Retina

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The retina is a layer of transparent membrane lining the posterior part of the eyeball wall, whose outer surface is close to the choroid and internal surface is attached to the vitreous.

1.1 Retinal Imaging

Light is projected onto the retina through the cornea, aqueous humor, pupil, lens, and vitreous. Retinal imaging is similar to the pinhole imaging principle, which states that if you put a plate with a pinhole between a screen and an object, an inverted image of the object will be formed on the screen and the size of the image will change with the back-and-forth movement of the plate in the middle. The pupils are equivalent to the pinhole on the plate and the retina to the screen. Since light travels along a straight line, after penetrating through the pupil, the light from above will be projected onto the inferior retina. Similarly, the light from below will be projected onto the superior retina, and the light from the temporal side will be projected onto the nasal retina, while the light from the nasal side will be projected onto the temporal retina. The principle is similar to that of a camera [1, 2].

1.2 Relationship Between Anatomy and Diseases of Retina

The retina develops from the optic cup formed by neuroectoderm in the embryonic stage. The outer layer of the optic cup forms the single retinal pigment epithelium, while the inner layer of the optic cup differentiates into the retinal neurosensory layer, which is again histologically divided into nine layers from outside to inside, including photoreceptor cell layer of cone cells and rod cells, external limiting membrane, outer nuclear layer, outer plexiform layer, inner nuclear layer, inner plexiform layer, ganglion cell layer, nerve fiber layer, and internal limiting membrane. A potential gap exists between the retinal pigment epithelium and the retinal neurosensory layers. In general, retinal detachment refers to the detachment between these two layers. Modern optical coherence tomography (OCT) can now display clearly the individual layers of the retina in vivo (Fig. 1.1).

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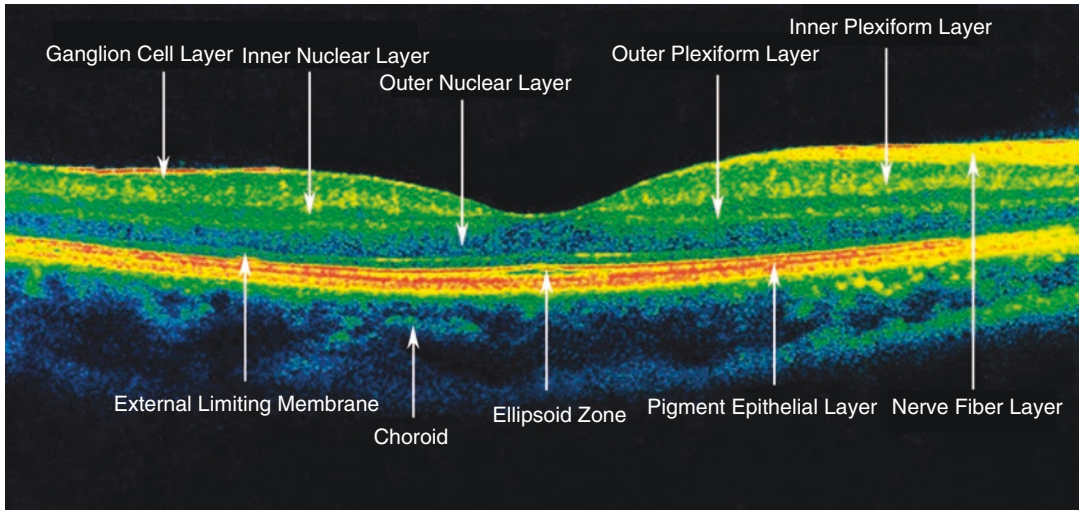

Fig. 1.1 OCT scanning image of the macula

The retina is like a cup with the rim at the ora serrata, which is located at the equator of the eyeball. There is a small shallow funnel-like sunken area about 2 mm in diameter in the posterior pole. It is called macula, which gains the name for rich lutein in this area. In its center, there is a small fovea called macular fovea, which, as the part with the most sensitive vision on the retina, mainly corresponds to the central visual field. If the macular area is impaired, mainly the central visual field will be damaged. The retina can be divided into nasal and temporal halves and superior and inferior halves by a hypothetical vertical line and horizontal line across the central fovea, respectively. The optic papilla is located at the supranasal quadrant above the horizontal line, which manifests as the physiological blind spot in visual field, because there is no photoreceptor cell here. Therefore, the physiological blind spot is situated at the inferotemporal quadrant below the horizontal line in the central visual field [3].

The diseases in different parts of the retina will result in the different visual field impairments in the reverse directions. For example, the retinal detachment in the supratemporal quadrant will lead to an inferonasal visual field defect. Age-related macular degeneration or central serous chorioretinopathy will cause central scotomas. The visual field impairments caused by retinochoroiditis, diabetic retinopathy, etc. are usually relative and multifocal, with variegated appearance of the whole visual field. The visual field defect resulting from retinal detachment is usually located in the peripheral part. In degenerative diseases, such as retinitis pigmentosa, the defect is ringlike, which is located in the mid-peripheral visual field at first and will gradually contract concentrically into tubular visual field. The degree of visual field impairment is also related to the degree of retinal tissue damage caused by a lesion. For example, as to retinal vascular occlusion, the arterial occlusion without timely treatments will produce typical absolute visual field impairment, whereas the venous occlusion will have relatively mild and variant visual field impairment.

Generally speaking, visual field defects simply caused by ocular diseases are usually fundus lesions (mainly the retina and/or optic nerve), except the impact of refracting media. Subtle changes of the central visual field sometimes can be felt by a patient with good central vision, but it is difficult to detect the corresponding fundus lesion under an ophthalmoscope. Auxiliary examinations, such as Amsler chart, OCT, fluorescein fundus angiography (FFA), electroretinogram (ERG), and visual evoked potential (VEP), will be required in such situation, and the retinopathy and/or

optic neuropathy corresponding to the visual field defect can usually be found. Consequently, the retinopathy and optic neuropathy should be screened firstly when a visual field defect is observed, especially a monocular defect, and then the intracranial lesions. Conversely, a binocular visual field defect indicating an intracranial lesion also necessitates careful screening of the retinopathy or optic neuropathy in additional to visual pathway examinations, because sometimes both of them may coexist.

1.3 Retinal Nerve Fiber Layer and Glaucoma

Retinal nerve fibers, or retinal ganglion cell axons, can be divided into papillomacular bundles, temporal arcuate fibers, and nasal radial fibers (Fig. 1.2). The numbers of retinal nerve fibers sent out from the respective parts of the retina are not the same. The retinal nerve fibers sent out from the macula, which are 65% of the total retinal fibers in quantity, constitute the papillomacular bundles. They enter into the temporal side of the optic disc in a more central and straighter course and correspond to the central 5° of the visual field. A central scotoma can be observed when damage occurs in these bundles. The retinal nerve fibers sent out from the nasal half of the retina are the second most and relatively sparse. Wedge-shaped, fan-shaped, or half-side visual field defects connecting with the physiological blind spot may appear when damage occurs in these fibers. The retinal nerve fibers sent out from the temporal half of the retina are the least. These fibers, which are originated from RGCs located at the temporal superior and inferior parts of the macula, are sent from the temporal retina without mixing at the horizontal raphe, bypass the macula and the papillomacular bundles arcuately, and enter the superior and inferior poles of the optic disc temporally. The fiber course is mainly in the area between 5° and 25° around the macula, where the visual field mainly corresponds to the Bjerrum area (the paracentral area, i.e., the area between 5° and 25° of the visual field) [3].

The typical damages caused by glaucoma mainly involve thinning of the retinal nerve fiber layer and excavation of the optic disc, with focal notching of the rim. The retinal nerve fiber layer is formed by the axons of RGCs. The typical visual field defect of glaucoma is pro-chiasm damage, a nerve fiber bundle defect. The superior and inferior retinal nerve fiber layers are thicker than the nasal and temporal ones. The nerve fiber bundles run through the lamina cribrosa of the sclera. The meshes in the upper and lower poles of the lamina cribrosa are relatively big, and the arcuate fibers from the temporal retina run through this

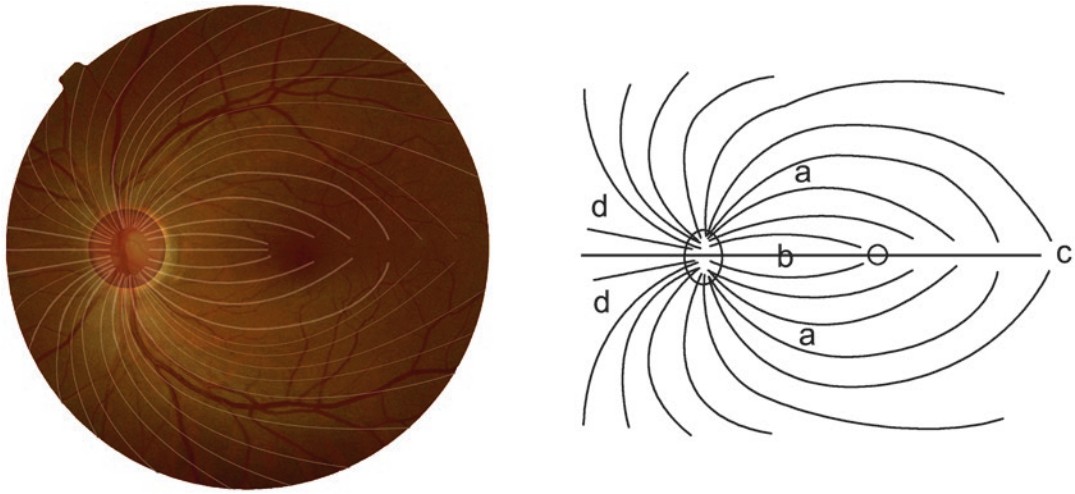

Fig. 1.2 Distribution of the central retinal nerve fibers. *a*: Superior and inferior arcuate fibers. *b*: Papillomacular bundles. *c*: Horizontal raphe. *d*: Nasal fibers

area. Furthermore, the lamellae forming these meshes are thin and fine, and the local connective tissue is relatively sparse, so these meshes are easily deformed when the intraocular pressure increases. Meanwhile, due to the absence of the support from connective tissue, the nerve fibers in these pores are susceptible to squeezing, which may lead to the disturbance or even interrupt of blood supply and axoplasmic transport, and then significant damage and corresponding visual field defect will appear. Nonetheless, the meshes at the nasal and temporal sides of the lamina cribrosa are smaller, and the lamellae are thicker and coarser with the relatively dense local connective tissue. The shear force resulting from the distortion of the lamina cribrosa and the dislocation of the meshes due to squeezed lamina cribrosa tissue under high intraocular pressure will lead to the axoplasmic flow blocking of the nerve fibers and then optic nerve damage of glaucoma.

The part of the lamina cribrosa the temporal nerve fibers run through is more susceptible to damage under high intraocular pressure due to lack of proper protection from the connective tissue. The typical visual field defects of early glaucoma are paracentral scotoma and nasal step in the Bjerrum area (the central 5° to 25° of the visual field) and correspond to the damages at the superior and inferior poles of the optic disc, enlargement of the vertical diameter of the optic cup and notch on the optic disc edge. Because the resistance to the high intraocular pressure in the meshes and the connective tissue of the part of the lamina cribrosa the radial nasal fibers and the papillomacular bundles run through is relatively strong, so the nerve fibers are not easy to be damaged at the early stage of glaucoma. This may be one of the mechanisms for the preservation of the central and temporal visual fields. It also explains why only the central tubular visual field and the temporal island of vision can be preserved in patients with advanced glaucoma.

As shown below, early glaucomatous damages can be found in the patient's right eye with a cup-disc ratio of 0.5 and the wedge-shaped defect of the inferior retinal nerve fibers (Fig. 1.3). The visual field impairments are nasal step, superior paracentral scotoma, and even small superior arcuate scotoma (Fig. 1.4). The OCT measurement reveals thinning of the inferior retinal nerve fiber layer (Fig. 1.5).

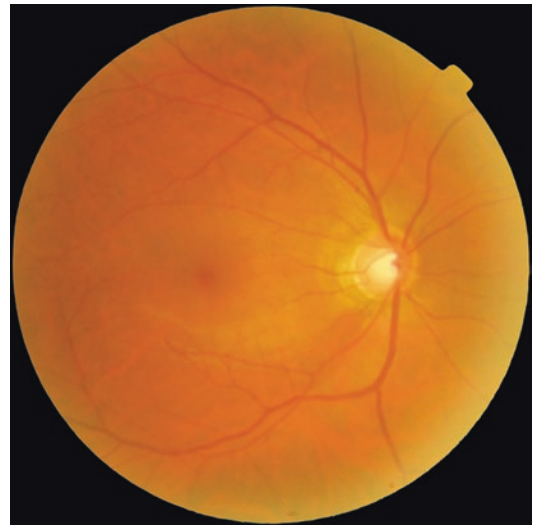

Fig. 1.3 The right fundus image of a glaucoma patient. The cup-disc ratio was 0.5. There's a wedge-shaped defect in the inferior retinal nerve fibers with narrowed cup rim

1.4 Retinal Blood Supply

The inner five retinal layers are mainly supplied by the central vascular system of the retina. The arterial and venous routes and distributions are roughly the same with no anastomotic branches. The outer five retinal layers are mainly supplied by the short posterior ciliary artery of the choroid vascular system. Both central retinal artery and short posterior ciliary artery are the branches of the ophthalmic artery which is a branch of the internal carotid artery. If there is any retinal vascular disease, it will cause corresponding tissue damage and function change (visual field impairment) [3, 4].

There is no retinal vessel in the center of the macula whose nutrition is mainly supplied by the choroidal vessels. Therefore, macula is relatively sensitive to choroidal vascular lesions. Age-related macular degeneration, commonly found in the senior population, is closely related to the changes in choroidal vessels. A series of changes

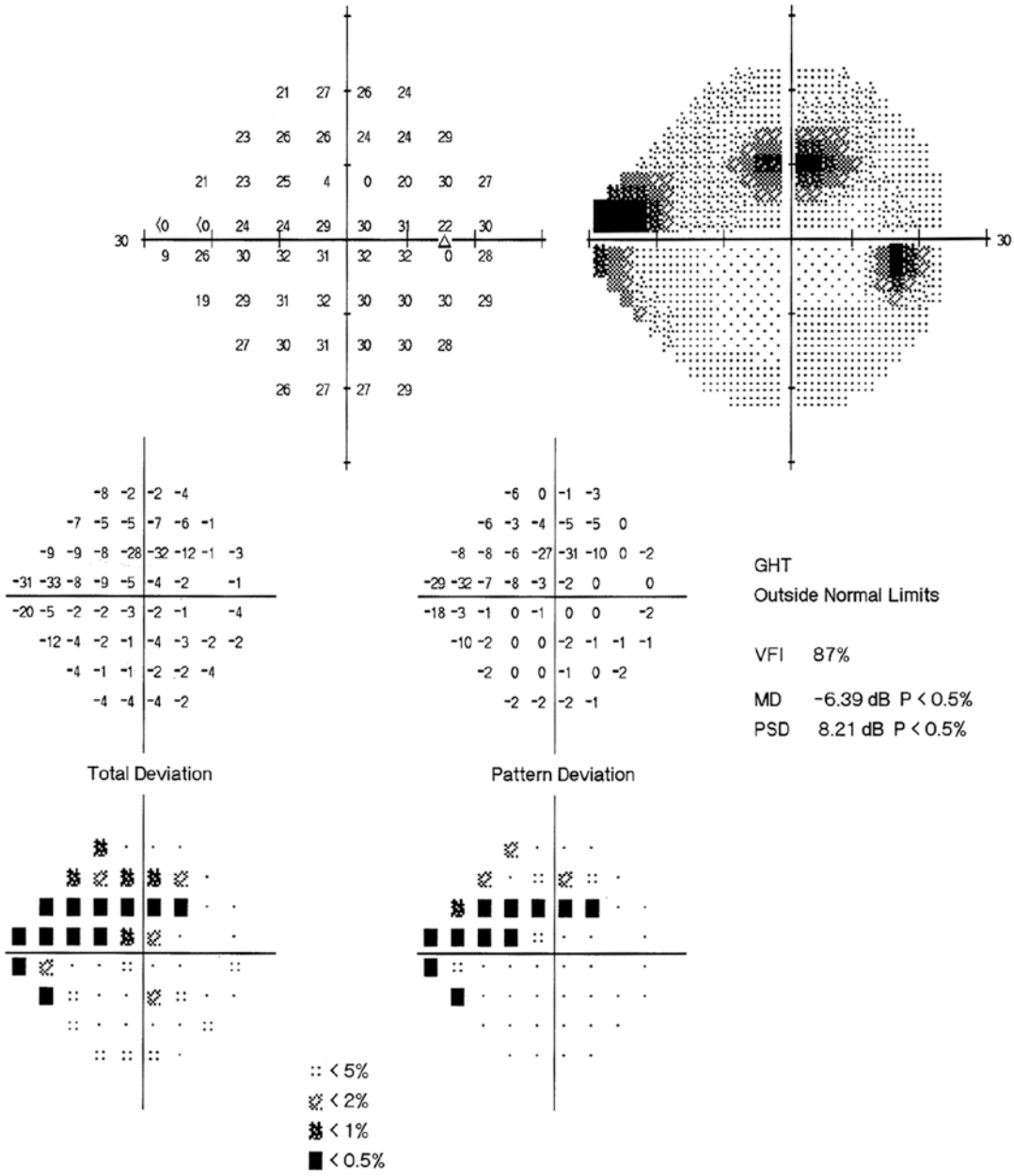

Fig. 1.4 Visual field defects of early glaucoma. Nasal step and a superior paracentral scotoma in the right eye

resulting from new vessels breaking through the retinal pigment epithelium layer into the inner retina can be found in such lesions.

The central retinal artery is one of the terminal arteries. The farthest distribution layers supplied by the capillaries of the central retinal artery are inner nuclear layer and inner plexiform layer. When the intraocular pressure becomes higher, the anatomical positions with the most serious ischemia should be the inner nuclear layer and the inner plexiform layer, and damages in these two layers can be found on an OCT scan. The area around the lamina cribrosa of the optic disc, including the lamina cribrosa and anterior area, is

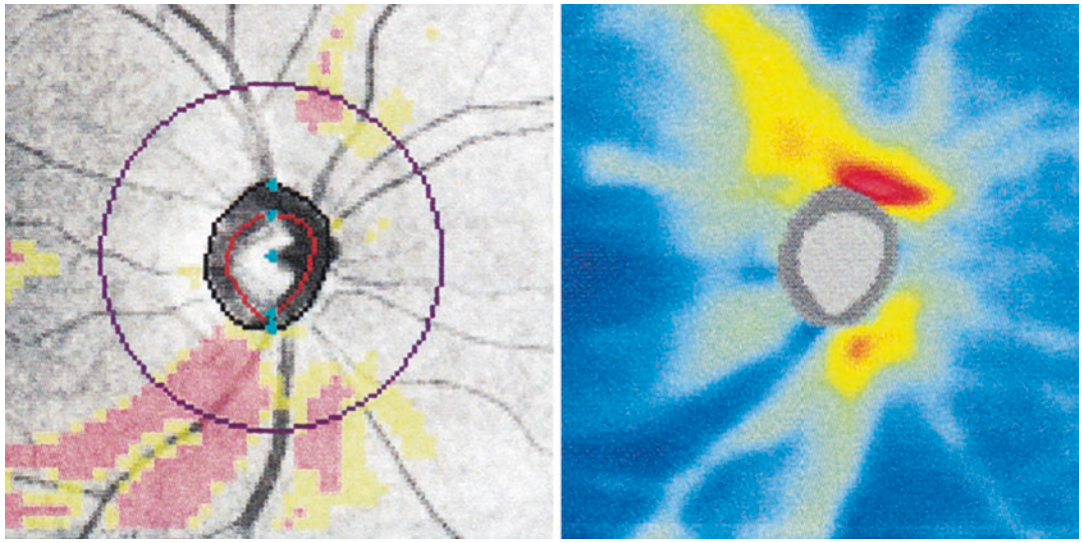

Fig. 1.5 OCT scan for the thickness of the nerve fiber layer around the optic disc in a glaucoma patient. Thinning of the inferior retinal nerve fiber layer in the right eye

supplied by 15–20 short posterior ciliary arteries. Ischemia of these arteries will cause hypoperfusion and vascular infarction of the anterior part of the optic nerve, which may lead to anterior ischemic optic neuropathy.

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