OCT in the Differential Diagnosis of Optic Neuropathies. A Review

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SUMMARY

Optical coherence tomography (OCT) has become a key tool in the differential diagnosis of optic neuropathies (ON), particularly in differentiating between glaucomatous and non-glaucomatous ON. Correct diagnosis is an essential factor for effective treatment management and prevention of progressive loss of vision. While glaucomatous ON is characterized by specific structural changes in the optic nerve head and retinal layers, non-glaucomatous neuropathies can be caused by a wide range of other causes, including inflammatory, ischemic or compressive processes. OCT allows visualization of the fine anatomical details of the optic nerve head and retina, providing valuable information for differential diagnosis. The importance lies in the physician's ability to correctly interpret these images and integrate them into the patient's overall clinical picture. This review focuses on the key features of glaucomatous and non-glaucomatous ON that can be detected early with OCT and highlights the importance of using this technique in everyday clinical practice.

Key words: optic neuropathy, OCT, ganglion cells, glaucoma

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INTRODUCTION

Optic neuropathies (ON) is the term used to designate a group of neurodegenerative diseases characterized by damage to the optic nerve and subsequent deterioration of vision [1]. The optic nerve is an important structure functioning as a link between the eye and the brain, and is composed of over a million ganglion cell axons with a diameter of 0.5–1 µm and a length of approximately 50 mm, which are in bundles separated by the glial cells and fibrous septa [2,3]. The ganglion cells are located in the inner retina and their cores are composed of the ganglion cell layer (GCL). These are neurons that receive visual information from the photoreceptors with the aid of two intermediary types of neurons: bipolar and amacrine cells [1,2]. The nerve fibers of the ganglion cells have no Schwann's sheath, and instead of this are myelinated by oligodendrocytes, which is probably the reason for insufficient structural regeneration in the case of damage to the optic nerve [1,3].

We can divide optic neuropathies into two large groups – glaucomatous (GON) and non-glaucomatous optic neuropathies (NGON). Glaucoma is an optic neuropathy defined by a characteristic cupping of the optic nerve papilla and changes in the visual field, for which intraocular pressure is the chief modifiable risk factor [4]. By contrast, non-glaucomatous optic neuropathy refers to a heterogeneous group of disorders which incorporates compressive, ischemic, inflammatory, traumatic and hereditary or toxic/metabolic (nutrition) conditions that have various clinical manifestations, treatments, visual prognoses and systemic associations [5]. It is important to be able to assess the difference between GON and NGON, because the management of these two entities is different. GON is sight-threatening and requires purely ophthalmological treatment, whereas NGONs often have systemic or neurological associations which threaten not only sight but also the patient's life [6] .

Fundamental manifestations of optic neuropathies

Acute or chronic loss of sight may be caused by any pathology of the visual pathway, including retinal disease. In many cases the symptoms of retinopathy are inconspicuous, and it is difficult to determine whether the cause of loss of vision is retinopathy or optic nerve neuropathy. For this reason it is important to determine the classic manifestations of optic neuropathy: uncorrectable deterioration of visual acuity, reduced color sensitivity, blind spots

in the visual field and Marcus-Gunn pupil (RAPD – relative afferent pupillary defect) [7].

Most ONs are manifested in a deterioration of central visual acuity (CVA) or blurred vision in one or both eyes. However, in these cases deteriorated visual acuity cannot be corrected by optical aids and is not caused by opacities in optic media or lesions in the retina and its vasculature. Examination of the ocular fundus may detect signs of lesions of the optic nerve (atrophy, tumescence, hemorrhage, tumor, infiltration, granuloma and others), though in many cases the fundus appears to be normal, at least in the initial stages of the pathology [8].

We examine color sensitivity with the aid of Ishihara tests or anomaloscope. When evaluating color sensitivity it is important to assess not only the number of tables identified by each eye, but also the speed of identification and the patient's subjective evaluation of the differences between the two eyes [7]. At the same time we determine the presence of desaturation, which patients mostly describe as a paler color when observing an object with the affected eye. Bilateral, slowly progressing ON with bluish-yellow dyschromatopsia in children evokes hereditary ON, for example dominant optic atrophy. By contrast, in the majority of acquired ONs we observe reddish-green dyschromatopsia [8].

Knowledge of the anatomy of the visual pathway is invaluable in the localization of defects of the visual field – scotomas [9]. The shape and localization of scotomas is generally varied – e.g. central, centrocecal, paracentral, arcuate, annular, hemianopic, altitudinal etc. [10]. For example, a lesion of the optic nerve papilla anteriorly from the lamina cribrosa influences the bundle of nerve fibers, which generates an arcuate scotoma respecting the horizontal medial axis. Pathologies affecting the axons of the papillomacular bundle such as toxic or hereditary ON cause centrocecal scotoma. Retrobulbar lesions may cause any kind of image of a defect of the monocular field [9]. In the case of damage to the central part of the chiasma opticum, the crossing nerve fibers are disrupted, leading to blind spots in temporal parts of the visual field in both eyes and thereby a defect which respects the vertical medial axis [9,10]. In the retrochiasmatic pathway, affliction of the ipsilateral temporal axons and contralateral nasal axons results in a homonymous defect in which there is a blind spot of the visual field in the same right or left half of the visual field in each eye [9] (Table 1).

Examination of the pupillomotor pathway is essential for a positive diagnosis of optic neuropathy [9]. Afferent pupillary defect is caused by low sensitivity of the affected eye to light, which is the result of a lesion of the optic nerve, retina, or other ocular pathologies [10]. Pupil reactions must be tested by a strong and concentrated light under photopic and scotopic conditions [9]. Light transferred very rapidly from the healthy side to the affected side, instead of causing pupil constriction causes dilation, and vice versa – transfer from the affected to the healthy side triggers pupil constriction [11]. Marcus-Gunn pupil does not appear in the case of chiasmatic and retrochiasmatic lesions, and similarly this phenomenon cannot be evaluated in diseases of the cornea, cataract, vitreous hemorrhages, papillary drusen or maculopathies [10,11].

However, when recording the patient anamnesis it is also necessary to consider further factors:

- Patient age – acute lateral painless loss of sight in a young individual, particularly a woman, may be an early indication of the first attack of neuritis in connection with multiple sclerosis (MS), whereas the same symptoms in an older individual may be a sign of neuromyelitis optica (NMO)
- Duration of deterioration of visual acuity – acute vs chronic – e.g. ischemic ON may be sudden and painless, whereas compressive ON may be slowly progressing and therefore difficult to detect
- Associated pathologies – coexisting arterial hypertension, diabetes and hyperlipidemia indicate non-arteritic ischemic ON
- Pharmaceutical and social anamnesis – ON triggered by pharmaceuticals may occur especially in the case of Ethambutol, Amiodarone and cytostatic drugs. It is also important to determine dietary and social habits (smoking, alcohol, drugs)
- Family anamnesis – hereditary diseases of autosomal and mitochondrial origin may manifest themselves in atrophy of the optic nerve
- Eye pain – is a common symptom of retrobulbar neuritis – inflammation of the optic nerve causes pain upon eye movement as a consequence of its proximity to the extraocular muscles [12].

OCT in the diagnosis of optic neuropathies

The option of high-resolution noninvasive volumetric imaging of the retina and optic nerve papilla has secured a stable place for OCT instruments in ophthalmological centers. Physiological and pathological changes of the thickness of the cores of the ganglion cell layer (GCL) and retinal nerve fiber layer (RNFL) range within fractions to units of microns, and early detection of these changes requires not only high resolution but also precision and speed (high frequency) of scanning.

OCT is capable of recording and quantifying loss of axons with the aid of measuring peripapillary thickness of the RNFL, and neuron damage with the aid of measuring thickness of the macular GCL, or combined thickness of the GCL and inner plexiform layer (IPL) or ganglion cell complex (GCC – consisting of a combination of the RNFL, GCL and IPL) [13].

The most modern approaches to structural analysis of the optic nerve papilla include the parameter of BMO-MRW (Bruch's Membrane Opening-Minimum Rim Width). The termination of the Bruch's membrane in the optic nerve papilla indicates the place via which the ganglion cell axons leave the eye and form the choroidal and scleral part of the nerve channel. Since axons are unable to pass through an unimpaired Bruch's membrane and leave the eye, this anatomical opening, termed a Bruch's membrane opening (BMO), is indicated as the boundary of the nerve tissue and therefore a stable anatomical point of orientation. The orientation of the neuroretinal rim in

Table 1. Overview of optic neuropathies and their most common ophthalmological findings

Type of optic neuropathy	Onset	Pattern of visual field loss	Pattern of GCL loss on OCT	Ophthalmoscopic finding	Additional features
Glaucoma	Progressive	defect respecting horizontal midline, often arcuate	most often starts in the inferotemporal sector, "snail shell" shape	cupping	often increased intraocular pressure, typical visual field changes, frequent lateral asymmetry
Demyelinating	Acute	central, cecocentral, arcuate	Regular peri/parafoveal loss, „red circle“ sign	75% normal disc (retrobulbar)	neurological signs of brain stem – diplopia, usually bilateral in pediatric, in adults usually unilateral
Ischemic	Acute	arcuate, altitudinal (defect respecting horizontal midline, in NAION)	focal thickness loss of GCL and pRNFL, respecting horizontal midline (present approximately 4 weeks after onset), "sunset" type	pallid swelling of the disc (AAION); swollen disc (usually sectoral) with disc hemorrhages (NAION)	headaches, transient visual loss or diplopia (AAION), 95% of ischemic ON is type NAION, mostly unilateral
Inflammatory	Acute, sub-acute	central, cecocentral, arcuate	Regular peri/parafoveal loss, „red circle“ sign	swollen disc	features of auto-immune diseases (skin rash, arthritis), exquisite responsiveness to systemic steroids
Compressive	Progressive	hemianopic, arcuate	depends on the location of the lesion (prechiasmatic, chiasmatic, postchiasmatic), the most common is chiasmatic and therefore the pattern respects the vertical central axis, "half-moon" type	normal or pale disc	MR will show a compressive mass
Hereditary	Progressive (dominant and recessive), acute (LHON)	central, cecocentral	depends on type of hereditary ON	pale (dominant and recessive) or mildly swollen with peripapillary teleangiectatic vessels (LHON)	onset in childhood with positive family history, mitochondrial DNA testing may reveal Leber`s mutation

NAION – non-arteritic anterior ischemic optic neuropathy, AION – anterior ichemic optic neuropathy, ON – optic neuropathy, GCL – ganglion cell layer, pRNFL – peripapillary retinal nerve fiber layer, MR – magnetic resonance, LHON – Leber's hereditary optic neuropathy, DNA – deoxyribonucleic acid

relation to the BMO changes within the area of the optic nerve papilla, because axons may exit the eye along various pathways, from those parallel to the visual axis to those perpendicular to it. With the aim of accounting for these variations correctly, studies have demonstrated that the minimal distance from the BMO to the internal limiting membrane represents the geometrically most precise measurement of the width of the neuroretinal rim. This measurement is termed the Bruch's Membrane Opening-Minimum Rim Width (BMO-MRW) [14].

In the following chapters we shall present a comprehensive view of the options for diagnosing optic neuropathies using the available structural OCT data (GMPE Spectralis, Heidelberg Engineering):

- Map of thickness of posterior pole
- Map of thickness of retinal nerve fiber layer (RNFL)
- Thickness of peripapillary nerve fiber layer (pRNFL)
- Map of thickness of macular ganglion cell layer (GCL)
- Deviation maps
- Analysis of optic nerve papilla with the aid of the BMO-MRW parameter.

Glaucomatous optic neuropathy (GON)

Glaucoma is a progressive optic neuropathy leading to irreversible damage to loss of sight, and is characterized by a gradual loss of ganglion cells and damage to the optic

nerve. Intraocular pressure, although it is not always raised, appears to be the sole modifiable risk factor. Due to the slow deterioration of sight, frequent lateral asymmetry of the pathology and the neurological mechanisms that compensate for the areas of missing vision, patients are often unaware of the loss of sight until the later stage [15,16].

The basis for diagnosis and monitoring of glaucoma is examination of the visual field and OCT, in which for many years the standard of diagnosis has been especially measurement of the thickness of the pRNFL as well as the macular GCL [9]. According to our findings, no consensus exists to date regarding which parameter is superior to the other, although the results of studies indicate that the superotemporal (ST) and inferotemporal (IT) sectors of the pRNFL manifest the best sensitivity and specificity for early glaucoma where defects of the visual field are not yet present (PPG – pre-perimetric glaucoma), and the universally common model for glaucoma is a decrease of thickness of the IT sector of the pRNFL and GCL (Figure 1) [18,20]. The results of studies vary in the BMO-MRW parameter, though they recommend it as a suitable complement to the pRNFL and add that in comparison with NGON and for the same level of decrease in thickness of the pRNFL, lower values of BMO-MRW are a specific marker of glaucoma, which reflects changes of the neuroglial architecture of the optic nerve papilla that are typical of glaucoma, and supports the fundamental pathophysiological differences [17–19]. Studies also assert that progression of decrease in thickness of the RNFL is more frequent, more rapid and/or more extensive in the GCL/GCIPL, and that the diagnostic capability of the pRNFL is better than the macular GCL/GCIPL in patients with PPG and perimetric glaucoma [20,21]. In the case of the "floor effect" in advanced glaucoma there is extensive loss of thickness of the pRNFL, which makes it impossible to determine further losses. Comparative analyses in this group have indicated that it is less probable that the GCL/GCIPL will manifest this "floor effect" in comparison with the pRNFL, and as a result it is a better indicator of progression in the case of advanced GON [9,20].

Neuritis associated with multiple sclerosis (MSON) and neuromeylitis optica spectrum disorder (NMOSD)

Optic neuritis is an acute inflammatory disease of the visual nerve associated with a deterioration of visual acuity and disorder of pupillary reactions as a consequence of a lesion of the sensory and pupillomotor fibers of the optic nerve [38]. Optic neuritis may be caused by various processes, infectious or non-infectious, but is most often associated with auto-immune neurological disorders, including MS and NMOSD [22,23]. The Optic Neuritis Treatment Trial demonstrated that 95% of patients suffered from unilateral visual affliction, and 92% patients registered the presence of retro-orbital pain, which worsened with eye movement [24]. From a clinical perspective we classify neurites as typical (in association with MS) and atypical, i.e. in association with non-MS disorders (neuromyelitis optica, NMOSD, systemic disorders (sarcoidosis, vasculitis), infectious, parainfectious, postvaccination) [38]. MS associated neuritis especially affects:

- young women of Caucasian ethnicity (ratio to men 2–3:1) [10,23,25]
- age 20–40 years [10,25]
- in 95% of cases unilateral [23]
- best corrected visual acuity (BCVA) in 66% of cases is < 6/60 [23,24]
- BCVA after one year in 72–90% of cases is < 6/12 [25,26]
- OCT finding (Figure 2):
  - pRNFL present slight segmental tumescence (> 110% of normal thickness, reason is blocked axonal flow, which causes tumescence of optic nerve papilla) in 82% of cases, and persists in 58% of eyes after 1 month (also in cases without clinically evident tumescence of optic nerve papilla [13,24]
  - a few weeks to approximately 3 months after an acute episode of MS, atrophy of optic nerve papilla occurs, measurable by decrease of thickness of pRNFL, especially in the temporal sector [13,24]
  - GCL/GCIPL analysis provides more sensitive and reliable measurement of retrobulbar axonal damage, since decrease of thickness occurs earlier, during the acute phase of MSON (approximately after 2 weeks), also when pRNFL remains edematous [16]

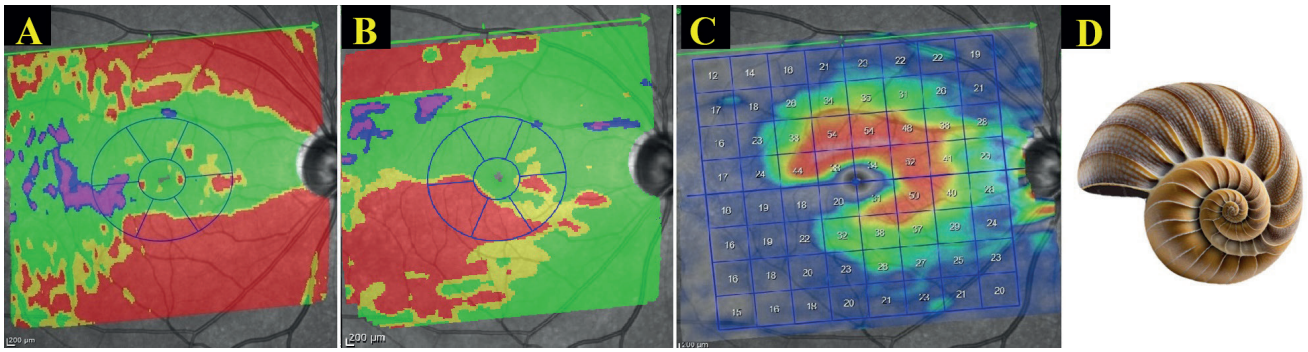


Figure 1. Typical damage patterns in advanced glaucoma: deviation maps show a marked loss of RNFL thickness along both temporal arcades (**A**) and a specific loss of the inferotemporal GCL sector (**B**). This loss correlates with the GCL thickness map generated from the PPoleH scan (**C**), which resembles the shape of a snail shell (**D**)

RNFL – retinal nerve fiber layer, GCL – ganglion cell layer, PPoleH – posterior pole horizontal scan

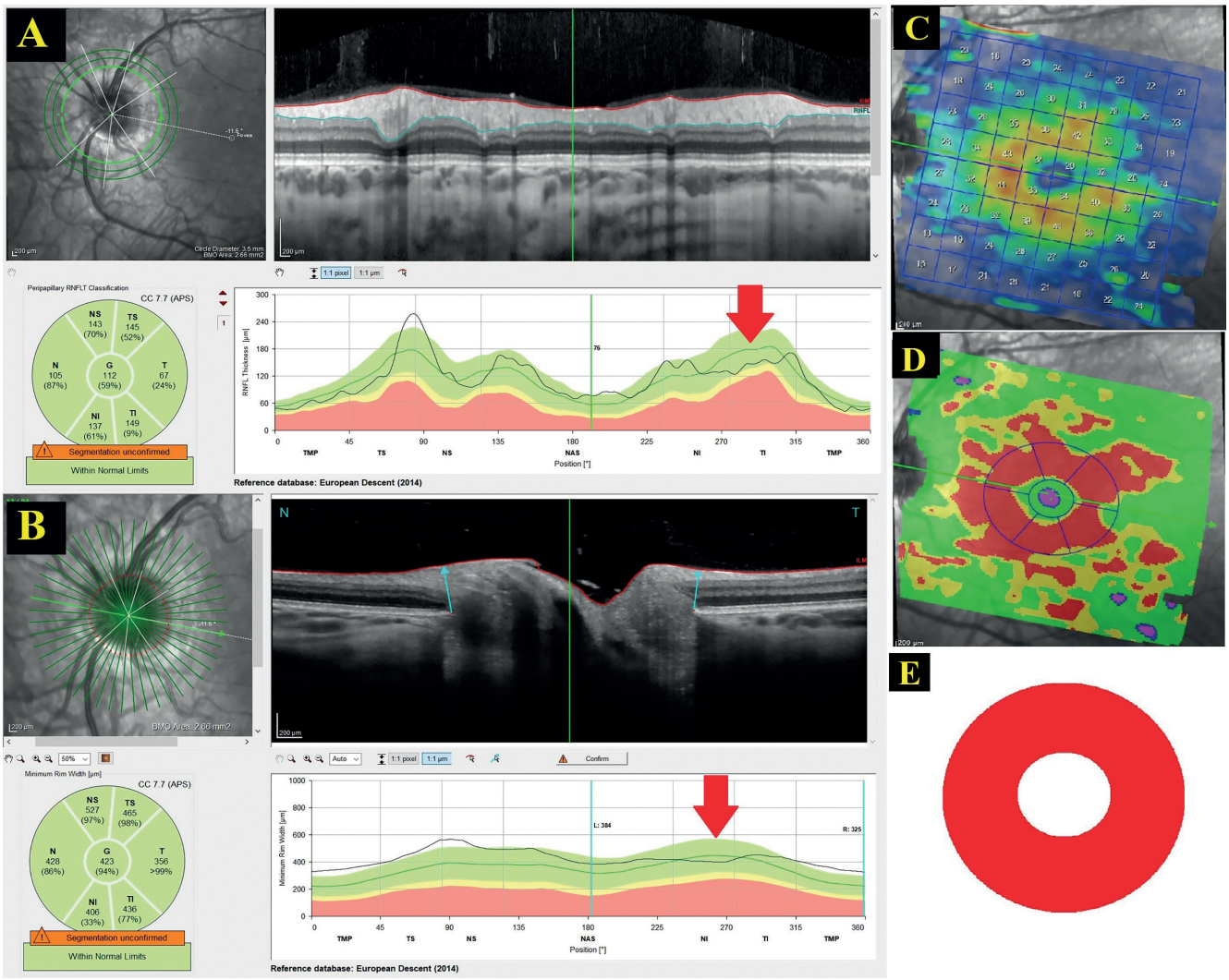


Figure 2. In a young female patient after the second attack of neritis associated with MS, sectoral OCT analysis shows both pRNFL (**A**) and BMO-MRW (**B**) values within normal limits. Note the graphs below the OCT scans, which show elevated character of the disc (**B**) in almost the entire length except for the inferior sectors – the same focal loss approaching the borderline values is also present in the pRNFL analysis (**A**) (marked by red arrows). Ganglion cells in the macular region, however, already show a significant loss of thickness visible on both the thickness map (**C**) and the deviation map (**D**), where we see a „red circle" pattern (**E**)

MS – multiple sclerosis, OCT – optical coherence tomography, pRNFL – peripapillary retinal nerve fiber layer, BMO-MRW – Bruch's membrane opening – Minimum rim width

- microcystic changes which are associated with activity of the pathology may occur parafoveally in region of papillomacular bundle (PMB) on the level of the inner nuclear layer (INL).

It is necessary to point out that microcystic changes are not unique to MS, and have been observed also in other ONs including those with non-inflammatory etiology, for example in compressive ON. Some studies state non-infiltration of contrast during fluorescence angiography, and it is therefore probable that these changes are present due to retrograde axonal degeneration, and a role in the formation of these microcysts is played by loss of function of the Müller cells [16].

NMOSD associated neuritis was once considered a variant of MS, at present it is described as a separate type of demyelination disorder with a specific clinical picture, which attacks the optic nerve and spinal cord [22,25]. Unlike MS it has the following manifestations:

- young women of all ethnicities, though most frequently Asian or African (ratio to men as high as 9:1) [23,25]
- age of 30–40 years [25]
- may be unilateral or bilateral [22,23]
- BCVA 66% < 6/60, 33% < 6/120, upon recurrent attacks up to 70 % < 6/120 [25,26]
- prognostically only 50–60% of patients return to BCVA of < 6/60 [25]
- frequently associated with systemic lupus erythematosus, Sjörgen's syndrome, autoimmune thyroiditis and myasthenia gravis [22]
- OCT finding:
  - NMO leads to more pronounced decrease of thickness of pRFNL and GCL/GCIPL than MSON

- Especially the upper and lower and quadrants are affected, unlike the temporal sector in MSON
- Microcystic changes are present more frequently than in the case of MSON, which may reflect the severity of the ON and the resulting retrograde degeneration of axons [16].

Leber hereditary optic neuropathy (LHON)

Leber hereditary optic neuropathy is a rare mitochondrial disease which is typically manifested in young men by means of a progressive loss of sight as a consequence of optic neuropathy [27]. Ninety percent of cases are caused by three mutations of mitochondrial DNA, which impairs oxidative phosphorylation, leading to a loss of retinal ganglion cells. The disease usually begins by unilateral affliction, and progresses painlessly for a period of days to weeks up to a state of CVA 6/60 and worse. In most patients the disease is manifested also in the other eye within eight weeks, in which approximately 97% of patients have both eyes affected within one year [28].

The natural course of LHON usually has three phases: presymptomatic, acute and chronic [29]. The OCT finding in these phases is described as follows (Figure 3):

- In the subacute preclinical phase the findings differ, some results indicate that:
  - pRNFL – thickening occurs in the upper and lower quadrants, or also in the nasal quadrants, or changes in these quadrants occur minimally. The discrepancies may be caused by the fact that slight pseudoedema of the optic nerve papilla occurs in this phase (tumescence without infiltration on fluorescence angiography), which may affect several quadrants, and the degree of this may cause variable results of measurement. The same applies to the temporal sector, the stability of which differs in the subacute phase – either without changes of thickness, or initial decrease occurs [30–32].
  - GCL/GCIPL – decrease of thickness occurs in these layers before the onset of symptoms, especially in the inner macular quadrants, and for this reason observation of these parameters is consider a better

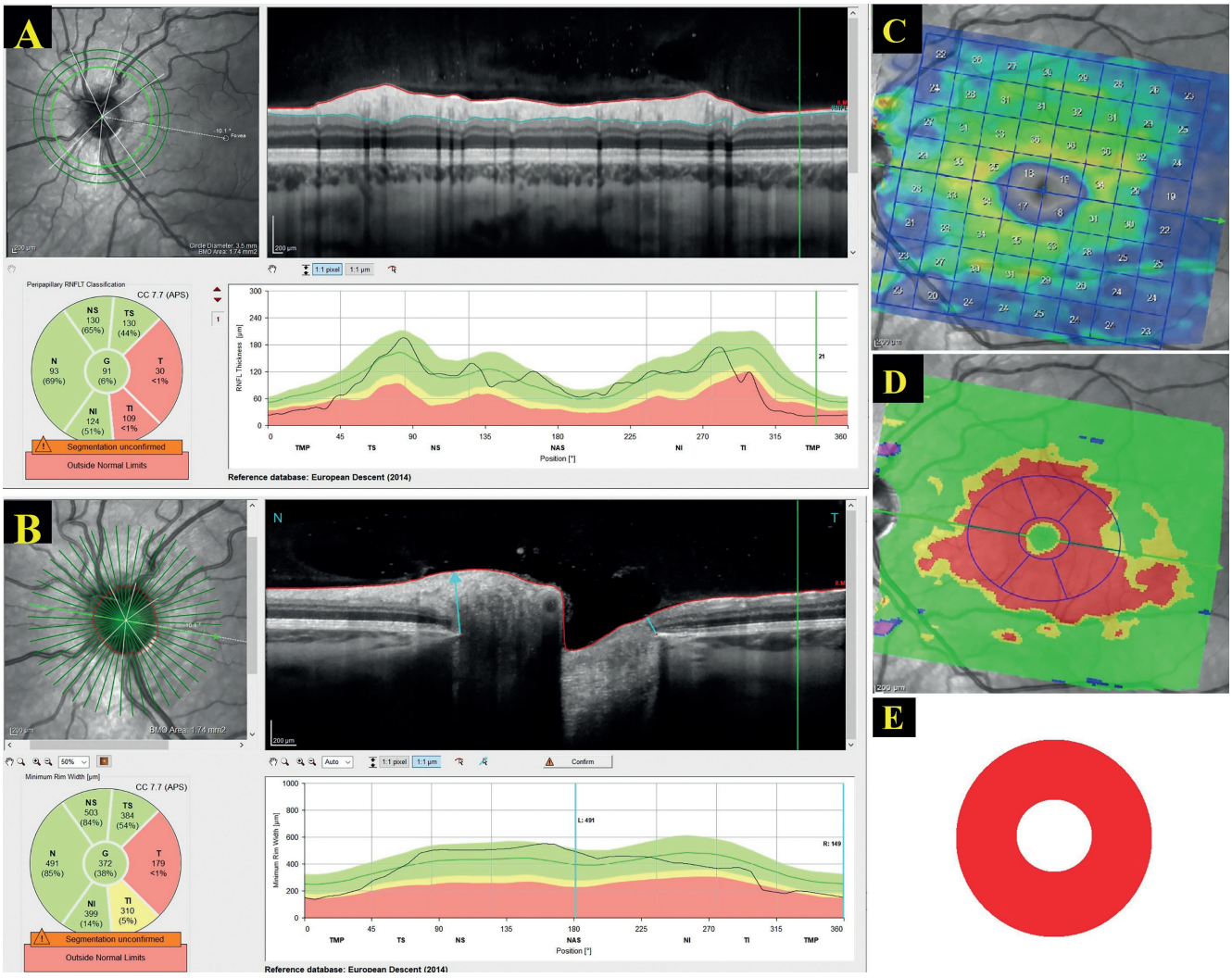


Figure 3. Young patient with genetically confirmed Leber's hereditary optic neuropathy presenting with structural damage to the optical nerve head in the temporal region as seen on both pRNFL (**A**) and BMO-MRW (**B**) analysis, and significant regular loss of macular ganglion cells on both the thickness map (**C**) and deviation map (**D**), again in „red circle" pattern (**E**)

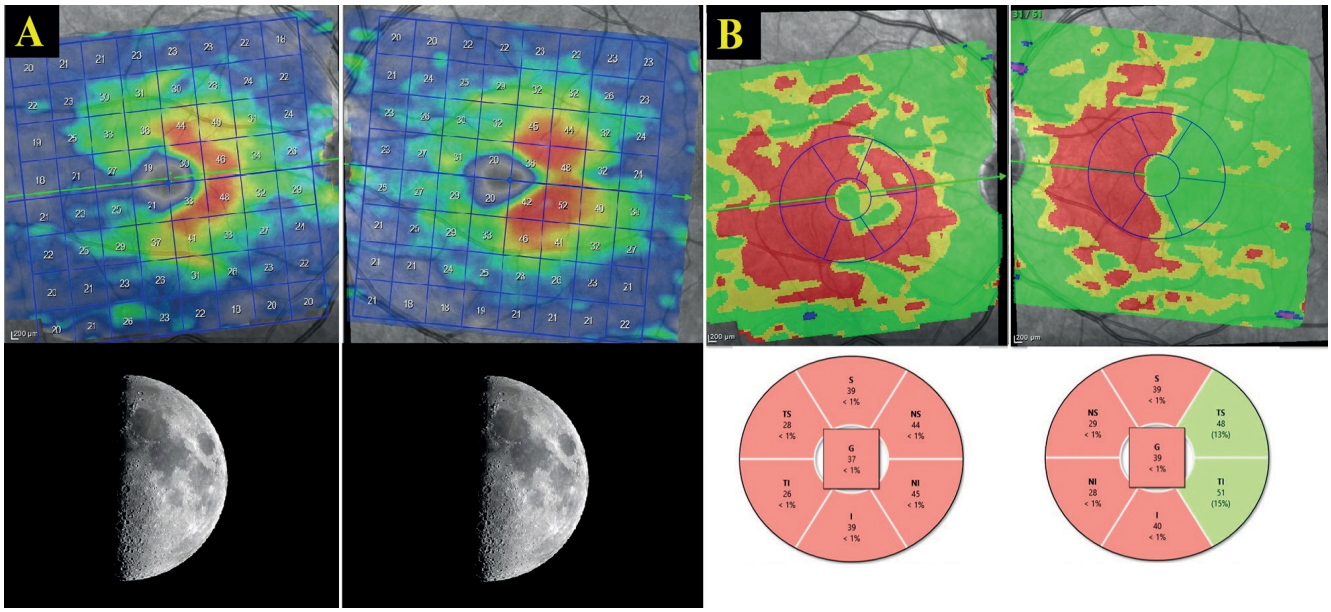


Figure 4. Ganglion cell layer maps of a 10-year-old boy who presented for examination because of rapid deterioration of subjective visual acuity. The left set of images (**A**) shows maps of GCL thicknesses of both eyes with a marked loss of thickness respecting a vertical midline, with a corresponding „half moon" pattern underneath. In the set of images in the right (**B**), the same but in deviation maps, where the visualization of the damage in the right eye encourages an incorrect evaluation – note that the thickness in the nasal sectors, despite the red labeling, are significantly higher than those in the temporal sectors. After OCT imaging, the patient was referred for an acute CT scan, which confirmed tumour expansion in the right hemisphere, causing compression of the right optic tract with slight protrusion into the chiasm area

CT – computed tomography

maker of changes than the pRNFL in the subacute phase [28,30].

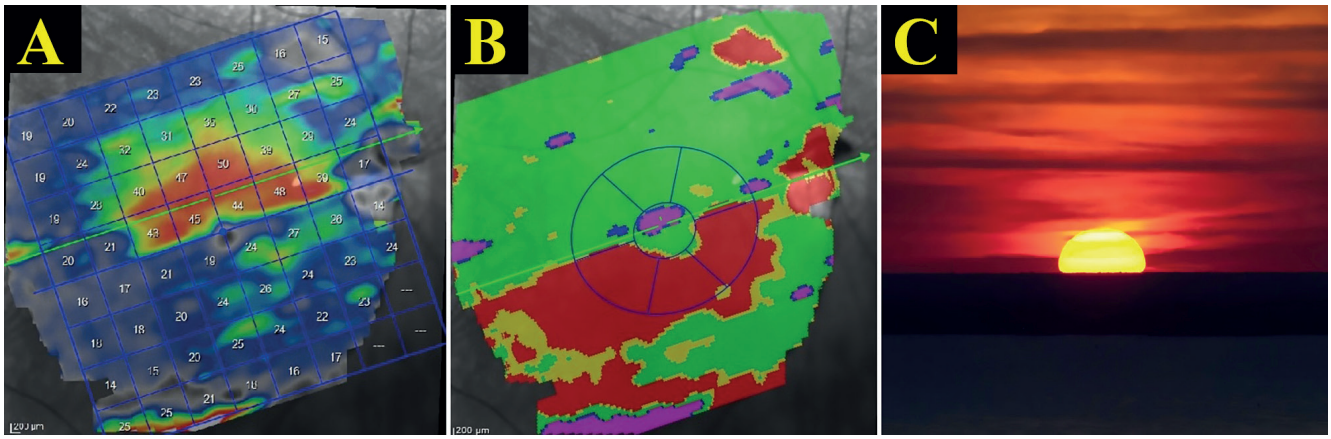
- In the acute phases the findings concur:
  - pRNFL – a rapid decrease of thickness takes place in all quadrants with dominance of the temporal. However, in some cases tumescence of the optic nerve papilla persists, which causes stability or further thickening of some quadrants, especially the upper and lower, though in the subsequent acute phase these cases of tumescence of the optic nerve papilla regress, with an attendant decrease of thickness of the pRNFL also in these quadrants [29]
  - GCL/GCIPL – a rapid decrease of thickness occurs, which further correlates with a deterioration of CVA. In this phase central and centrocecal scotomas occur due to the aforementioned structural changes [29].

The chronic phase is characterized by atrophy of the optic nerve papilla, and a further decrease of thickness of the pRNFL and GCL/GCIPL takes place [29]. A decrease in the thickness of these layers is recorded within a period of 12–24 months of the onset of the disorder, on average by 50–70% of their original state [28,30].

Compressive optic neuropathy (CON)

Compressive optic neuropathy is a heterogeneous disease, which results from damage to the axons of the retinal ganglion cells in the optic nerve by mechanical compression. It is usually manifested in slow, progressive and painless structural and functional changes of the optic nerve. Etiologically this may concern different pathological tissues of varying size, speed of growth or biological composition. The retinotopic restructuring of the axonal fibers in the place of compression itself may be regionally specific according to the location of deposit of the compressing mass – intraocular, intraorbital, intracanalicular, in the cranial section of the optic nerve, chiasm or optic tract [33]. Compression of the visual pathway appears most commonly in the region of the chiasm, for example lesion of the hypophysis, but compression of the optic nerve may occur anywhere, for example by the meningioma or in the orbit by enlarged muscles in the case of a disorder of the thyroid gland [13]. As a result, the clinical picture incorporates the model or severity of damage, the speed of progression of loss of visual functions, morphology of the optic nerve papilla and other neurological and systemic symptoms and symptoms at the time of diagnosis, which may be variable [33]. Compression causing damage to the optic nerve may be detectable by OCT earlier than it is visible by fundoscopy, and for this reason plays an important role in diagnosis (Figure 4):

- A decrease of thickness of the pRNFL and GCL/GCIPL also takes place, in which the GCL/GCIPL parameter is generally extraordinarily sensitive in the case of CON – decrease of thickness occurs here earlier than in the pRNFL, and changes are visible earlier than on perimetry
- Lesions compressing the optic chiasm from above or below exert pressure predominantly on the crossing nasal fibers, which leads to a retrograde loss of the pRNFL on the nasal and temporal side of the optic nerve papilla, resulting in "bow-tie" atrophy (this need not necessa-



Obrázok 5. Ganglion cell layer thickness map (**A**) and GCL deviation maps (**B**) show a significant altitudinal defect in the lower half respecting the horizontal midline, typical for AION; „sunset" pattern (**C**)
AION – predná ischemická optická neuropatia

rily cause a decrease of the pRNFL only in the temporal and nasal quadrant, though it is proportionally more pronounced in these quadrants)
- The model of decrease of the GCL/GCIPL is more consistent (binasal thinning appears upon compression of the chiasm, homonymous upon compression of the tract or damage to the corpus geniculatum laterale (CGL)
- Post-CGL lesions may also lead to homonymous thinning of the GCL/GCIPL by means of transsynaptic retrograde degeneration, but it may take more than a year before this occurs [13].

Anterior ischemic optic neuropathy (AION)

Anterior ischemic optic neuropathy is one of the most common causes of acute, unilateral and painless neuropathy of the optic nerve in individuals over the age of 50 years, and is caused by acute ischemic optic nerve papilla [34,35]. It is classified as arteritic (A-AION) when it occurs secondarily after vasculitis, or non-arteritic/idiopathic (NAION) if there is a different cause. In both cases tumescence of the optic nerve papilla is present (usually regressing after 8–12 weeks), as well as Marcus-Gunn pupil and defect of the visual field respecting the horizontal medial axis, most frequently in the lower hemisphere. In the case of NAION, peripapillary hemorrhages are most often present, with a small diameter of the scleral canal and C/D (cup-to-disc) ratio of < 0.2. Excavation of the optic nerve papilla is rare, but present as a consequence of A-AION [36,37]. The following signs are present on OCT:
- During the first two months after the onset of the pathology there is a progressive thinning of the pRNFL in as many as 80% of patients
- Atrophy occurs between 2–4 months after the onset of the pathology, and stability is typically achieved 6 months after the onset of the disease
- Thinning of the GCL/GCIPL occurs earlier than the pRNFL, usually within a few days of onset, and typically is in the form of decrease respecting the horizontal axis corresponding with scotoma present on perimetry (Figure 5)
- Differential diagnostics with branch retinal artery occlusion (BRAO):
  - NAION – thinning occurs in layers of RNFL, GCL, IPL
  - BRAO – thinning occurs in layers of RNFL, GCL, IPL, INL [13].

CONCLUSION

The article provides a comprehensive overview of the most common optic neuropathies, their manifestations and typical models of damage detected by OCT instruments. Determination of the final diagnosis is not straightforward, and requires a number of examinations and often also interdisciplinary cooperation. However, a correct understanding of pathophysiological processes of this group of diseases in combination with analyses provided by modern OCT instruments may speed up the entire process of diagnosis. Early diagnosis is of key importance especially in the case of neuropathies in which the onset and course is rapid, and patient management has a more pronounced influence on the patient's quality of life.

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