# Chapter 1
# Clinical Applications of Retinal Optical Coherence Tomography


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The developments of medical imaging techniques and medical image analysis methods are always motivated by the needs arising from clinical applications. This chapter introduces anatomy of the eye and the retina, describes various types of eye diseases that can be visualized with OCT imaging, and therefore presents the must-know background knowledge for readers interested in retinal OCT image analysis.

## 1.1 Anatomy of the Eye and Retina

### *1.1.1 Simple Anatomy of the Eye*

The eye is an organ that perceives light and visual information. There are five senses in human body, including vision, hearing, smell, touch and taste. More than 80% of information we received is obtained through vision perceived by the eyes.

The structure of the eye is like a ball, although it is not a perfect sphere. There are three layers of coats, enclosing three intraocular components: (1) The front of the eyeball is cornea, which is transparent and contribute to most of the refractive power of the eye; the posterior part of the outermost layer is sclera, which consists of fibrous tissue and protects the inner structures. (2) The middle layer of the eyeball is vascular tunic or uvea, which consists of iris, ciliary body and choroid. The center of iris is open and called pupil. The muscles inside the iris control the size of pupil and the amount of light getting into the retina. Ciliary body is responsible for the generation of aqueous humor and accommodation. The choroid is located just outside the retina and provides nutrition and oxygen for the outer part of retina. (3) The innermost

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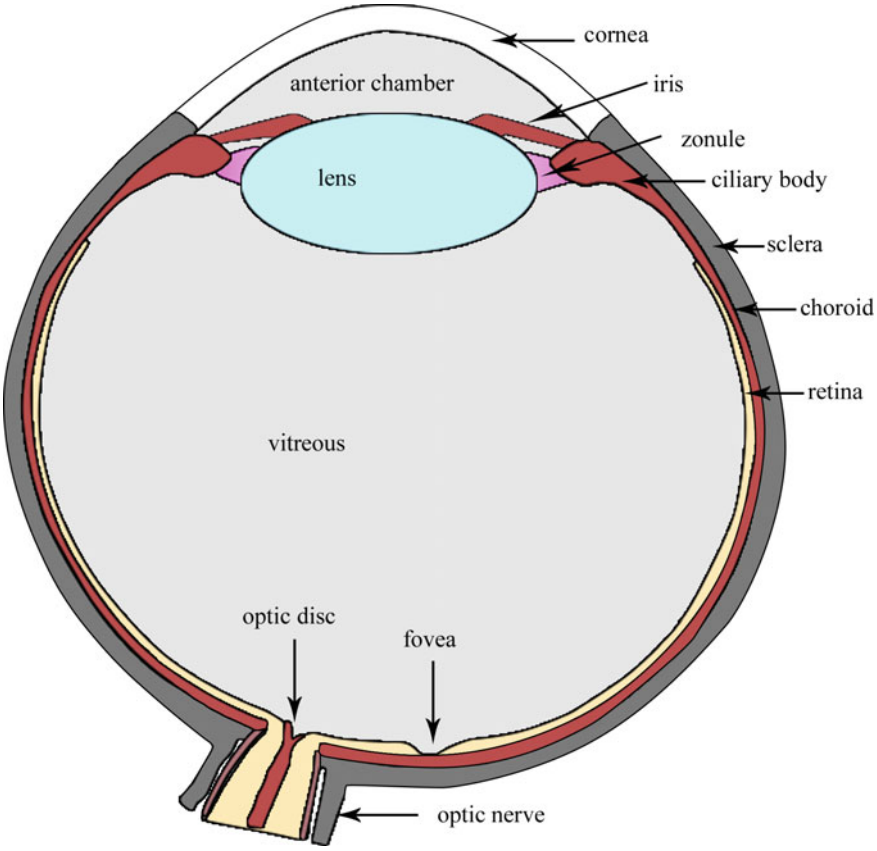


**Fig. 1.1** Illustration of the anatomy of human eye

layer is retina, which is an extension of central nerve system and responsible for the transduction of visual signal into neural signal. The intraocular components include aqueous humor, lens and vitreous body. The lens is connected to ciliary body by the zonules. Aqueous humor and vitreous body locate in front and back of the lens (Fig. 1.1).

The eye is a very special organ. The optical media, including cornea, aqueous humor, lens and vitreous, are transparent. This character allows light getting into the innermost layer, retina, and also allows visualization of retinal structure using various instruments, including optical coherence tomography.

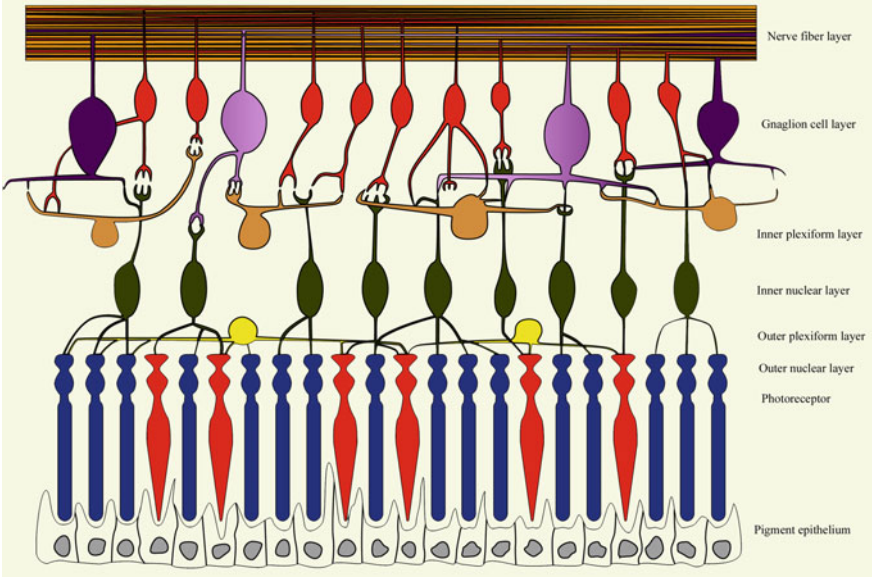


**Fig. 1.2** Cross-sectional diagram showing the structure of human retina

### *1.1.2 Simple Histology of Retina*

Retina is the most important structure of the eye. It is a neural tissue and transduces light into neural signal.

The histology of the retina consists of ten layers. From inner to outer, they are internal limiting membrane, retinal nerve fiber layer, retinal ganglion cell layer, inner plexiform layer, inner nuclear layer, outer plexiform layer, outer nuclear layer, external limiting membrane, photoreceptor inner and outer segment, retinal pigment epithelium. Retina is transparent except for the blood vessels and retinal pigment epithelium monolayer (Fig. 1.2). Transparency of retina allows light passing though and reaching the photoreceptors, where the photo-neural transduction occurs.

There are two blood supply systems to retina, retinal vascular system and choroidal vascular system. The retinal vascular system rises from optic disc, branches on the retinal nerve fiber layer, and forms three layers of capillary, located in the retinal ganglion cell layer, inner plexiform layer and outer plexiform layer. The retinal vascular system supplies the inner layers of retina. The outer retina is avascular, and oxygen and nutrition is supplied from the choroidal capillary through retinal pigment epithelium.

On fundus photography, the optic disc is an important landmark of the retina. It is about 1.5 mm diameter, with a cup at the center. It is located about 2.5 optic disc diameter nasal to the fovea, which is the center point of the macula. The fovea is special because it consists of abundant number of cone photoreceptors, which is

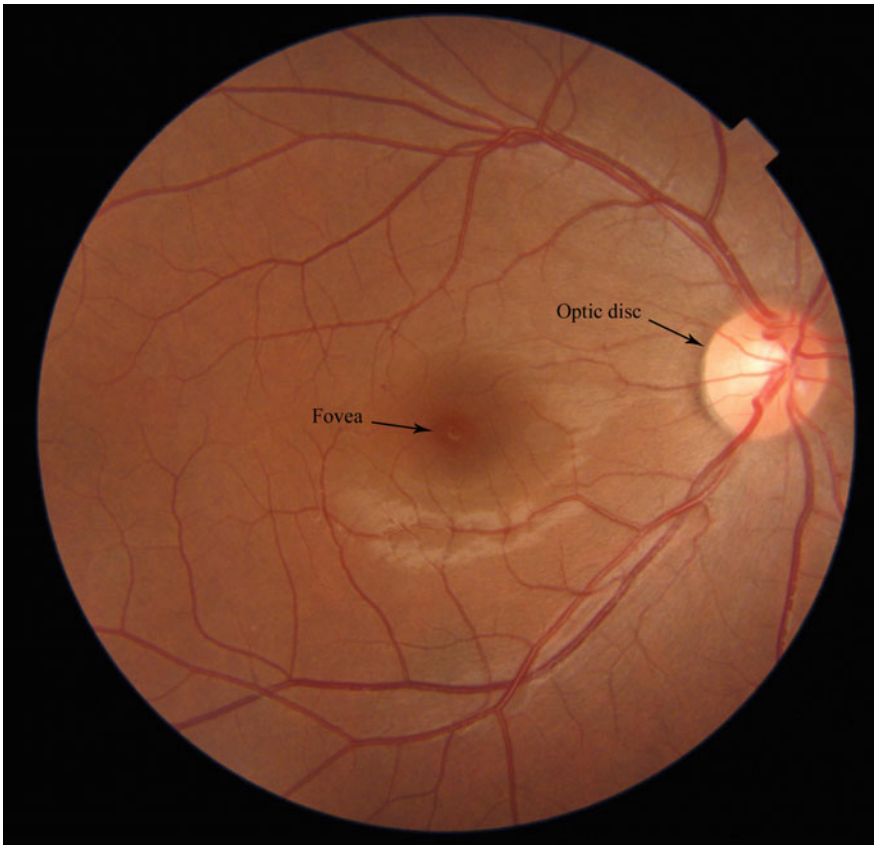


**Fig. 1.3** Fundus photography of a normal human retina

responsible for fine vision and color vision. There is no inner retinal structure in fovea, hence, there is no blood vessel and allows light to reach the photoreceptors without any disturbance (Fig. 1.3).

### *1.1.3 Normal Macular OCT Image*

OCT provides high resolution imaging for the cross-sectional structure of retina. The reflectivity of tissue is determined by the optical character of tissue itself. Vitreous has the lowest reflectivity in normal subjects. The highest reflective band at the inner retina is retinal nerve fiber layers, which is thickest at the parapapillary region and thinnest just temporal to the fovea. Generally, the nerve fiber layers have higher reflectivity compared with nuclear layers. OCT not only demonstrates the 10 layers of retina, but it is also able to visualize the detail structures of photoreceptor and choroid

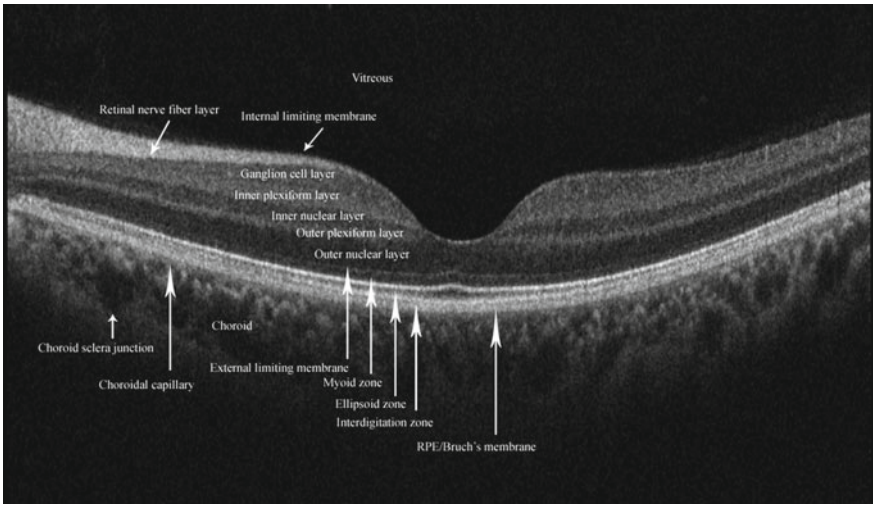


**Fig. 1.4** Retinal layers on optical coherence tomography in normal subject

[1]. There are four hyper-reflective bands at outer retina: external limiting membrane, inner segment ellipsoid zone, interdigitation zone, and RPE/Bruch's complex. The choroidal-scleral interface can be identified and the thickness of choroid can be measured (Fig. 1.4).

## 1.2 Vitreomacular Interface Diseases

The vitreous body is a transparent structure which fills the space in front of the retina. In physiological condition, vitreous body provides mechanical support for retina. However, vitreous may degenerate with aging or under pathologic conditions. The disorders at vitreomacular interface cannot be well-recognized until recently due to the availability of OCT, which provides high resolution cross-sectional imaging for visualizing the vitreomacular interface.

There are several disorders at vitreomacular interface, including vitreomacular adhesion, vitreomacular traction, macular hole, epiretinal membrane. The disorders of vitreomacular interface can be seen in aging population, and also secondary to other diseases, such as high myopia, proliferative diabetic retinopathy, etc.

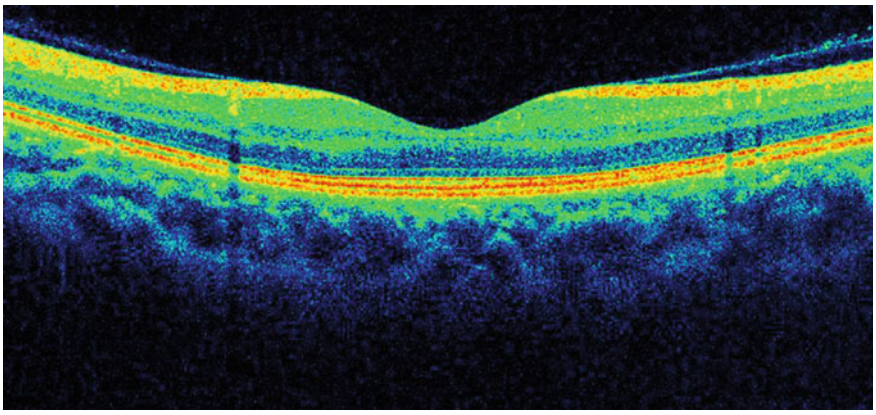

**Fig. 1.5** Vitreomacular adhesion. There is separation of vitreous from retina at perifoveal region. Please note there is no morphological change of intraretinal structure

### *1.2.1 Vitreomacular Adhesion*

Vitreomacular adhesion is defined as perifoveal vitreous detachment with the adhesion of vitreous at fovea, without changing the intraretinal structures [2]. Vitreomacular adhesion is a physiological status, because most eyes have complete vitreoretinal adhesion at birth, and develop to posterior vitreous detachment with aging. The vitreous is adhered to retina most tightly at fovea, optic disc and peripheral retina. The process of posterior vitreous detachment begins at perifoveal region. Vitreomacular adhesion can be further classified as focal ($\leq$1500 um) and broad (>1500 um) adhesions according to the size of adhesion (Fig. 1.5).

### *1.2.2 Vitreomacular Traction*

Vitreomacular traction is defined as perifoveal vitreous detachment with the adhesion of vitreous at fovea which changes the intraretinal structures [2]. Traction at the fovea may result in contour changes at the inner surface, intraretinal pseudocyst and separation of retina from the RPE. These changes lead to metamorphopsia and vision decrease. Vitreomacular traction can also be classified as focal ($\leq$1500 um) and broad (>1500 um) adhesion according to the size of adhesion (Fig. 1.6).

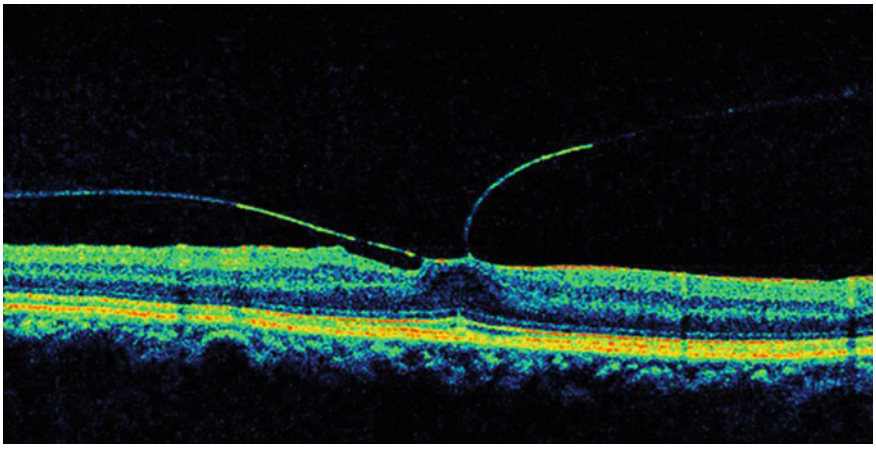

**Fig. 1.6** OCT of focal vitreomacular traction shows the high reflectivity band incompletely detached from the retina and changes of the contour of the intraretinal structures due to the slightly elevated inner surface of fovea

### *1.2.3 Full Thickness Macular Hole (FTMH)*

Macular hole was defined as interruption of full thickness retina tissue at fovea. The opening of retina involves all neural retinal layers, from internal limiting membrane to photoreceptor. On fundus photography, macular hole is usually round, with a circus of edema around the hole.

Macular hole is classified into 4 stages by Gass based on biomicroscopic examination: stage 1: impending macular hole; stage 2: small hole; stage 3: large hole; stage 4: full thickness macular hole with PVD [3]. Now, with OCT, the staging is redefined: stage 1 is now called vitreomacular traction; stage 2 is a small or medium macular hole with vitreomacular traction (Fig. 1.7); stage 3 is a medium or large macular hole with vitreomacular traction; stage 4 is a macular hole without vitreomacular traction (Fig. 1.8) [2].

OCT can help measuring the diameter of macular hole. The diameter is not consistent at different layers. It is most narrow in the middle. Therefore, usually two diameters are measured, the minimal diameter and the basal diameter. The diameter of macular hole usually ranges from 50–1000 μm. Furthermore, OCT demonstrates the detail morphological changes of macular hole. There are usually some intraretinal cysts around the hole.

OCT can also help differential diagnosis of macular hole. There are some cases with pseudo macular hole, which is actually caused by epiretinal membrane that elevated retina around fovea without interruption of neural retinal tissue (Figs. 1.9 and 1.10). There are also some cases with lamellar macular hole, with interruption of inner layers, but not the full thickness of retina.

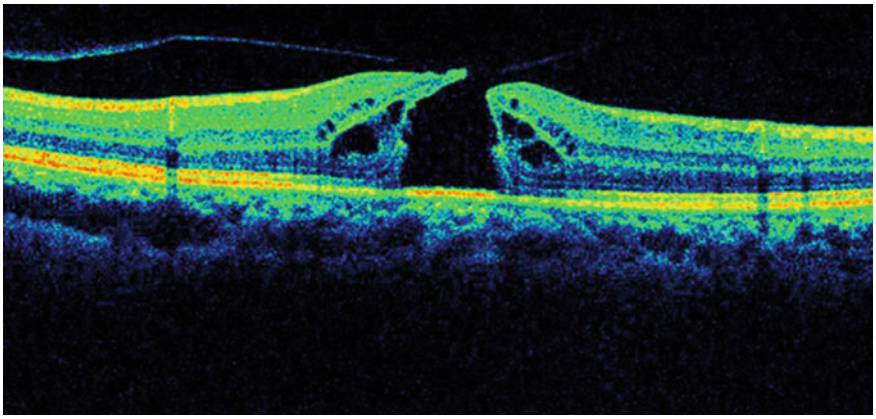

**Fig. 1.7** Small full thickness macular hole with vitreomacular traction. The OCT image shows attachment of the vitreous to the lid of the hole and cystic changes

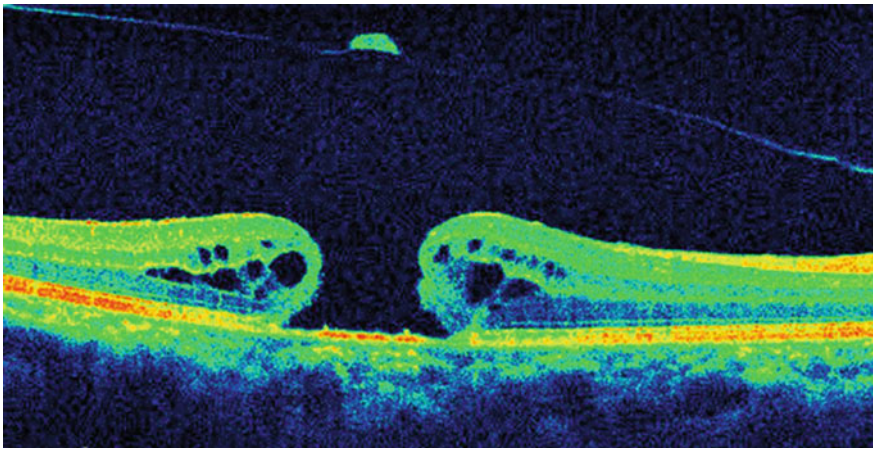

**Fig. 1.8** Large full thickness macular hole without vitreomacular traction. The OCT image shows a full-thickness macular hole with intraretinal cystic spaces and an overlying operculum

OCT is also helpful in following up of macular hole after surgical repair. It shows that the inner layer of macular hole connects with each other first. Repairing of the outer retinal layers disruption takes a longer time which explains the prolonged visual disturbance after surgery. The morphology of repaired macular hole can be categorized into three patterns, U type (normal foveal contour), V type (steep foveal contour) and W type (foveal defect of neurosensory retina). This classification system correlates with visual recovery [4].

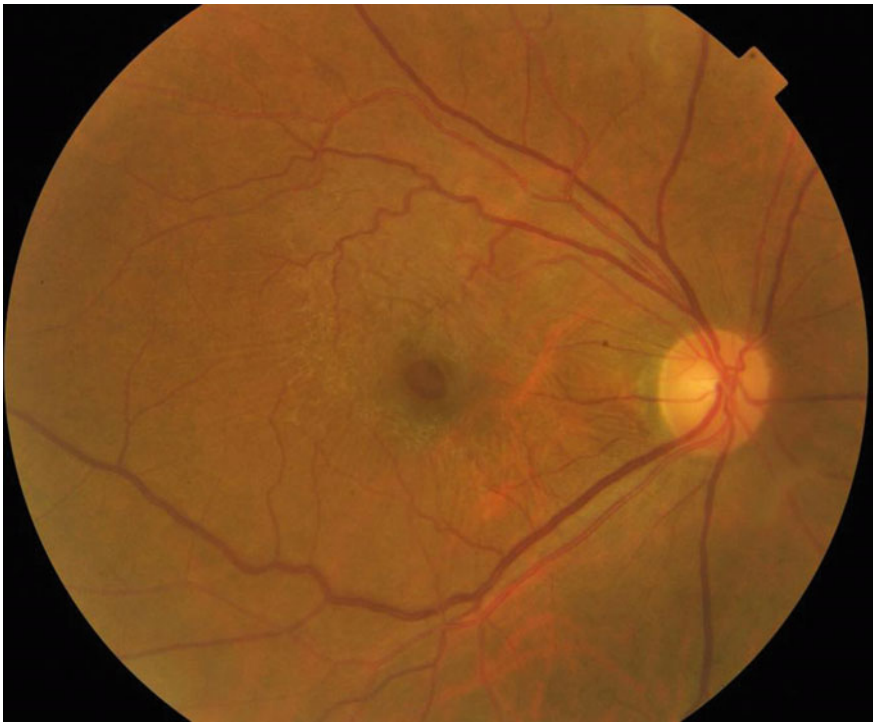

**Fig. 1.9** Fundus photography demonstrates fibrocellular proliferation on macular region except fovea. It appears like a macular hole

### *1.2.4 Epiretinal Membrane*

Epiretinal membrane (ERM) is due to proliferation of fibrocellular tissue on the inner surface of retina (Fig. 1.9). On OCT, it is characterized by medium to high reflective lines above neural retina. There are some adhesions of the epiretinal membrane to the inner surface of retina in multiple locations. Some adhesions may be wide and some may be focal. Sometimes in early stages of epiretinal membrane, the adhesion maybe very wide and the deadhesion area may not be identifiable. This is characterized by flattening of the retina inner surface (Fig. 1.10).

OCT is used not only for diagnosing epiretinal membrane, but also help planning for operation. From OCT images, the surgeon can identify the region where the distance between ERM and ILM is maximum which is the best location for initiation of membrane grasping to avoid damage of retinal tissue [5].

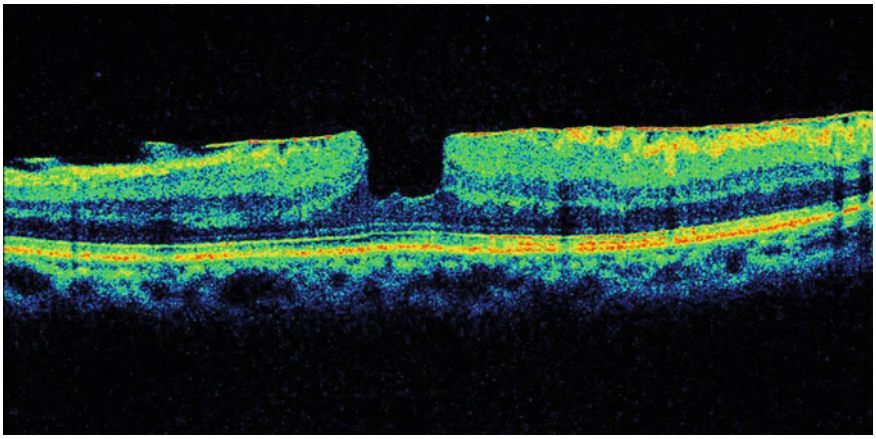

**Fig. 1.10** OCT of the case in Fig. 1.9 shows high reflectivity anterior to the retina. Retinal inner surface is flattened and the retinal thickness is increased. There is no disruption of retina tissue and the fovea appear as a macular pseudohole

### *1.2.5 Myopic Traction Maculopathy*

The pathogenesis of high myopia is axial elongation of the eyeball. The sclera remodels and elongates, however, the retina tissue and choroid do not elongate equivalently with the sclera, especially the internal limiting membrane and the retinal vessels [6].

The morphological characters of myopic traction maculopathy include the following:

1. Vitreous adhesion and traction at vitreomacular interface. The adhesions are not limited to the fovea, but also at other locations, especially large vessels.
2. Retinoschisis, which is the separation of retinal layers, caused by the traction force on the inner retinal surface.
3. Disruption of photoreceptor, which may manifest as "outer lamellar hole".
4. Localized retinal detachment at fovea, without full thickness macular hole (Fig. 1.11).
5. Full thickness macular hole with or without retinal detachment.

## 1.3 Glaucoma and Optic Neuropathy

Glaucoma is a group of ocular disorders characterized by progressive degeneration of optic nerve. OCT provides early diagnosis and monitoring of the disease progression [7]. Because retina is an extension of the central nervous system (CNS), many CNS diseases may manifest in the retina, where the optic nerve head is located.

There are several different commercially available OCT devices providing different scans and analyses modules. Generally, there are three important measurements

in retinal OCT for glaucoma and optic neuropathy, including parapapillary retinal nerve fiber layer thickness, retinal ganglion cells complex layer thickness, and morphology of optic nerve head.

### *1.3.1 Parapapillary Retinal Nerve Fiber Layer Thickness*

Retinal nerve fibers are the axons of retinal ganglion cells which make up the retinal nerve fiber layer (RNFL) lying just below the internal limiting membrane. RNFL has high reflectivity and can be easily identified and segmented on OCT images. The nerve fibers exit the eyeball and connect to the brain via optic nerve. Therefore, RNFL thickness is the highest at the parapapillary regions. Typically, OCT provides a cube scan centered on the optic nerve head and produces a RNFL thickness map. The RNFL thickness at the 3.4 mm diameter circle is calculated at each pixel. The average thickness at each clock hour and quadrant are also calculated. The results have been compared to the distribution of normal database and displayed as false color scale. The symmetry of RNFL thickness has been calculated [8]. In early stage of glaucoma, the RNFL reduced mostly on the superotemporal and inferotemporal bundles, but the late stage, RNFL thinning involves the entire region (Fig. 1.12).

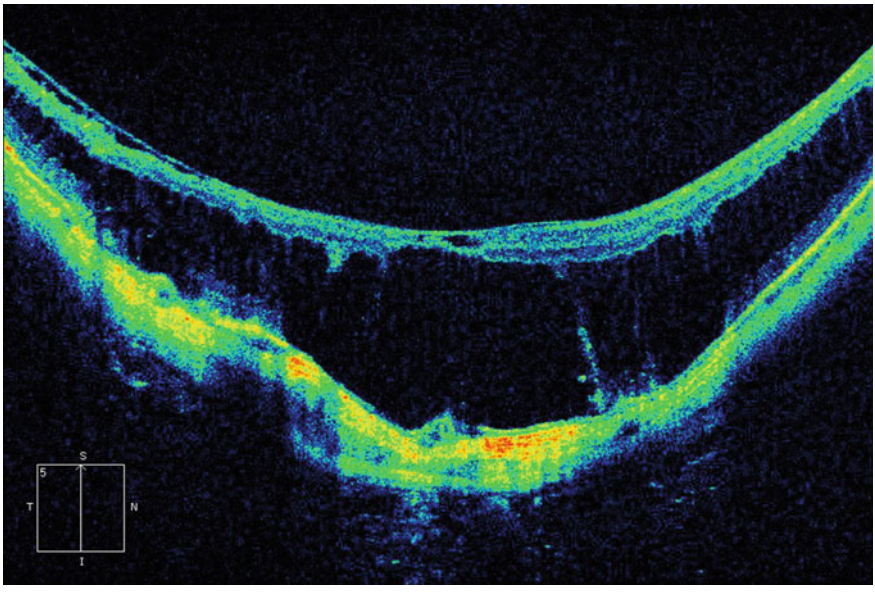


**Fig. 1.11** OCT of a patient with high myopic maculopathy. There is vitreomacular interface traction, retinoschisis, disruption of photoreceptor

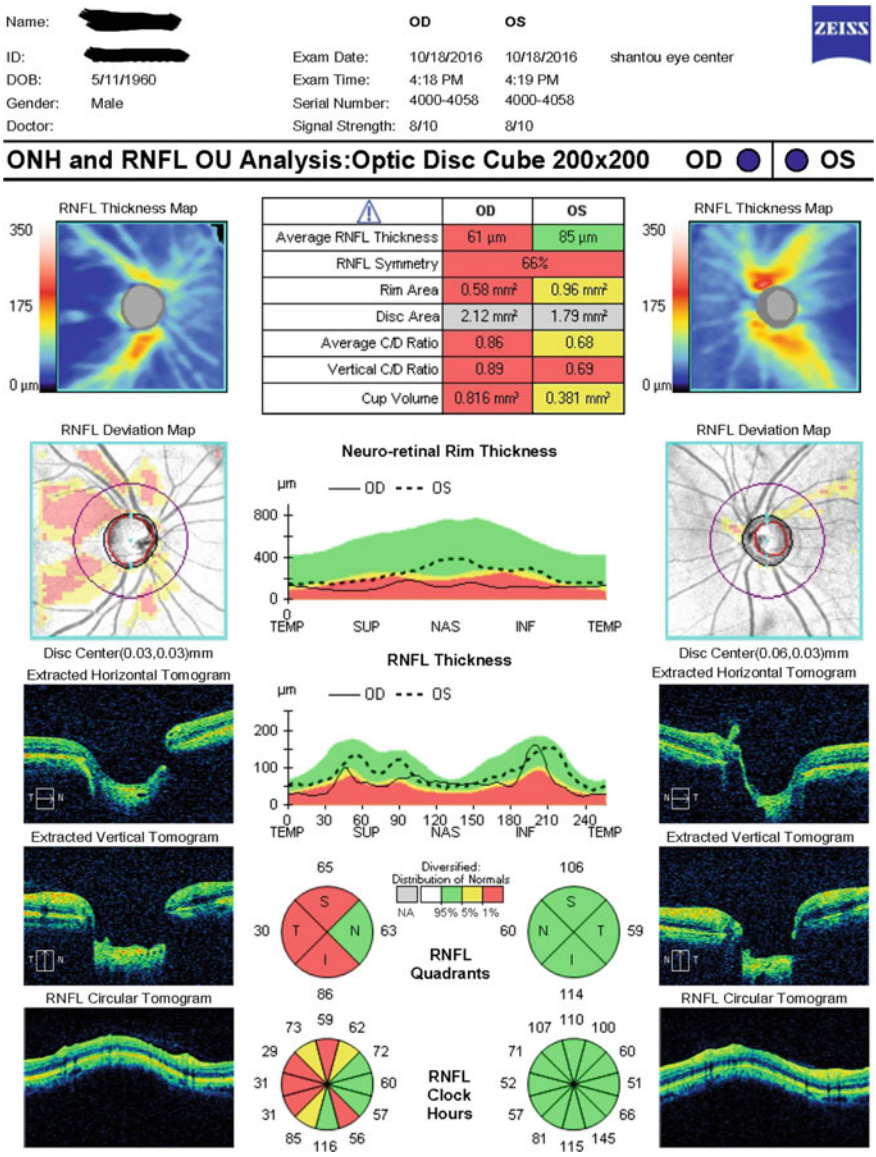


**Fig. 1.12** Parapapillary retinal nerve fiber layer thickness and optic nerve head analysis of a case with glaucoma

### *1.3.2 Macular Ganglion Cell Thickness*

The ganglion cell layer appears as a relatively hyporeflective layer between the RNFL and inner plexiform layer on OCT. It is the thickest at the macular region. However, it is difficult to segment ganglion cell layers based on reflectivity. Therefore, it was measured with other layer(s) together. In Zeiss Cirrus OCT, the Ganglion Cell Analysis consists of ganglion cell layer and inner plexiform layer. In Optovue OCT, the Ganglion cell complex include RNFL, GCL and IPL. Topcon OCT provides macular RNFL, GCL+IPL, and NFL+GCL+IPL thickness measurements. The scan region is divided into various sectors. The macular ganglion cell thickness map is displayed using similar color scale which are compared to normal database. In glaucoma patients, the ganglion cell thickness is reduced [9] (Fig. 1.13).

### *1.3.3 Optic Nerve Head Morphology*

The morphology of optic nerve head is an important parameter for analyses of the amount of optic nerve damage using biomicroscope. In OCT, the borders of optic disc and optic cup can be automatically segmented in a similar way to the evaluations performed by ophthalmologists. The border of optic disc is defined as the termination of Bruch's membranes. The software can then calculate the disc area, cup area, rim area, average cup to disc ratio, vertical cup to disc ratio. These parameters are then compared to a normative database [10]. In patient with glaucoma, the optic cup/disc ratio is increased and the neuro-retinal rim thickness is reduced (Fig. 1.12).

## 1.4 Retinal Vascular Diseases

Retinal vascular diseases include retinal artery occlusion, retinal vein occlusion, retinal vasculitis, diabetic retinopathy, and others.

### *1.4.1 Retinal Artery Occlusion*

Retinal artery occlusion is the sudden blockage of retinal blood supply. It can be further classified into central retinal artery occlusion and branch retinal artery occlusion. On fundus photography, retinal artery occlusion is characterized by whitening and opacity of retina, which is transparent in physiological condition. The fovea lacks inner retinal tissue and therefore, cannot be affected by the retinal artery occlusion. The fovea remains red but surrounded by the white opacity retina, which is known as "cherry red spot" (Fig. 1.14). On OCT, retinal thickness increases at acute stages

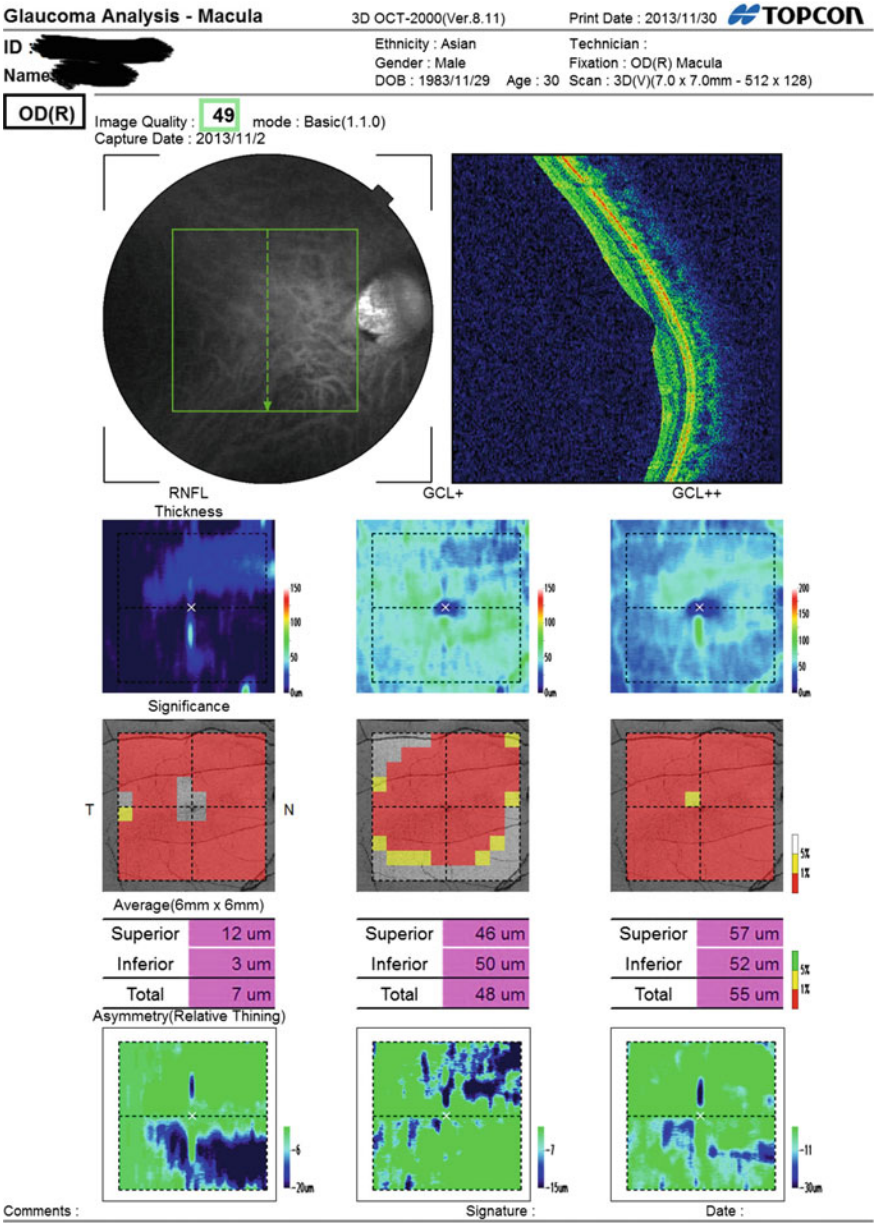


**Fig. 1.13** Macular ganglion cell complex thickness analysis in a patient with primary open angle glaucoma

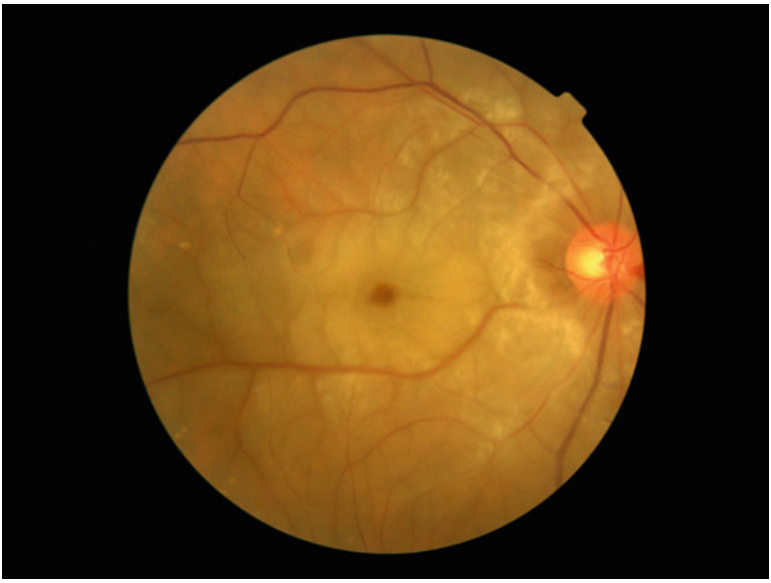

**Fig. 1.14** Fundus photography of a CRAO case with a 'cherry-red spot' at the macula

and then reduces after regression of the whitening, mostly on the inner retinal layers [11]. Besides change of retinal thickness, the optical intensity of inner retinal layers increase, which suggest retinal ischemia, while the optical intensity of outer retinal layers decrease, which is caused by the shadowing effect of inner retinal layers (Fig. 1.15) [12]. Further study suggested that the optical intensity ratio of inner and outer layers is correlated with the visual outcome in patients with central retinal artery occlusion [13].

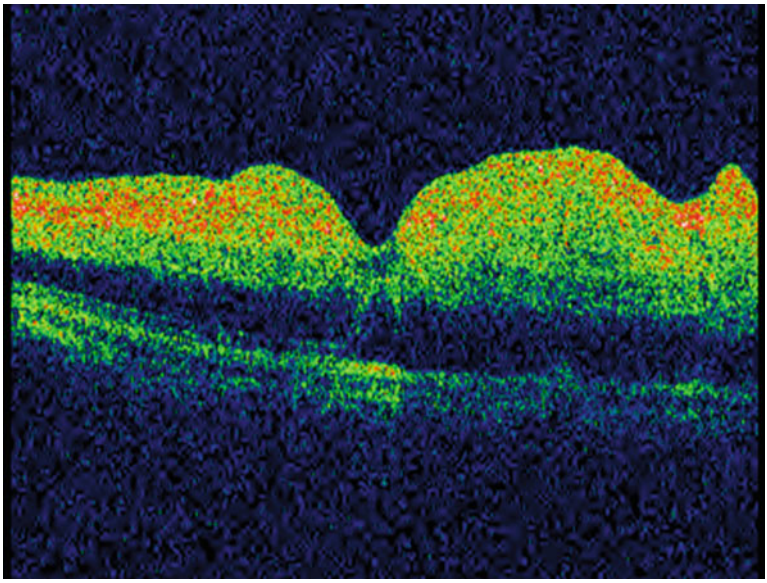

**Fig. 1.15** OCT of the same eye as Fig. 1.14 shows diffuse retina thickening, high reflectivity at inner retina and reduced reflectivity at photoreceptor and RPE

### *1.4.2 Diabetic Retinopathy*

Diabetic retinopathy is the most common cause of vision loss among people with diabetes and a leading cause of blindness among working-age adults. It is caused by damage to microvascular endothelial cells. The clinical manifestations of diabetic retinopathy include retinal hemorrhage, hard exudates, cotton wool spots and macular edema (Fig. 1.16).

On OCT, cotton wool spots are localized hyperreflectivity at retinal inner layers. Its appearance suggests retinal ischemia. Hard exudates are also hyperreflective spots, but located at deeper layers. They are caused by extracellular lipid which has leaked from abnormal retinal capillaries.

Macular edema is accumulation of fluid in macular region. Clinically, macular edema is defined as thickening of macular retina. On OCT, there are three types of macular edema, serous retinal detachment, intraretinal cysts and diffuse retinal thickening. Most macular edema are the combination of these characters (Fig. 1.17). Besides the cross-sectional morphology, OCT also provides quantitative measurement of retinal thickness at macula which is divided into 9 ETDRS regions (Fig. 1.18). The retinal thickness is useful for monitoring the progress of macular edema and its response to treatment [14].

### *1.4.3 Retinal Vein Occlusion*

Retinal vein occlusion may involve either the central or branch retinal veins. The fundal manifestation of retinal vein occlusion also includes retinal hemorrhage, cotton wool spots, retinal hard exudates, macular edema. The OCT appearances are similar to those of diabetic retinopathy (Figs. 1.19 and 1.20).

## 1.5 Outer Retinal Degenerative Diseases

Outer retinal layers include outer nuclear layer, photoreceptor inner and outer segment, retinal pigment epithelium. Degeneration of outer retina can be caused by genetic mutation, or secondary to other factors, including traumatic impact, retinal detachment, inflammation, toxicity, or age related macular degeneration.

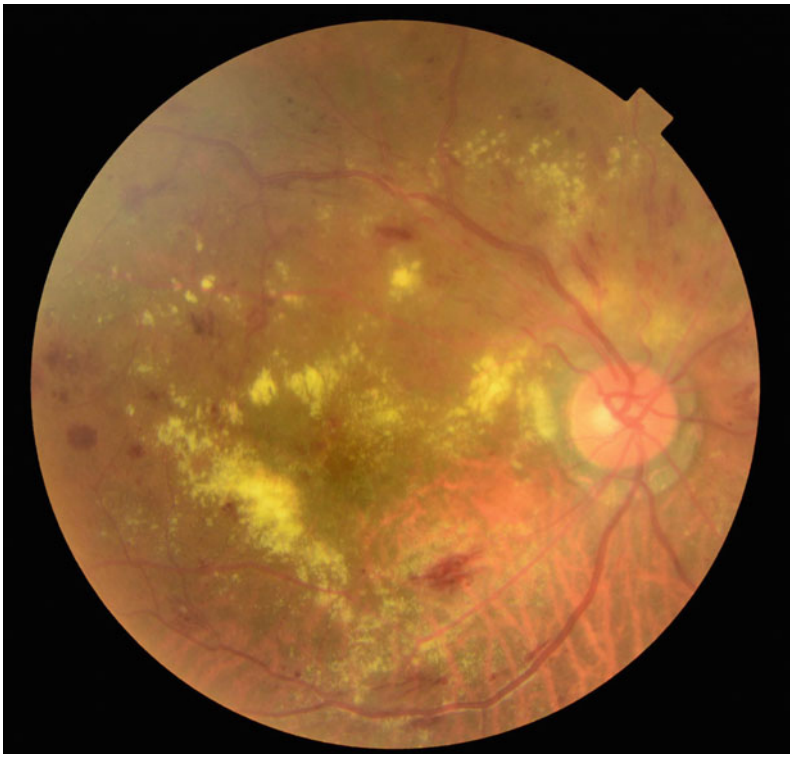

**Fig. 1.16** Fundus photography of a patient with diabetic retinopathy showing extensive hard exudates around macular fovea and scattered dot hemorrhages

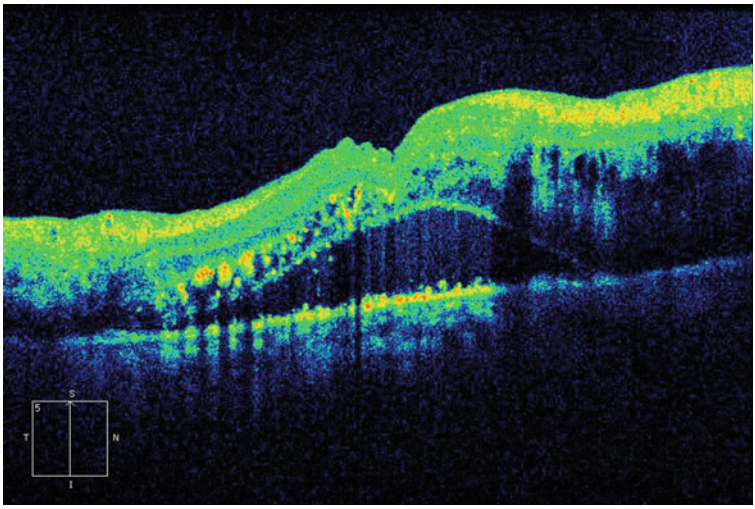

**Fig. 1.17** OCT of the same eye as Fig. 1.16 shows high reflectivity spots at the intra neuroepithelial layer corresponding with extensive hard exudates, intraretinal cysts and serous retinal detachment

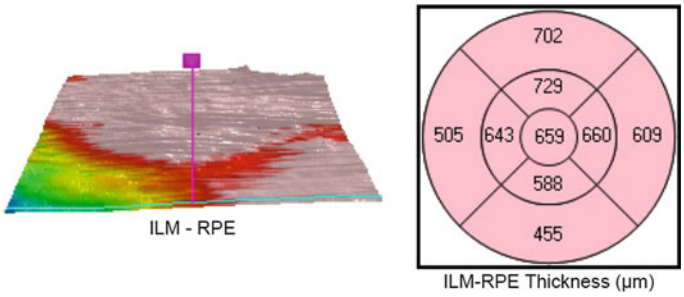


**Fig. 1.18** Retinal thickness map (left) and the average retinal thickness at 9 ETDRS regions

Degeneration of photoreceptor is the major character of outer retinal diseases. Photoreceptor plays a critical role in vision. Therefore, degeneration of photoreceptor will lead to visual impairment. On OCT, disruption of photoreceptor inner segment ellipsoid zone is correlated with visual loss. Furthermore, retinal thickness would reduce, especially in outer retinal layers (Figs. 1.21 and 1.22) [15].

On fundoscopy, RPE changes are characterized by focal areas of mobilization of pigment (hypopigmentation and hyperpigmentation). On OCT, it appears as RPE deformation or thickening that may form irregularities which may progress to RPE atrophy in some cases. On OCT, in addition to the loss of RPE layer, the optical intensity of choroid would increase due to loss of shadowing effect of RPE (Figs. 1.21 and 1.22) [16].

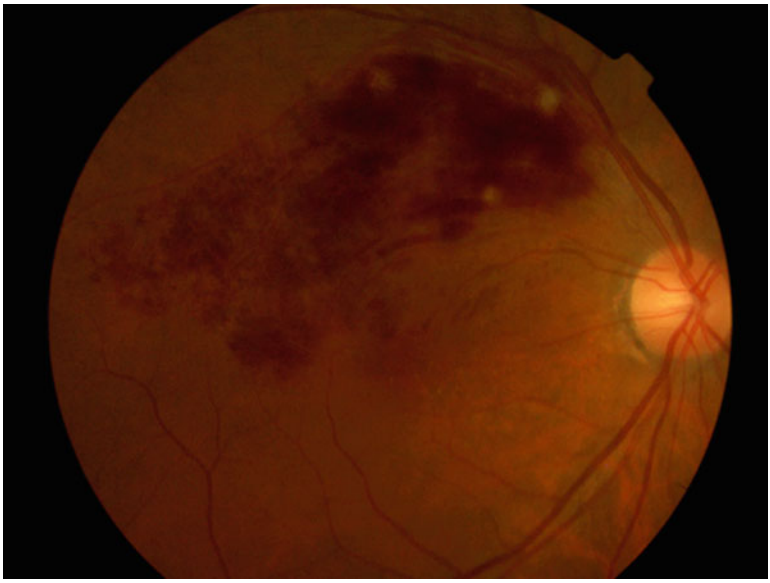

**Fig. 1.19** Fundus photography of branch retinal vein occlusion shows flame-shaped and blot hemorrhages, cotton wool spots and venous tortuosity at superotemporal retina

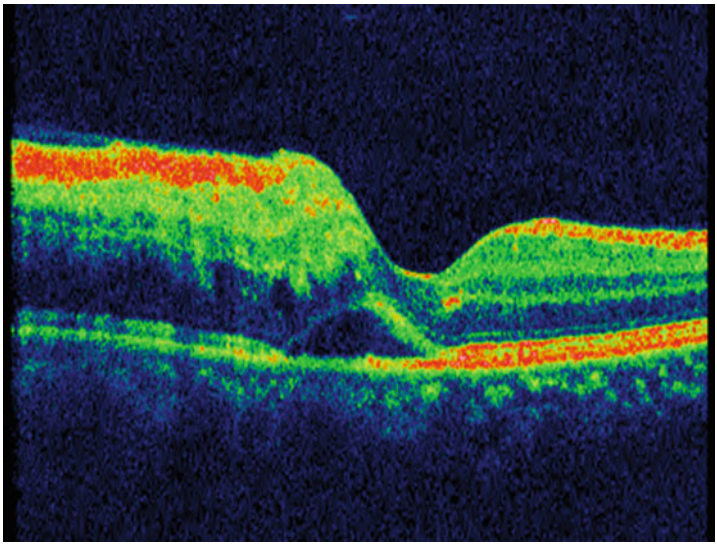

**Fig. 1.20** OCT of the same eye as (Fig. 1.18) shows retinal thickening, inner retinal high reflectivity and serous retinal detachment at fovea

Drusen is degenerative nodular formations located in Bruch's membrane and beneath RPE. It consists of proteins, lipids, mucopolysaccharides, and other components. It is a landmark character of age-related maculopathy. On OCT, it is characterized by sub-RPE nodules with low or middle reflectivity (Figs. 1.21 and 1.22) [17].

## 1.6 Choroidal Neovascularization and Polypoidal Choroidal Vasculopathy

Choroidal neovascularization is the growth of new blood vessels from choroid vasculature through a break of the Bruch's membrane into sub-retinal or sub-RPE space. The etiology of choroidal neovascularization includes age related macular degeneration (AMD), high myopia, ocular inflammation and ocular trauma. Age related macular degeneration is a leading cause of irreversible blindness in elderly patients. It is usually located at macula and can cause central scotoma and severe visual loss. The choroidal neovascularization would lead to hemorrhage, exudation, and fibrosis. It can be further classified into type 1, type 2 and type 3. Type 1 neovascularization is also called occult. It refers to the neovascular tissue under RPE. Type 2 neovascularization refers to the neovascular tissues that break through RPE and grow into subretinal space. Type 3 neovascularization is also called retinal angiomatous proliferation and characterized by retinal-choroidal anastomosis [18].

On OCT, choroidal neovascularization is demonstrated as moderate to high reflective lesion located under or in front of RPE. It is usually accompanied by subretinal

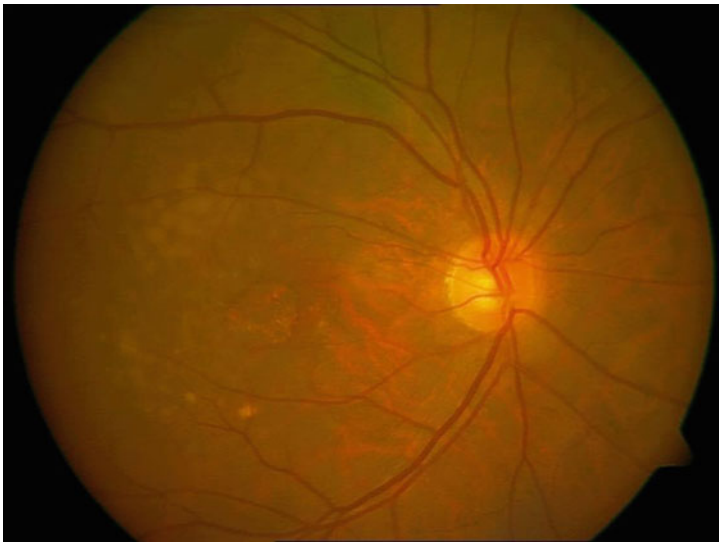

**Fig. 1.21** Fundus photography shows geographic atrophy of retinal pigment epithelium at fovea and some soft drusen at perifoveal region

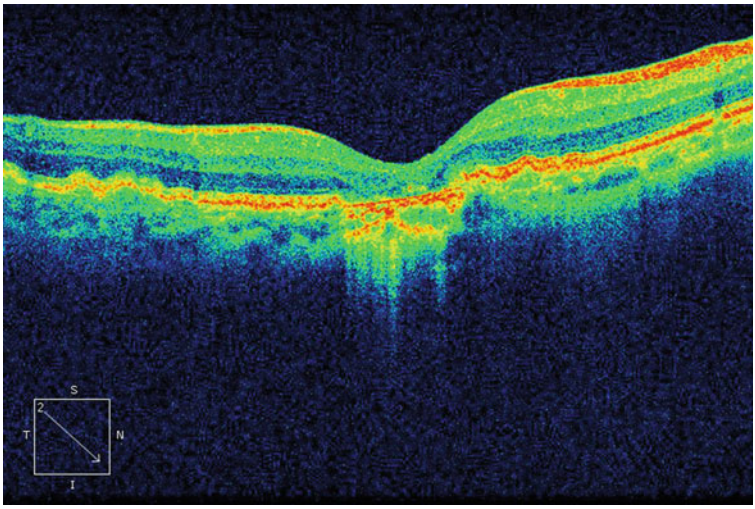


**Fig. 1.22** OCT image shows defect of retinal pigment epithelium at fovea. Please note that the reflectivity of choroid increase because of reduced shadowing effect of RPE. Photoreceptor inner segment ellipsoid zone disruption and atrophy of outer nuclear layer can be found in the region of geographic atrophy. At temporal perifoveal region, RPE is elevated and there is mid-reflective material under RPE. These are drusen

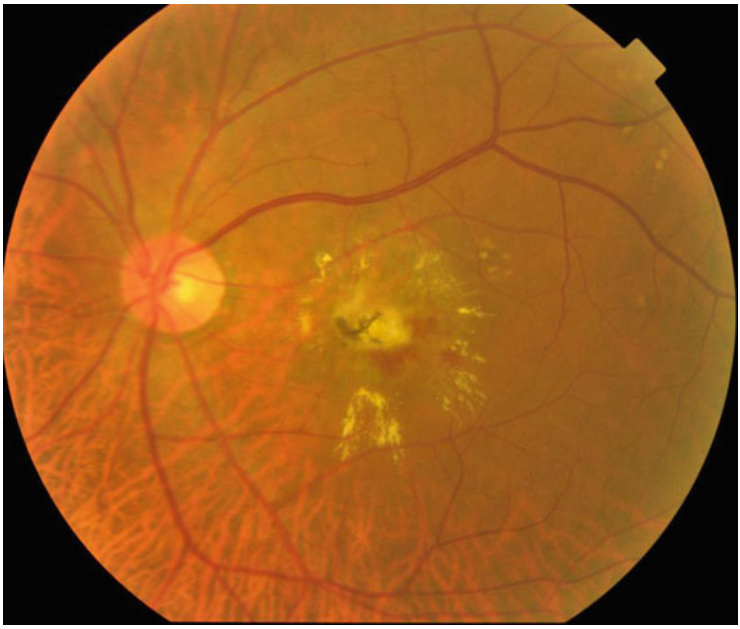

**Fig. 1.23** An subretinal fibrovascular lesion in macula surrounded by some intraretinal hard exudate

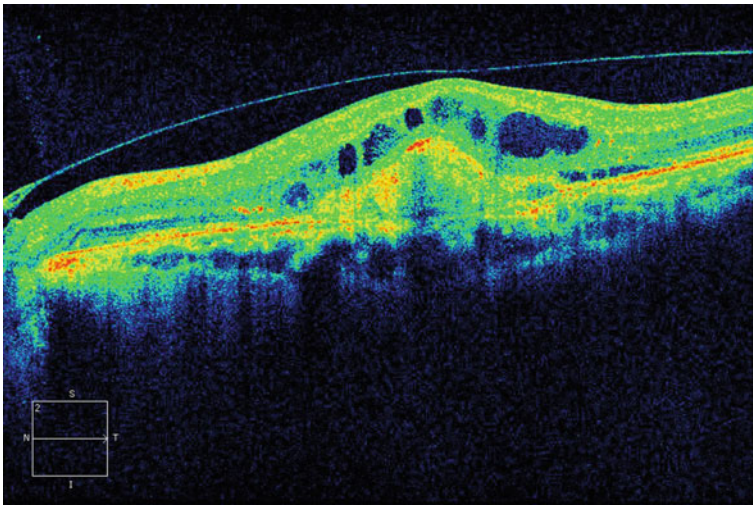


**Fig. 1.24** OCT of the same eye as Fig. 1.23 showing a sub retina high reflectivity mass with subretinal and intraretinal fluid

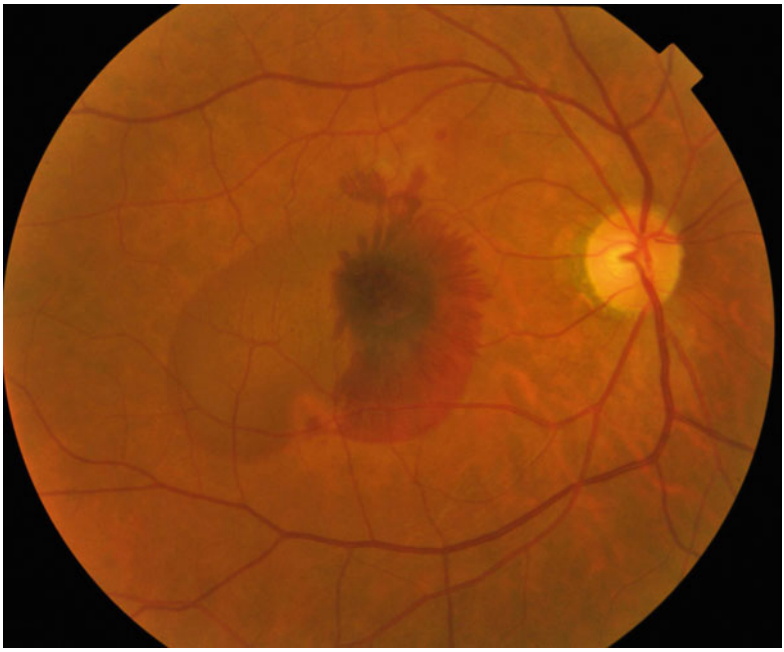

**Fig. 1.25** Serosanguineous retinal pigment epithelial detachment with associated subretinal hemorrhage

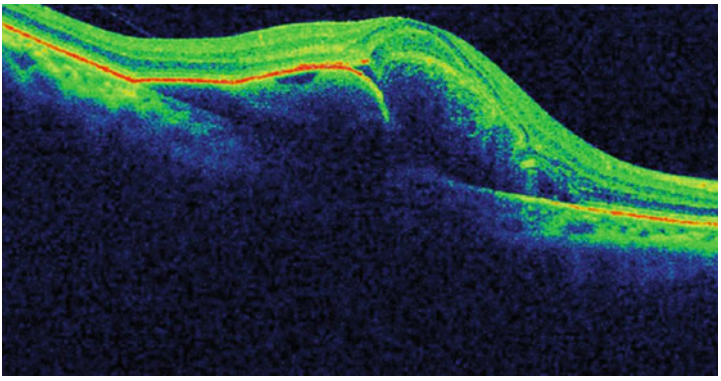

**Fig. 1.26** OCT of the same eye as Fig. 1.25 showing sub-retinal hemorrhage and retina pigment epithelial detachment

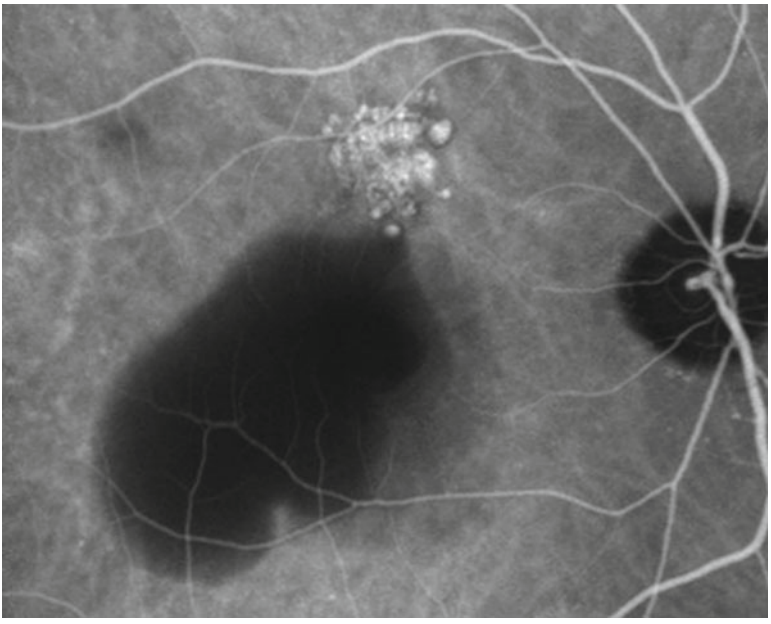

**Fig. 1.27** The same eye as Fig. 1.25 on ICGA image showing hyperfluorescence due to polyps, branching vascular network and subretinal hemorrhage

or sub-RPE hemorrhage or fluid. In the late stage, choroidal neovascularization may progress to fibrosis and appear as hyperreflective lesion. The leaky neovascularization can cause intraretinal cysts which appear as non-reflective cysts in retina (Figs. 1.23 and 1.24). The thickness of macula can also be measured at the 9 ETDRS regions which are important parameters to monitor the prognosis of disease or response to therapy [18].

Polypoidal choroidal vasculopathy (PCV) is characterized by a branching vascular network from choroidal vasculature with polypoidal lesions under RPE. It is still controversial whether PCV is a subtype of AMD or a distinct disease. On OCT, pigment epithelium detachment, double-layer sign, and thumb-like polyps are more common in PCV eyes than in AMD eyes [19] (Figs. 1.25, 1.26 and 1.27).

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