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DIABETIC RETINAL NEURODEGENERATION: CASE STUDY

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Abstract

According to the International Diabetes Federation, there are 537 million people with diabetes in the world [1]. Patients with diabetes mellitus (DM) have a high risk of developing complications of the underlying disease, including the risk of developing diabetic retinopathy (DR). Diabetic retinopathy is not only a common complication of diabetes, but is also recognized as the most severe, since it often leads to irreversible vision loss. In 2021, there were 103 million people worldwide suffering from some form of diabetic retinopathy, which is 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]. Electroretinography (ERG) also showed signs of glial activation.

The aim of the study was to analyze morphofunctional changes in the visual organ in a patient with retinal neurodegeneration and the absence of signs of DR.

Material and methods of research.

This paper presents the results of the examination of two patients with diabetes mellitus and signs of retinal neurodegeneration. Both patients underwent routine ophthalmological examinations, including visometry with keratorefractometry and determination of maximally corrected visual acuity, non-contact tonometry, static perimetry on the APS-6000BER computer autoperimeter, biomicroscopy of the anterior segment of the eye and Biomicroophthalmoscopy with high-diopter lenses (60 and 78D). Optical coherence tomography was performed on the DRI OCT Triton tomograph, which presents both multimodal fundus imaging and Swept Source tomography. In addition, patients underwent routine laboratory tests: blood glycemia levels, biochemical blood tests, lipid profile indicators, etc. In addition,

consultations were held with related specialists: endocrinologist, neuropathologist, otorhinolaryngologist, nephrologist, etc. The patient gave her voluntary informed consent to participate in the examination and publish the results of her examination in the specialized press.

Results

Case No. 1. A young girl G., born in 2004 (19 years old) with a diagnosis of type 1 diabetes mellitus, with a 15-year experience of the disease, applied to the Consultative Polyclinic of the RSPMCE named after Academician E.Kh. Turakulov. The patient did not complain about the eye organs. Visometry showed a slight decrease in visual acuity to 0.9 in the left eye, which could not be corrected. In the paired right eye, visual acuity remained high = 1.0. Refractometry values sph +0.25 on both Eyes. No pathological changes in the anterior segment (cornea, conjunctiva, anterior chamber) were found in both eyes. Slight clouding of the lens was found in both eyes of the patient, despite the patient's youth. The opacities were probably due to the long-term course of type 1 diabetes and poor glycemic control. At the same time, according to the ultrasound biomicroscopy, there was no thickening of the lens sufficient to displace the iridochorlens diaphragm. As a result, the intraocular pressure in both eyes did not exceed the norm and amounted to 16 and 17 mmHg. on the Nidek pneumotonometer. Ophthalmic biomicroscopy revealed microaneurysms, single hemorrhages on the fundus, and corkscrew-like convolutions in the macular area. The most significant changes were recorded on the OCT (Figs. 1 and 2). The map of the total thickness of the inner and outer layers was compiled on the basis of the Retina Map protocol in the study of nine zones: fovea (diameter 1 mm), parafovea (diameter 3 mm), perifovea (diameter 5 mm). The para- and perifovea were divided into four quadrants: upper, lower, nasal, and temporal. The GCC protocol was also used to determine the thickness of the retinal ganglion cell complex, the volume of their focal and global losses.

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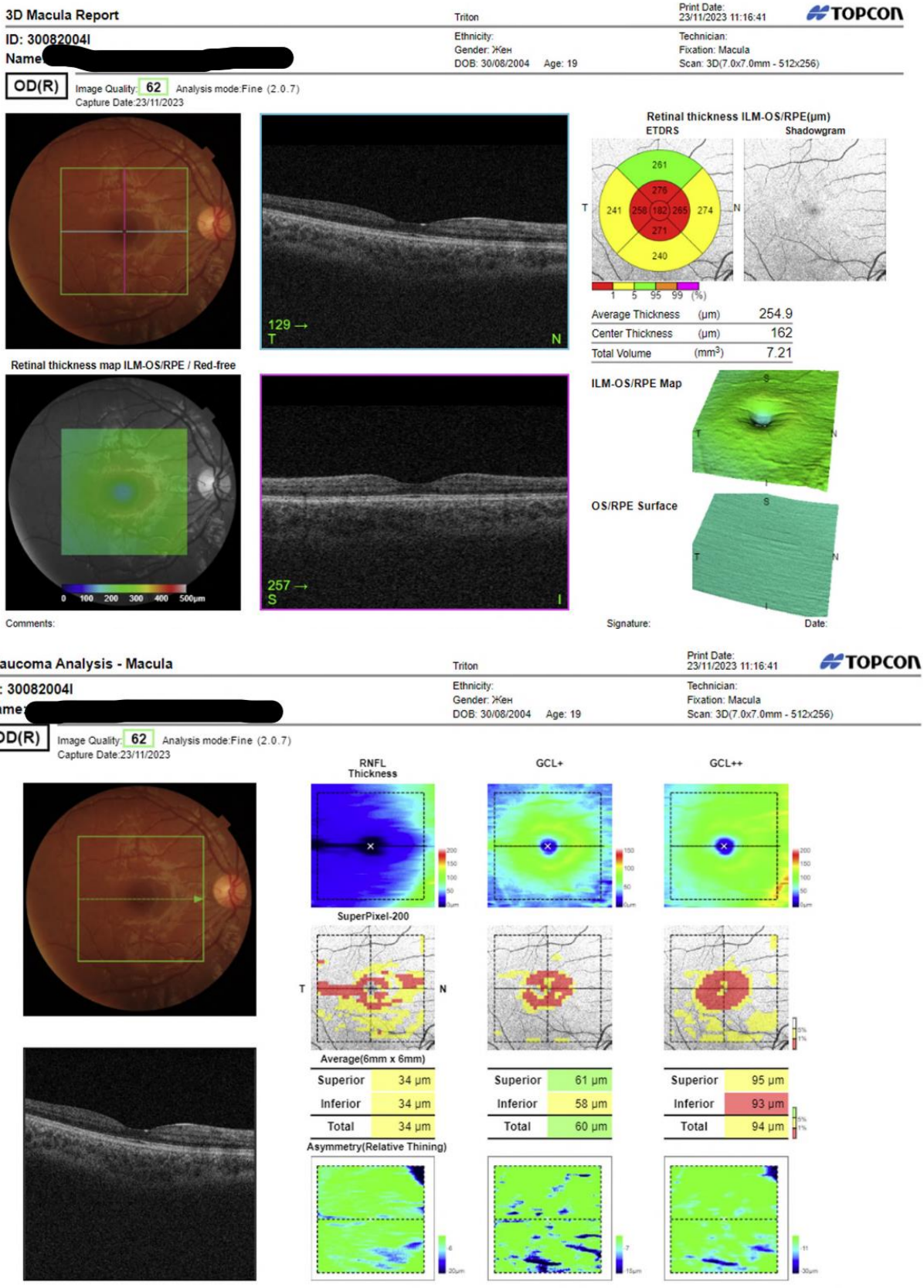

Rice. 1. OCT data of the right eye of patient G.

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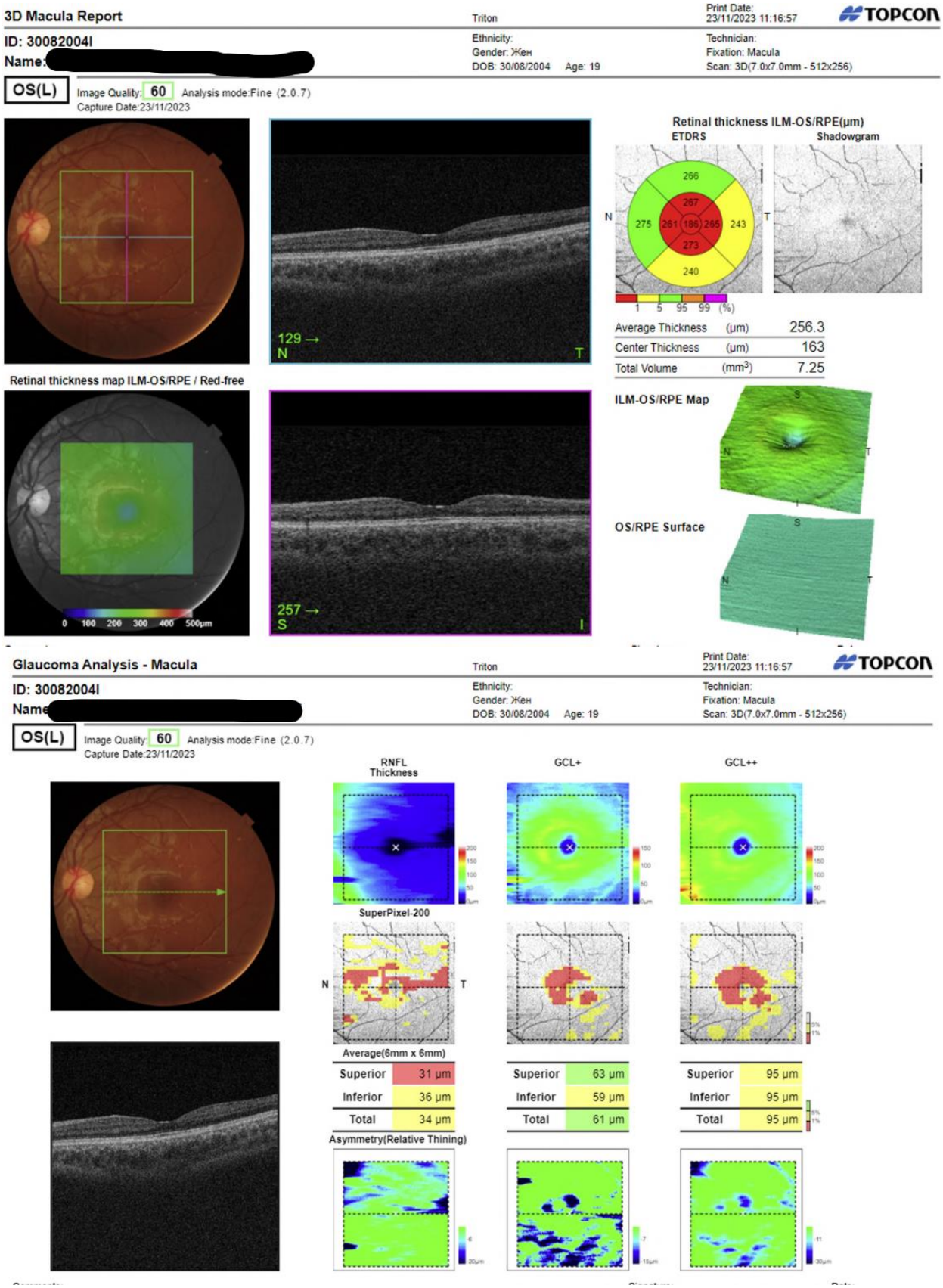

Rice. 1. OCT data of the left eye of patient G.

According to the results of OCT in both eyes of patient G., retinal thinning was registered in the macular area in the right eye in 8 out of 9 zones, and in the left eye in 7 out of 9 areas of the Macular Map. The mean retinal thickness in this area was 254.9 μm in the right eye and 256.3 μm in the left eye, the total volume of the macular zone was 7.21 mm3 in the right eye and 7.25 mm3 in the left eye. The most significant thinning in both eyes was recorded in the retinal nerve fiber layer (RNFL) is the same in the lower and upper segments. And in the ganglion cell layer (GCC), thinning was more pronounced in the lower segment. According to the laboratory tests, only the levels of glycemia were elevated in our patient, the remaining indicators remained within the age norm. According to the MRI data, no pathological changes in the brain were detected.

Conclusion

Structural changes in the retina detected in our patient according to OCT data may indicate neurodegenerative thinning of the retina due to DM exposure. The results of OCT indicate a significant increase in the focal loss of ganglion cells in the observed patient, which coincides with the data obtained by other authors and confirms the hypothesis that in DM, the processes of retinal neurodegeneration begin with apoptosis of ganglion cells [6, 7].

References

1. International Diabetes Federation. IDF Diabetes Atlas, 10th edn. Brussels, Belgium: International Diabetes Federation, 2021.
2. Kulybysheva V.S., Ronzina I.A., Gamidov A.A. Neurodegenerative changes in the retina in patients with diabetes mellitus. Bulletin of Ophthalmology.2019; 5:31–7.
3. Shawky SS, Elagouz MH, Ismail AM, Elhawwary AM. Macular thikness in healthy controls and diabetics without diabetic macular edema. Egypt Retina J. 2018; 5:1–5.
4. Kadlubowska J, Malaguarnera L, Waz P, Zorena K. Neurodegeneration and neuroinflammation in diabetic retinopathy: potential approaches to delay neuronal loss. Curr Neuropharmacol. 2016; 14(8):831–9.
5. Simo R, Stitt AW, Gardner TW. Neurodegeneration in diabetic retinopathy: does it really matter? Diabetologia. 2018; 61(9):1902–12.
6. Srinivasan S, Dehghani C, Pritchard N. et al. Corneal and retinal neuronal degeneration in early stages of diabetic retinopathy. Investig Ophthalmol Vis Sci. 2017; 58:6365–73.
7. Hegazy AI, Zedan RH, Macky TA. Et al. Retinal ganglion cell complex changes using spectral domain optical coherence tomography in diabetic patients without retinopathy. Int J Ophthalmol. 2017; 10(3):427–33.