

Optical Coherence Tomography of the Outer Retinal Layers

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Abstract

Spectral domain optical coherence tomography (OCT) provides high-resolution images of the different layers of the macula approximating histological sections. It plays a significant role in the management of outer retinal diseases such as exudative age-related macular degeneration, central serous chorioretinopathy, and polypoidal choroidal vasculopathy. The OCT is an important complementary tool in detecting activity of diseases such as choroidal neovascularization and polypoidal choroidal vasculopathy, and is indispensable in monitoring the response to treatment and decision-making regarding retreatment. The use of OCT, together with anti-vascular endothelial growth factor therapy, has greatly improved visual outcomes of many outer retinal diseases.

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An understanding of the normal outer retinal structure, as represented on the spectral domain optical coherence tomography (OCT) (fig. 1), is crucial in the evaluation of the morphological changes which result from retinal diseases. Using the time-domain OCT, the detailed imaging of the individual layers and structures of the

outer retina was not possible. This has changed with excellent spatial resolution afforded by spectral domain OCT technology, which delivers sections of the macula closely approximating histological specimens viewed microscopically.

The outer retinal layers of the normal eye show three distinct bands on the spectral domain OCT: (1) retinal pigment epithelium (RPE) band – consisting of the RPE, Bruch's membrane and choriocapillaris; (2) anterior to the RPE – comprising external limiting membrane, inner segment-outer segment (IS-OS) line, and Verhoeff's membrane; (3) posterior to the RPE – middle and outer layers of the choroid.

Further, Pircher et al. [1] described four distinct bands as follows:

- Band 1: external limiting membrane.
- Band 2: interface of the inner and outer segments of the photoreceptor layer (IS-OS junction).
- Band 3: outer segment RPE interdigitation (Verhoeff's membrane).
- Band 4: RPE/Bruch's membrane complex (fig. 2).

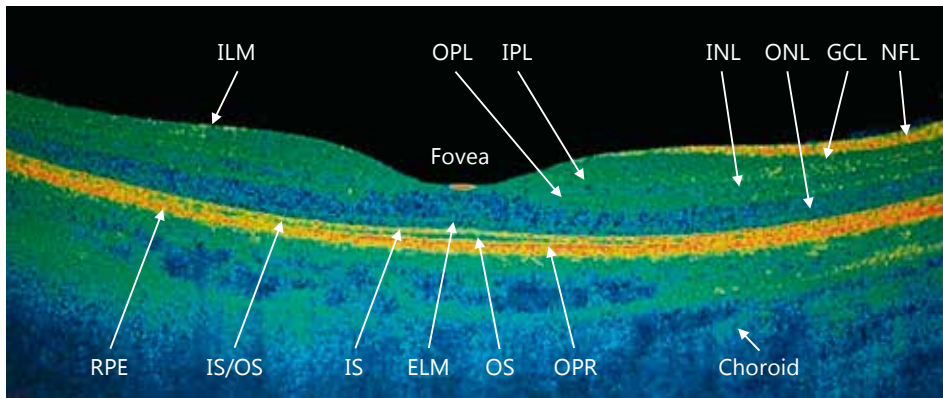


Fig. 1. Spectral domain OCT of the normal macula. NFL = Nerve fiber; OPL = outer plexiform layer; ILM = internal limiting membrane; ONL = outer nuclear layer; GCL = ganglion cell layer; ELM = external limiting membrane; IPL = inner plexiform layer; IS = photoreceptor inner segment; INL = inner nuclear layer; OS = photoreceptor outer segment; RPE = retinal pigment epithelium; IS/OS = interface between IS and OS.

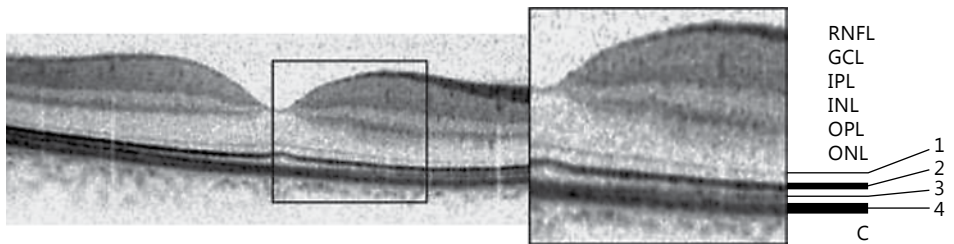


Fig. 2. Four distinct bands of the outer retina on spectral domain OCT according to Pircher et al. [1]. Band 1: ELM. Band 2: interface of the inner and outer segments of the photoreceptor layer (IS-OS junction). Band 3: outer segment-RPE interdigitation (Verhoeff's membrane). Band 4: RPE/Bruch's membrane complex. RNFL = Retinal nerve fiber layer; GCL = ganglion cell layer; IPL = inner plexiform layer; INL = inner nuclear layer; OPL = outer plexiform layer; ONL = outer nuclear layer; C = choriocapillaris and choroidea.

How to Evaluate the Macular OCT

(1) Study carefully the distinct most prominent hyper-reflective RPE band: look for irregularities, thickening, fragmentation, breaks, disruption, shadowing and separation of RPE from Bruch's membrane.

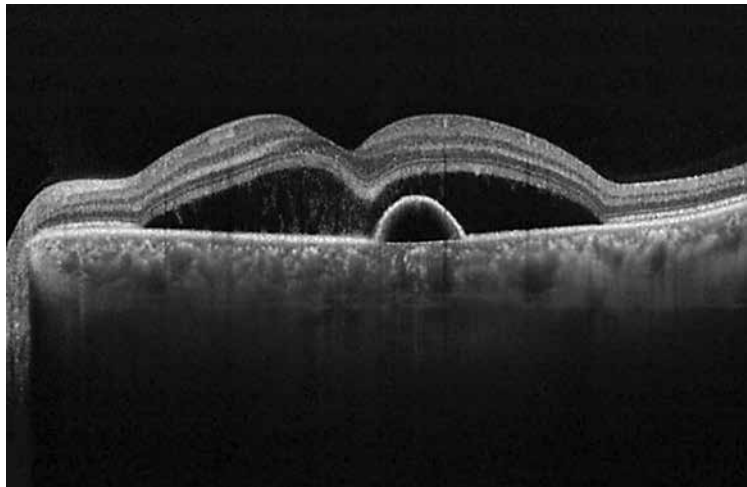
(2) Turn your attention to the zone anterior to the RPE band, taking note of the following fea-

tures: retinal thickness, presence of cavities, deposits and hyper-reflective dots.

(3) Analyze the neurosensory retinal layers, membranes and vitreoretinal interface.

(4) Examine the zone posterior to the RPE band to determine if there is hyper-reflectivity (choroidal atrophy) or hyporeflectivity (shadowing).

Fig. 3. Neurosensory detachment in CSC, with a small pigment epithelial detachment just temporal to the fovea. Elongated outer segments of photoreceptors are clearly visible on the underside of the neural retina. The 'septae' traversing the subretinal space on the nasal aspect of the detachment represent retinoschisis, indicative of the long-standing nature of the detachment.



OCT Features of Central Serous Chorioretinopathy

The classic OCT finding in active central serous chorioretinopathy (CSC) is localized neurosensory detachment. In addition, elongated outer segments and pigment epithelial detachment may accompany the neurosensory fluid. Long-standing cases may show a 'split' in the neural retina (retinoschisis), or RPE thinning and atrophy (fig. 3).

Some cases may be caused by an optic disc pit, which shows a deep defect in the optic nerve margin, associated with a schisis-like separation between the inner and outer retina. Enhanced depth imaging (EDI)-OCT is a new imaging modality to enable high-resolution OCT imaging of external retinal layers, the choroid and lamina cribrosa [2].

In a study of 19 patients with CSC, Imamura et al. [3] found a mean subfoveal choroidal thickness of 505 μm (SD 124 μm , range 439–573 μm), significantly higher than normative data reported previously. Among those who had unilateral CSC, choroidal thickness was also increased in the disease-free fellow eye [4]. Increased choroidal thickness is thought to be due to increased cir-

ulation and vascular dilatation, consistent with indocyanine green (ICG) angiography studies which show diffuse ICG leakage in the choroid in both eyes, even if only one eye has clinically demonstrable CSC. Furthermore, Maruko et al. [5] have shown that choroidal thickness is reduced after successful treatment with photodynamic therapy compared to laser photocoagulation. Photodynamic therapy is thought to reduce choroidal vascular hyperpermeability, leading to reduction in choroidal thickness as measured on EDI-OCT (fig. 4).

OCT in Age-Related Macular Degeneration (AMD)

The OCT shows different features depending on the type of age-related macular degeneration (AMD). In dry AMD, OCT shows drusen and geographic atrophy. In wet AMD, the OCT findings include choroidal neovascularization (CNV type I, II), pigment epithelial detachment, RPE tear/rip, retinal angiomatous proliferation (RAP) and polypoidal choroidal vasculopathy (PCV).

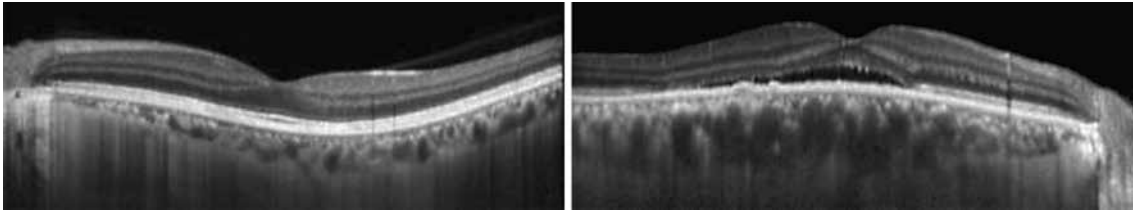


Fig. 4. Normal choroidal thickness on EDI-OCT (right); marked increased choroidal thickness on EDI-OCT (left) in a patient with acute CSC. The diffuse choroidal vascular hyperpermeability results in significant thickening of the choroid as measurable on EDI-OCT. With verteporfin photodynamic therapy, the choroidal thickness normalizes, unlike focal laser photocoagulation, in which the choroidal thickness remains unchanged.

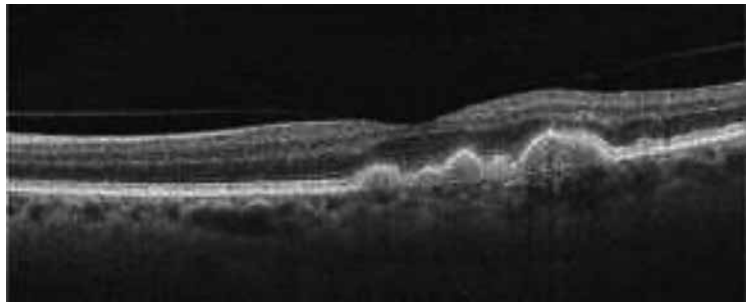


Fig. 5. The OCT in dry AMD showing nodular deposits between the RPE and Bruch's membrane, causing focal elevation of the RPE.

OCT in Dry Age-Related Macular Degeneration

Soft drusen may be visualized as focal, shallow elevations of well-defined RPE band depositions between the RPE basal lamina and the inner collagenous layer of Bruch's membrane (fig. 5). Geographic atrophy manifests as reduced macular thickness, loss of RPE cells, shown as thinning or absence of the RPE band, increased deep reflectivity stemming from the unimpeded penetration of light allowed when RPE melanin is absent and collapse of the outer retinal layers immediately adjacent to the zone of atrophy.

Choroidal Neovascularization

Typical features of wet AMD include macular thickening from neurosensory, intraretinal and sub-RPE fluid; RPE fragmentation, fusiform

thickening, splitting or disruption; and high reflectivity of the choriocapillaris (fig. 6).

Two main types of CNV based on the location of the lesion in relation to the RPE layer have been described. The clinical observations were correlated to histopathology sections and predated OCT examination. Type 1 CNV was described as sub-RPE CNV, where the lesion was limited to the area between the RPE and Bruch's membrane. This is clearly illustrated in OCT imaging of type 1 CNV. Type 2 CNV is described as neovascularization which occurs in the subretinal space above the level of the RPE. Since this early classification system, other authors have added a type 3 CNV, which refers to retinal angiomatous proliferation within the deep neural retina [6].

The OCT is indispensable in detecting exudative activity of the CNV in AMD. Figure 7 shows a case which illustrates the difficulty in determining the cause of reduced vision and metamorphopsia