

<https://doi.org/10.31288/oftalmolzh202454954>

Edema and features of optic nerve architecture in inflammatory and ischemic neuropathies. Clinical cases

Moyseyenko N. M. 

Ivano-Frankivsk National Medical University

Ivano-Frankivsk (Ukraine)

Purpose. To describe clinical cases of optic nerve edema in inflammatory and ischemic neuropathies and features of optic disc architecture revealed by optical coherence tomography.

Methods. 4 patients were examined. Two patients were diagnosed with optic neuritis, and the other two with ischemic neuropathy. Ophthalmoscopically, signs of optic disc edema were found in all patients. Visometry, ophthalmoscopy, optical coherence tomography (OCT), and magnetic resonance imaging (MRI) were used.

Result. Changes in the architecture of the optic nerve head were detected in cases of optic nerve edema in inflammatory (2 patients) and ischemic neuropathy (2 patients). In all examined patients, according to the conducted study, the results of OCT showed that optic nerve edema, except for case № 4, does not extend to the temporal segment of the head. In cases of inflammatory damage, cases 1 and 2, optic nerve atrophy was observed at later stages during repeated examinations. A light crescent zone is observed in the peripapillary area, which separates the temporal border of the optic nerve head (sectoral atrophy).

Conclusion. Optic nerve edema in inflammatory and ischemic neuropathies is characterized by an increase in the thickness of the nerve fiber layers and a change in the configuration of the disk, which later transforms into segmental atrophy. The changes in the architecture of the optic disc were found to be the basis of diagnosis, monitoring, and evaluation of the effectiveness of treatment in acute optic neuropathies.

Key words:

optic nerve edema, optic disc, optic nerve, inflammatory neuropathy, ischemic neuropathy, optic neuritis, OCT

Introduction

Optic nerve edema is a symptom that may develop in diseases of different etiologies. In cases of optic nerve inflammation of a demyelinating nature, such as Devic's disease or neuromyelitis optica, as well as in infectious neuritis, optic nerve edema occurs within the first few hours after injury and progresses rapidly [1]. Optic nerve edema, in ischemic optic neuropathy, facilitates the spread of the affected area due to secondary damage factors [2]. Vascular embolization in the retrobulbar part of the optic nerve leads to disc edema. Optic disc edema due to mechanical factors causes compartment syndrome [3]. At the same time, there is an increased leakage of interstitial fluid [4, 5]. As a result, cystic edema of the inner retinal layers develops within the prelaminar zone [6]. These factors collectively lead to the development of retrograde transsynaptic degeneration, ganglion cell apoptosis, and axon atrophy.

The mechanisms of the development of optic nerve atrophy under the influence of edema [7], as well as the edema in existing atrophy conditions, require more thorough study, as they are fundamental to the differential diagnosis of forms of acute neuropathies [8].

Current diagnostic techniques provide an increasing amount of data on the pathogenesis of optic nerve edema and thus contribute to the development of more effective treatment modalities.

The purpose: to describe clinical cases of optic nerve edema in inflammatory and ischemic neuropathies and features of optic disc architecture revealed by optical coherence tomography.

Methods. 4 patients (8 eyes) were examined. Two patients were diagnosed with optic neuritis in both eyes. Ischemic neuropathy of the left eye was diagnosed and is shown in example 3; ischemic neuropathy of both eyes is shown in example 4. Ophthalmoscopically, all patients showed signs of optic disc edema on the affected side.

Consent was obtained for the processing of personal data and use for scientific research. Declaration protocol of compliance with ethical standards during the study № 125/22 dated 24.03.2022.

Visometry, ophthalmoscopy, optical coherence tomography (OCT), and magnetic resonance imaging (MRI) were used.

The examination was conducted at the Department of Ophthalmology of Ivano-Frankivsk National Medical University and at the Ophthalmology Center "LUX VISION."

Results. Example 1. Patient K, 24 years old. The medical history began in December 2022, when she first visited an ophthalmologist at the polyclinic at her place of residence. At that time, the complaints were of a spot in front of her eyes. Visual acuity with correction of both eyes was determined to be reduced to 0.7. Hyperemia of the optic disc and blurred margins were observed in the fundus of the eye. Excavation of both eyes was performed (Fig. 1).

The second visit was at the Ophthalmology Center "LUX VISION" in March 2023 because of reduced vision in both eyes to 0.2. Ophthalmoscopy revealed monotonous paleness of the optic disc of the right eye and crescent-shaped paleness in the peripapillary area of the temporal segment (sectoral atrophy) of the left eye. Computer perimetry showed multiple scotomas of varying intensity in the central zone 30° from the fixation point.

Preliminary diagnosis: bilateral optic neuritis complicated by sectoral atrophy of the optic nerve of the left eye.

The patient was referred for MRI of the brain, orbits, and spinal cord with contrast. Accumulation of contrast was detected in the orbital part of the optic nerve sheaths and several paraventricular foci. The neurologist confirmed the demyelinating nature of the foci found in the brain. An analysis for Aquaporin-4 was conducted. The result was positive.

Clinical diagnosis: bilateral optic neuritis complicated by sectoral optic nerve atrophy in both eyes. Opticomyelitis (Devic's disease).

The OCT results (2022) of the patient are presented in Fig. 2. A bilateral increase in the thickness of the nerve fiber layer of all sectors (white) except for the temporal sector (green) was detected. The configuration of the optic nerve head is V/W-shaped.

Example 2. Patient P., 42 years old. The patient's medical history began in December 2021, when he first visited an ophthalmologist at the polyclinic at his place of residence. At that time, complaints were of worsening eyesight of the right eye: visual acuity with correction for both eyes reduced to 0.7. The fundus of the right eye (Fig. 3) in 2021: the optic disc was waxy, with blurred nasal borders that correspond to edema. The optic disc of the left eye was normal, but venous engorgement was observed.

The second consultation was at the Ophthalmology Center "LUX VISION" in May 2023. The patient had a decrease in vision in the right eye to 0.4 and in the left eye to 0.6 with correction. Ophthalmoscopy of the right eye showed total paleness of the optic disc with clear borders, likely corresponding to optic nerve atrophy. A light crescent-shaped zone was observed in the peripapillary area. The optic disc of the left eye was waxy with a blurred lower border and a reduction in the caliber of the lower vascular

bundle. Computer perimetry in 2023 showed multiple scotomas of varying intensity in the central 30° zone from the fixation point, more expressed in the right eye.

Preliminary diagnosis in 2023: optic neuritis of the right eye complicated by partial optic nerve atrophy.

Additional investigations included MRI of the brain and orbits with contrast. It showed signs of sinusitis and ethmoiditis (effusion, mucosal thickening, polyp and septal deviation). The patient had ENT consultation.

Clinical diagnosis: optic neuritis of both eyes associated with paranasal sinusitis (ethmoiditis) complicated by partial optic nerve atrophy.

The OCT results of the right eye in 2023 are in Fig. 4. An increase in the thickness of the nerve fiber layer in the upper-nasal segment was detected on the affected side. A yellow sector with reduced thickness of the nerve fiber layer was up to 2% compared to the age norm was observed in the temporal segment. The configuration of the optic nerve head was S-shaped.

Example 3. Patient, 37 years old. She had worsening eyesight of the left eye upon waking in the morning, which was accompanied by a headache. The fundus of the left eye (Fig. 5): the optic disc was waxy with blurred nasal borders corresponding to edema. Retinal paleness along the lower vascular bundle was observed. Perimetry showed a lower arcuate scotoma in the central 30° zone from the fixation point in the left eye, while the right eye remained within normal limits.

Preliminary diagnosis: non-arteritic anterior ischemic optic neuropathy of the left eye.

Additional examinations included MRI of the brain, which showed no pathological changes. Blood tests showed dyslipidemia and hypercoagulation. Electrocardiogram indicated arrhythmia. Nighttime hypotension was detected through 24-hour blood pressure monitoring.

Consultations with a neurologist and cardiologist resulted in a concurrent diagnosis of dyscirculatory encephalopathy and ischemic heart disease. To exclude arteritic ischemic optic neuropathy, an ultrasound of the temporal artery and a complete blood count with erythrocyte sedimentation rate assessment were performed. The results were normal.

Clinical diagnosis: non-arteritic anterior ischemic optic neuropathy of the left eye.

The OCT results of the patient are in Fig. 6. An increase in the thickness of the nerve fiber layer of all sectors except the temporal sector was detected on the affected side. The configuration of the optic nerve head is V/W-shaped.

Example 4. Patient B., 52 years old, complains of worsening eyesight in the left eye, observed for 1 month. The patient associates the onset of complaints with stress and excessive physical exertion (in the gym). Visual acuity with correction was 1.0 and 0.6 for the right and left eyes, respectively. Perimetry showed a central scotoma in the central 30° zone from the fixation point in the left eye, while the right eye remained within normal limits.

Fundus examination of both eyes: the optic disc was waxy with a blurred lower-nasal border and partial filling

of physiological excavation. Angiosclerosis was observed in both eyes (Fig. 7).

Preliminary diagnosis: non-arteritic ischemic optic neuropathy of both eyes.

Additional examinations: MRI of the brain showed no pathological changes. Blood tests indicated dyslipidemia and hypercoagulation. Electrocardiogram revealed arrhythmia. Neurologist and cardiologist consultations were needed. Associated diagnosis: atherosclerosis. An ultrasound of the temporal artery and a complete blood count with erythrocyte sedimentation rate assessment were performed to exclude arteritic ischemic optic neuropathy. The results were normal.

The OCT results of the patient are in Fig. 8. An increase in the thickness of the nerve fiber layer in the temporal segment was detected in the right eye. The configuration of the optic nerve head is S-shaped. A bilateral decrease in the thickness of the nerve fiber layer in the lower segment (yellow and red sectors) by 2-5% was observed.

Discussion. The study revealed changes in the architecture of the optic nerve head in cases of optic disc edema due to inflammatory (2 patients) and ischemic neuropathy (2 patients). An increase in the thickness of the nerve fiber layer was observed in all segments except the temporal one.

According to other authors, the introduction of OCT into clinical practice has allowed for a more precise assessment of structural changes in the optic nerve, and in some cases, it has enabled the study of pathogenesis in clinical settings without resorting to experimentation. In particular, in cases of acute neuropathies (inflammatory and ischemic), OCT diagnostics provide a better understanding of the role of primary and secondary damages, as well as the balance between neurodegenerative and neuroprotective processes. Therefore, OCT indicators are increasingly used as biomarkers for inflammation [9] and ischemia [10].

According to the conducted study, in all examined patients, the OCT results showed that optic disc edema did not extend to the temporal segment of the optic nerve head, except in case № 4.

In cases of inflammatory damage, such as in examples 1 and 2, optic nerve atrophy was observed in the later stages; follow-up examinations showed a light crescent-shaped zone in the peripapillary area, separating the temporal border of the optic nerve head (sectoral atrophy). Several authors explain this phenomenon, observed in papillitis associated with chorioretinitis, as a feature of microcirculation organization in the temporal zone [11].

Thus, understanding the structural changes of the optic nerve head due to edema caused by inflammation and ischemia through more detailed studies will help explain the pathogenic mechanisms of secondary damage. This knowledge can serve as a basis for developing diagnostic and monitoring criteria for acute optic neuropathies.

To conclude, optic nerve edema in inflammatory and ischemic neuropathies is characterized by an increase in the thickness of the nerve fiber layer and changes in the

configuration of the disc, which eventually transforms into segmental atrophy. The detected changes in the architecture of the optic nerve disc can serve as a basis for diagnosis and monitoring, as well as for assessing the effectiveness of treatment in acute optic neuropathies.

References

1. Chan JW. Optic neuritis in multiple sclerosis. *Ocul Immunol Inflamm.* 2002;10:161-86.
2. Berry S, Lin WV, Sadaka A, Lee AG. Nonarteritic anterior ischemic optic neuropathy: cause, effect, and management. *Eye Brain.* 2017 Sep 27;9:23-28.
3. Beck RW, Servais GE, Hayreh SS. Anterior ischemic optic neuropathy. IX. Cup-to-disc ratio and its role in pathogenesis. *Ophthalmology.* Nov 1987;94(11):1503-1508.
4. Song D, Leng B, Gu Y, Zhu W, Xu B, Chen X, Zhou L. Clinical Analysis of 50 Cases of BAVM Embolization with Onyx, a Novel Liquid Embolic Agent. *Interv Neuroradiol.* 2005 Oct 5;11(Suppl 1):179-84.
5. Bioussé V, Newman N. Retinal and optic nerve ischemia. *Continuum (Minneapolis, Minn).* 2014 Aug;20(4 Neuro-ophthalmology):838-56.
6. Lujan BJ, Horton JC. Microcysts in the inner nuclear layer from optic atrophy are caused by retrograde trans-synaptic degeneration combined with vitreous traction on the retinal surface. *Brain.* 2013 Nov;136(Pt 11):e260.
7. Rodríguez Villanueva J, Martín Esteban J, Rodríguez Villanueva LJ. Retinal Cell Protection in Ocular Excitotoxicity Diseases. Possible Alternatives Offered by Microparticulate Drug Delivery Systems and Future Prospects. *Pharmaceutics.* 2020 Jan 24;12(2):94.
8. Margolin E. The swollen optic nerve: an approach to diagnosis and management. *Pract Neurol.* 2019 Aug;19(4):302-309.
9. Zhou J, Song S, Zhang Y, Jin K, Ye J. OCT-Based Biomarkers are Associated with Systemic Inflammation in Patients with Treatment-Naïve Diabetic Macular Edema. *Ophthalmol Ther.* 2022 Dec;11(6):2153-2167.
10. Sun CB, Zhou X, Jiang H, Zhou H. Editorial: Biomarkers in the diagnosis, prognosis, and prediction of autoimmune and hereditary optic neuropathies. *Front Neurol.* 2023 Oct 24;14:1304227.
11. Talisa E, Bonini Filho MA, Adhi M, Duker JS. Retinal and choroidal vasculature in birdshot chorioretinopathy analyzed using spectral domain optical coherence tomography angiography. *Retina.* 2015;35 (11):2392-2399.

Disclosures

Received: 13.08.2024

Accepted: 25.09.2024

Corresponding author: Nataliya Mykhailivna Moyseyenko – MD, associate professor, Department of Ophthalmology, Ivano-Frankivsk National Medical University, E-mail: natalymoyseenko@gmail.com

Disclaimer: the opinions expressed in this article are my own and do not reflect the official positions of the institution or funding body.

Sources of support: none.

Conflict of interest declaration: none.

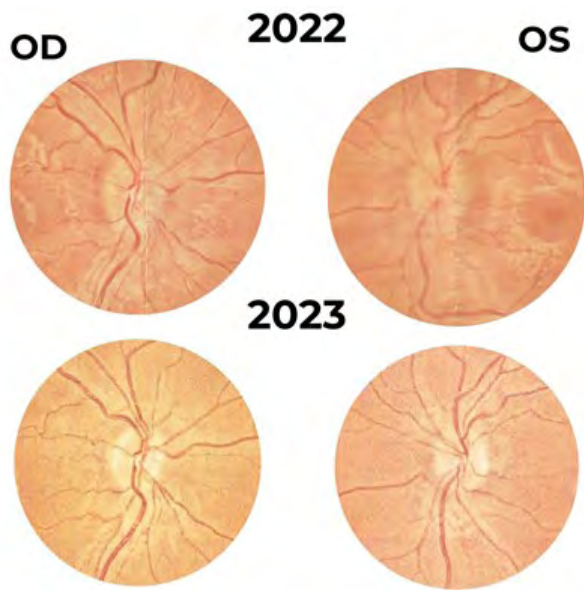


Fig. 1. Fundus of patient K., 24 years old, in 2022 and 2023. Clinical diagnosis: bilateral optic neuritis complicated by sectoral optic atrophy in both eyes. Opticomyelitis (Devic's syndrome).

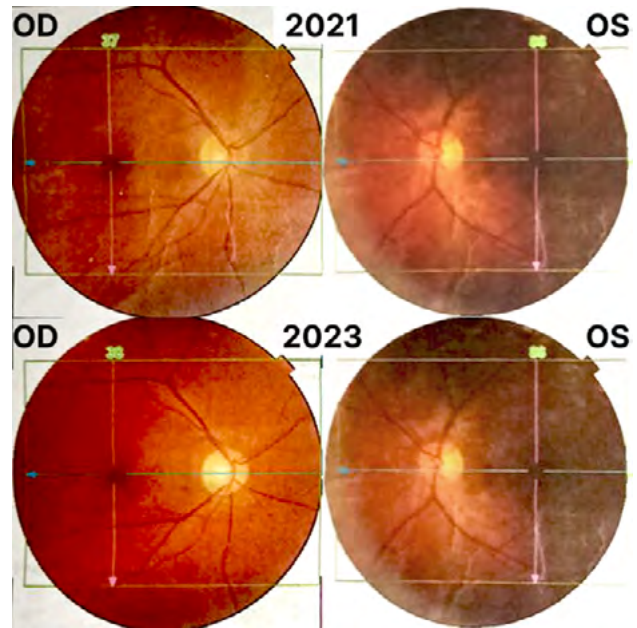


Fig. 2. OCT of patient K., Huvitz Disc 3D. Clinical diagnosis: bilateral optic neuritis complicated by sectoral optic atrophy in both eyes. Opticomyelitis (Devic's syndrome).

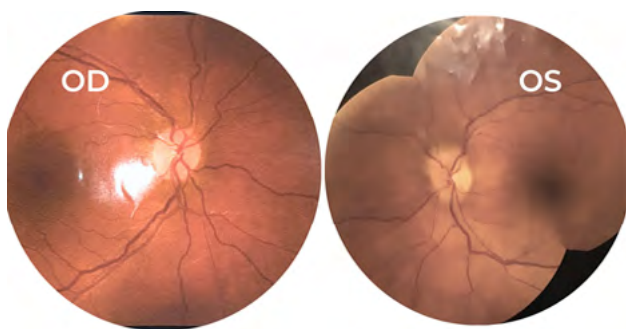


Fig. 3. Fundus of patient P., 42 years old. Clinical diagnosis: bilateral optic neuritis associated with paranasal sinusitis (sinusitis, ethmoiditis) complicated by partial optic nerve atrophy.

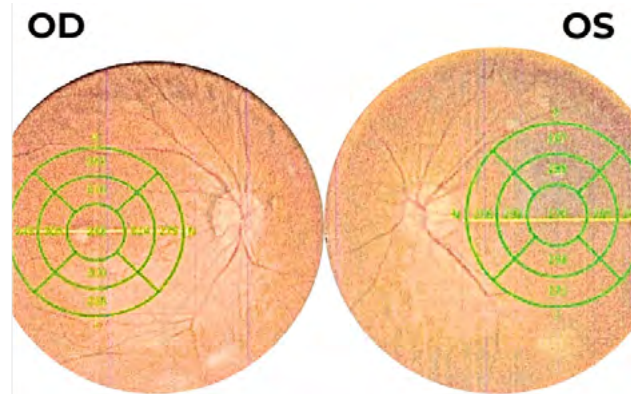


Fig. 4. OCT of patient P., Huvitz Disc 3D. Clinical diagnosis: bilateral optic neuritis associated with paranasal sinusitis (sinusitis, ethmoiditis) complicated by partial optic nerve atrophy.

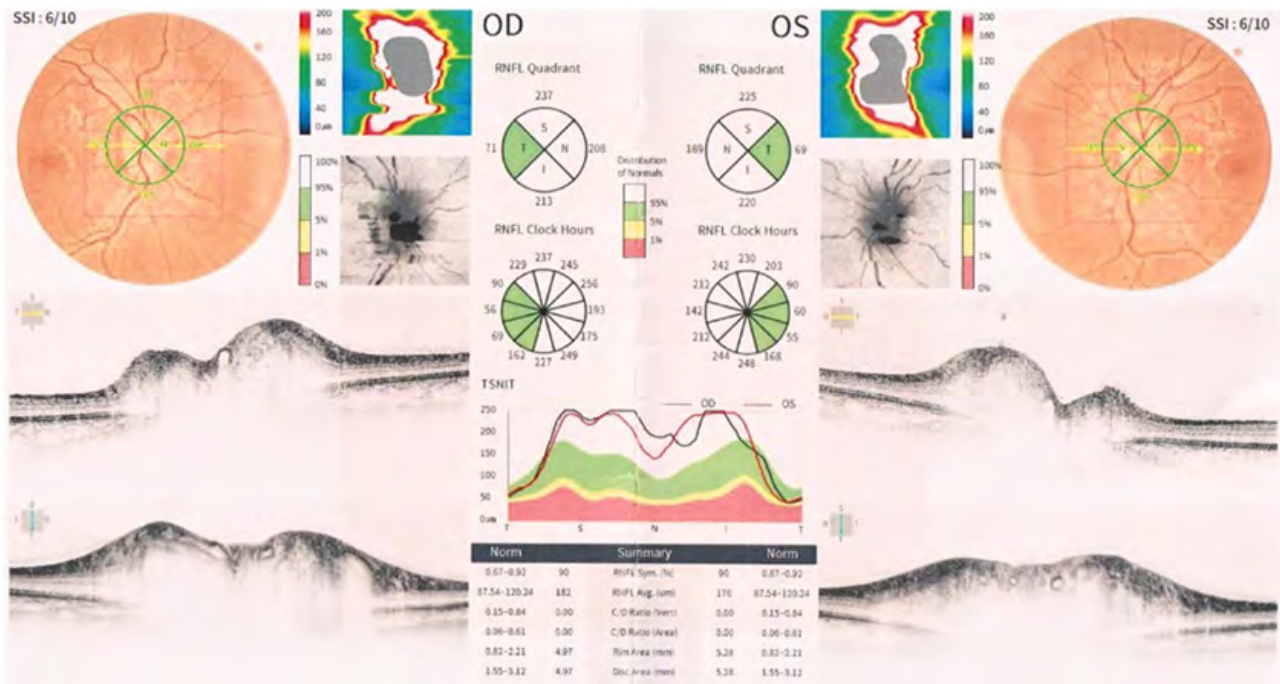


Fig. 5. Fundus of patient N., 37 years old. Clinical diagnosis: anterior non-arteritic ischemic optic neuropathy of the left eye.

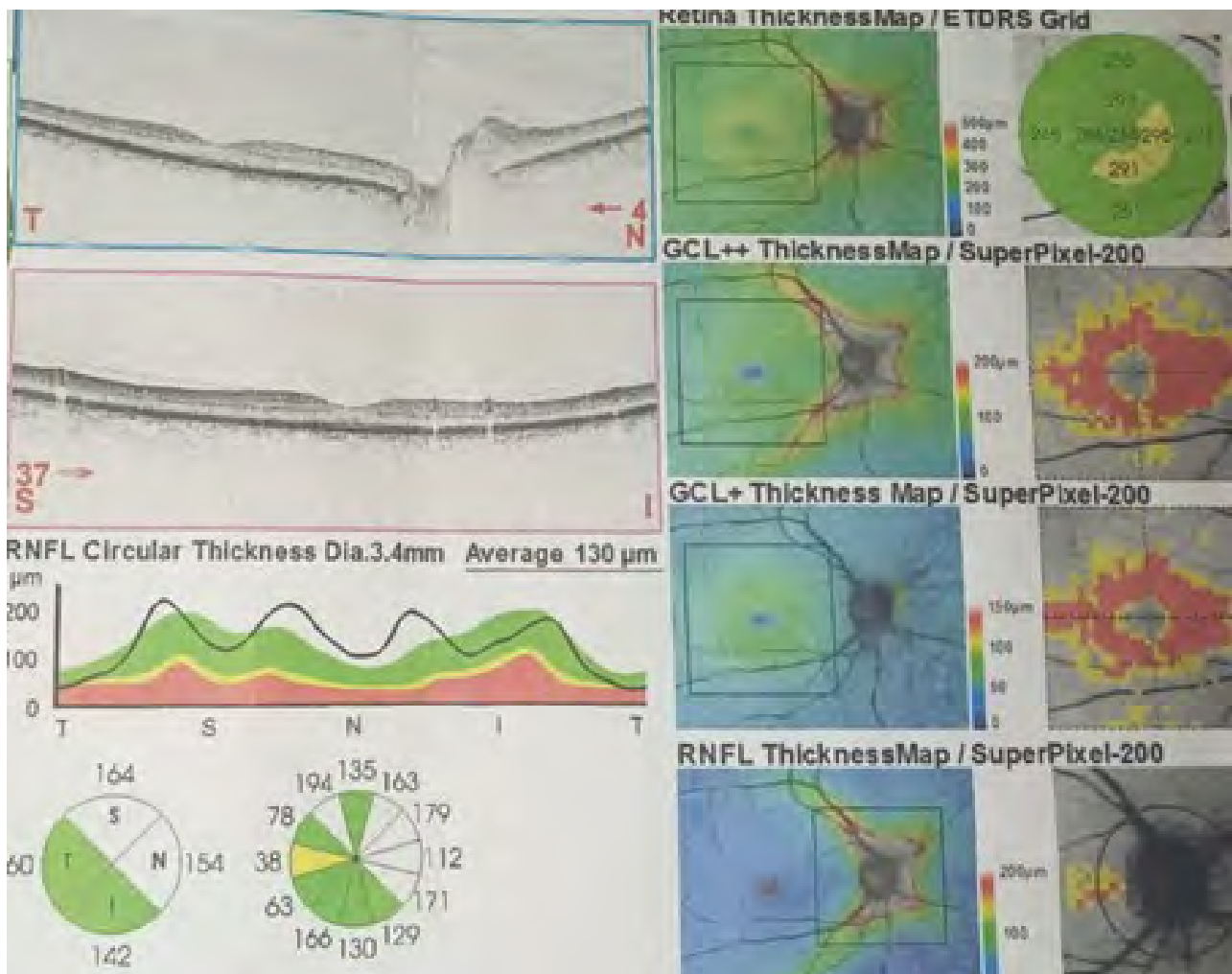


Fig. 6. OCT of patient N., 37 years old. Clinical diagnosis: anterior non-arteritic ischemic optic neuropathy of the left eye.

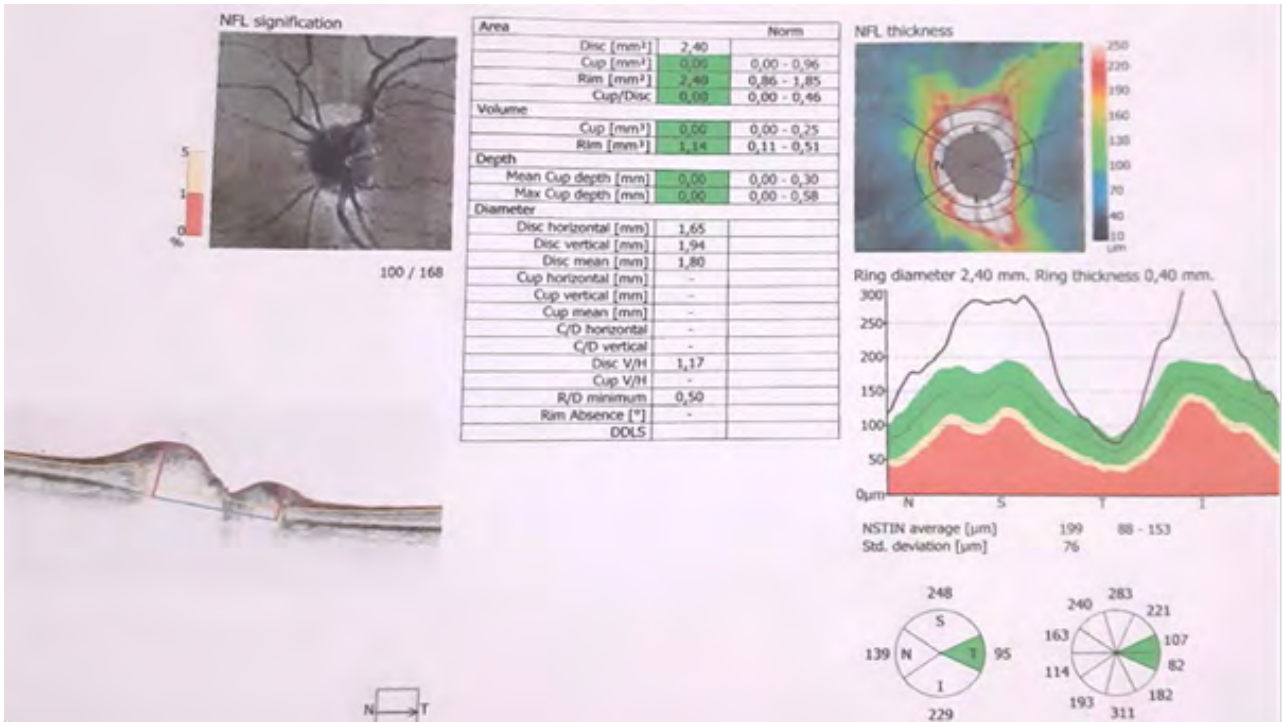


Fig. 7. Fundus of patient B., 52 years old. Clinical diagnosis: anterior non-arteritic ischemic optic neuropathy of both eyes.

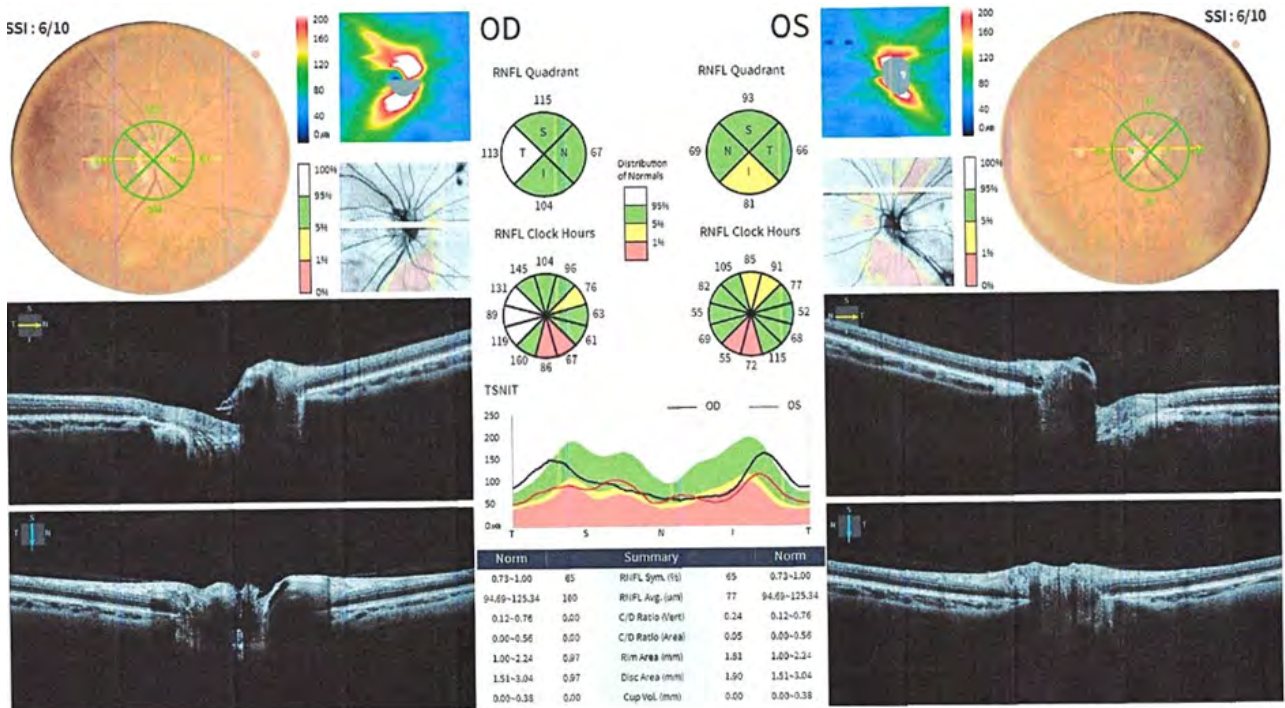


Fig. 8. OCT of patient B. from 2024. Huvitz Disc 3D. Clinical diagnosis: anterior non-arteritic ischemic optic neuropathy of both eyes.