mm). The para- and perifovea were divided into four quadrants: superior, inferior, nasal and temporal. Also, using the GCC protocol, the thickness of the retinal ganglion cell complex and the volume of their focal and global losses were determined.

According to OCT results, in both eyes of patients with and without signs of DR, thinning of the retina in the macular region of varying severity was recorded. Thinning affected 3 to 8 of 9 Macular zones Map. The average retinal thickness in this zone was $232.6 \pm 34.1 \mu\text{m}$ in both eyes, the total volume of the macular zone averaged $6.91 \pm 0.6 \text{ mm}^3$ in both eyes. The most significant thinning in both eyes was recorded in the retinal nerve fiber layer (RNFL) equally in the inferior and superior segments. And in the ganglion cell layer (GCC), thinning was more pronounced in the lower segment. According to laboratory tests, only glycemic levels were elevated in our patient; the remaining indicators remained within the age-related norm. According to MRI, no pathological changes in the brain were detected.

Conclusion. The signs of retinal neurodegeneration identified in patients with type 2 diabetes, namely its thinning in the macular region according to OCT data, were recorded in both patients with DR and in patients without signs of DR. This suggests that retinal neurodegeneration precedes the signs of DR, but also occurs in parallel with the appearance of DR.

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