Myopic Macular Retinoschisis

16

Kyoko Ohno-Matsui

16.1 Myopic Retinoschisis and Associated Lesions

In 1997, Takano and Kishi first identified and reported that the foveal retinal detachment and macular retinoschisis (MRS) were observed in highly myopic eyes before developing macular hole retinal detachment by using OCT [1]. This finding provided a clue why macular holes in highly myopic eyes tend to develop retinal detachment unlike idiopathic macular holes in non-myopic eyes. The schisis was found to occur in the outer retinal layer unlike congenital macular retinoschisis; however, the morphological details of the separated retina were unclear at this point mainly due to a limited resolution of OCT [1–3]. With the advent of OCT, MRS has been increasingly recognized as important causes of vision decrease in eyes with pathologic myopia. MRS is found in 9–34 % of highly myopic eyes with posterior staphyloma [1, 2, 4].

Despite numerous publications on this condition, no clear definition of MRS has been found in the literature. The hallmark of MRS is the appearance of schisis of the retinal layers, most commonly in the outer plexiform layer (outer retinoschisis) (Fig. 16.1) [5–7]. In minority cases, the appearance of schisis may occur within more internal retinal layers (inner retinoschisis) with detachment of the internal limiting membrane (ILM) (Fig. 16.1) [7, 8]. Although the terminology "myopic retinoschisis" still prevails, it is now believed (due in part to improved imaging with recent OCT) that eyes with myopic retinoschisis have traction, resulting in elongation of Henle's nerve fiber rather than a splitting of the retina. Thus, MRS is a totally different condition from congenital macular retinoschisis, which is a splitting of the retinal nerve fiber layer from the rest of the sensory retina disrupting a synaptic transfer between bipolar cells and ganglion cells. A lack of central scotoma in eyes with MRS supports this concept.

Pannozzo and Mercanti [4] proposed to unify all of the pathologic features generated by traction in the myopic environment under the name of myopic traction maculopathy.

16.2 Clinical Features of MRS

Most of the patients with MRS may be relatively asymptomatic especially while the eyes do not develop more serious complications like full-thickness MH or foveal RD [2], and MRS may persist for many years before affecting vision significantly. Vision loss attributed to MRS has been associated with the development of foveal RD and/or macular holes in most cases. The best-corrected visual acuity (BCVA) of the patients with MRS ranged widely from 20/40 to 20/200 [2, 9]. Some patients complain metamorphopsia or distorted vision before the visual acuity is decreased. However, what makes difficult for clinicians to suspect the MRS is that some of the patients with MRS do not notice the change in vision [2], due to coexisting myopic retinochoroidal lesions like myopic CNV, myopic chorioretinal atrophy, or myopic optic neuropathy. Thus, a periodic examination using OCT is recommended for highly myopic eyes with posterior staphyloma even when they do not recognize the change of visual symptoms.

The mean age of diagnosis of MRS in highly myopic patients was in 60s [2, 9] and it is uncommon to detect MRS in the patients under the age 40, although MRS in the patients with age 28 was reported [9]. Baba et al. [2] reported that the mean refractive error of the eyes with MRS was −18.4 D (range, −13.0 to −27.0) and the mean axial length was 29.8 mm (range, 28.6–32.2 mm). Similarly, Fujimoto et al. [5] reported that the axial lengths ranged from 26.8 to 34.2 mm (mean, 29.7 ± 2.0 mm).

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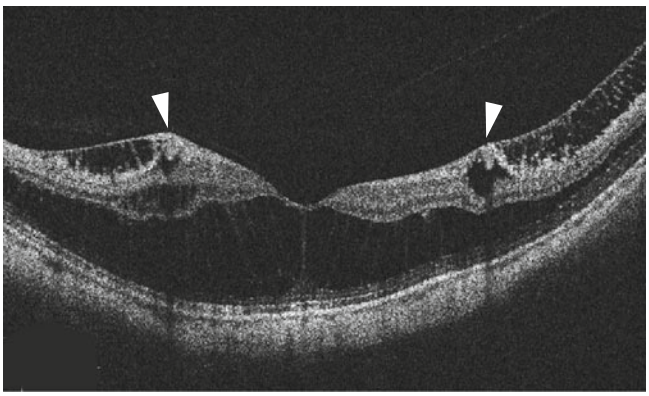


Fig. 16.1 Representative OCT images of myopic macular retinoschisis. The tissue splitting of outer retina is observed and many columnar structures are seen within the retinoschisis. The columnar structures are almost perpendicular to the line of retinal pigmented epithelium at the fovea and tend to be slightly inclined away from the fovea. The retinal blood vessel is protruded toward the vitreous like vascular microfolds (*arrowheads*). The posterior hyaloid is attached onto the retinal blood vessels, and the inner retinoschisis (ILM detachment) is seen around the retinal blood vessels. Paravascular retinal cysts are also observed around the retinal vessels

MRS almost exclusively develops in severely myopic eyes with posterior staphyloma and does not develop in eyes without staphyloma [2, 5].

16.3 Diagnosis of MRS

OCT is an indispensable tool to diagnose MRS and associated lesions, although the presence of MRS can be suspected ophthalmoscopically in some cases. In some cases, the MRS can be observed as a shallow retinal elevation by the stereoscopic fundus examinations using magnified lens (e.g., +90 D lens). Such retinal elevations are more easily detected along and within the patchy chorioretinal atrophy, along the retinal vascular arcade, and along the temporal margin of myopic conus.

By using SD-OCT, MRS is observed as the splitting of the inner retina from the outer retinal layers with multiple columnar structures connecting the split retinal layers (Fig. 16.1) [3, 5, 9]. Different from retinal detachment, the remnants of outer retina are observed on the RPE layer. On enhanced SD-OCT images, Fujimoto et al. [5] reported that the splitting of the outer retina appeared to be present between the outer plexiform layer and the outer nuclear layer. Columnar structures are almost perpendicular to the RPE at the fovea and tend to become inclined away from the fovea, which corresponds to the course of Henle's nerve fiber layer in the macula [10]. Thus, columnar structures are considered to represent a retention of Henle's fiber layer. In addition to the retinoschisis in the outer retina, retinoschisis was also found at the level of the inner plexiform layer or an ILM detachment (Fig. 16.1) [7, 8]. Foveal RD often coexists with MRS. An outer lamellar MH is reported to predispose to the MRS to a foveal RD [11]. Mostly, an outer lamellar MH is observed in eyes with foveal RD if we analyze multiple OCT sections. MRS tends to be present in and around the macular atrophy around the regressed CNV due to pathologic myopia [2]. MRS in the eyes with atrophic stage of myopic CNV appeared markedly less column-like than the MRS seen in highly myopic eyes without CNV [12], and it was difficult to differentiate a foveal RD from MRS in the eyes with atrophic stage of myopic CNV.

Other methods than OCT might help diagnosis of MRS or visualization of the full extent of MRS in the posterior fundus. Retromode of the F10 (Nidek, Aichi, Japan) scanning laser confocal ophthalmoscope (SLO) uses an infrared laser and an aperture with a modified central stop. This optical arrangement allows for pseudo-three-dimensional image, which can detect abnormalities in the deeper retinal layers. By using retromode imaging by F10, Tanaka et al. [13] showed a characteristic fingerprint pattern at the corresponding area of the MRS (Fig. 16.2). The fingerprint pattern consisted of radiating retinal striae centered on the fovea and many light dots and lines that ran in parallel to the striae or formed a whorled pattern surrounding the radiating striae. Also, the area of MRS showed various retinal vascular abnormalities (capillary telangiectasia, microaneurysm formation, and dye leakage) by fluorescein angiography [14]. Sayanagi and associates [15] reported the different patterns of fundus autofluorescence (FAF) between macular hole retinal detachment and MRS.

Various macular lesions coexist in eyes with MRS, such as lamellar macular hole (lamellar MH), full-thickness macular hole (FTMH), and foveal retinal detachment (RD) (Fig. 16.3).

16.4 Pathologic Findings of MRS

Tang and associates [6] examined both eyes of a 73-year-old woman with high myopia and showed that the degenerative retinoschisis with interbridging strands in the outer plexiform layer of the macular region was found (Fig. 16.4). Interestingly, there was multiple cystic degeneration in the outer plexiform layer and there appeared to be folding of the inner layers of the retina, which were novel findings not observed previously with OCT.

16.5 Factors Related to MRS Development

MRS is considered to be caused by various factors. Wu and associates [16] reported that three factors were independently associated with MRS and foveal RD without MH in

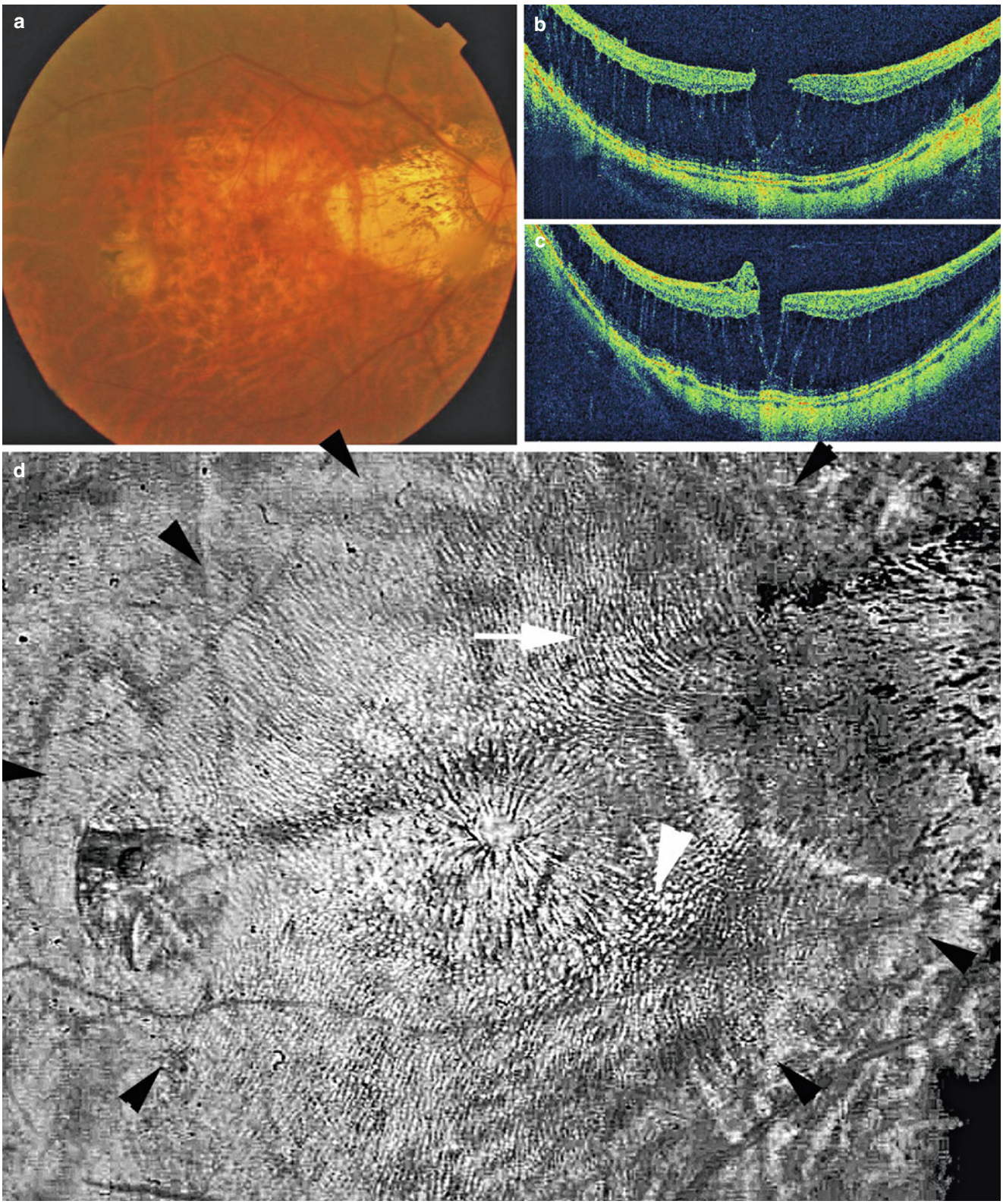


Fig. 16.2 Representative retromode images of an eye with an outer macular retinoschisis. (**a**) Fundus photograph of the right eye of a 77-year-old woman showing diffuse chorioretinal atrophy in the posterior fundus. (**b**, **c**) Horizontal and vertical scans across the central fovea by optical coherence tomography (OCT) showing the macular retinoschisis with inner lamellar hole in the central fovea. (**d**) Retromode image by F10 showing a fingerprint pattern (*black arrowheads*) consisting of central radiating retinal striae and surrounding multiple dots (*arrowhead*) and lines (*arrow*). Many lines appear in parallel or in a whorled pattern. The inner lamellar hole is observed as a circular defect at the central fovea

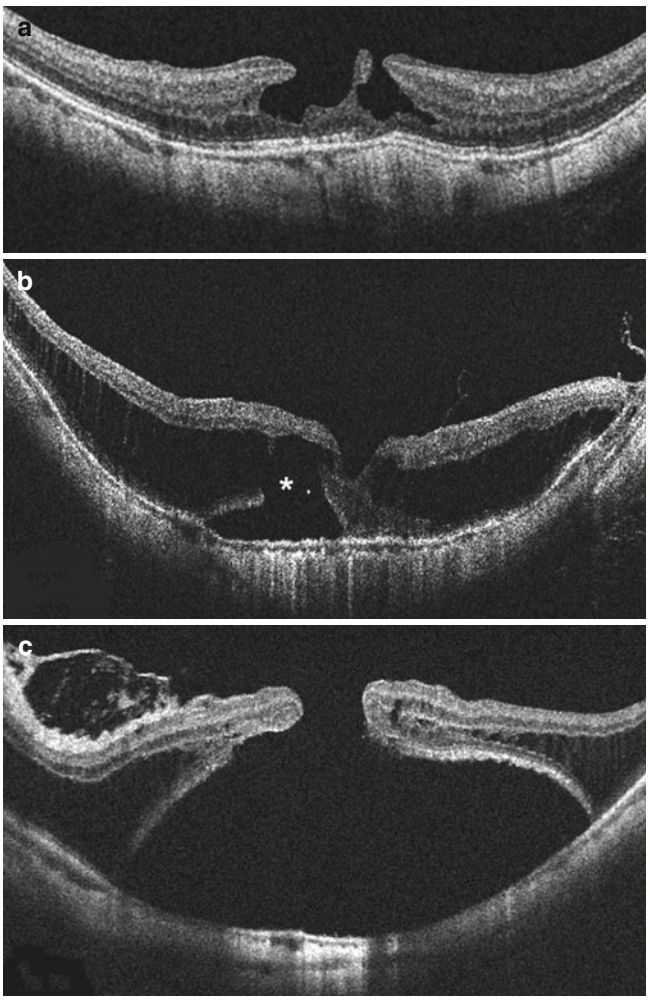


Fig. 16.3 Macular lesions associated with myopic macular retinoschisis. (**a**) Inner lamellar macular hole. (**b**) Outer lamellar macular hole (*asterisk*). (**c**) Full-thickness macular hole and foveal retinal detachment

high myopia in the multivariate analysis: axial length, macular chorioretinal atrophy, and vitreoretinal interface factors. MRS tends to develop in highly myopic eyes with advanced chorioretinal atrophy [2]. Johnson [17, 18] suggested four major traction mechanisms: vitreomacular traction (from perifoveal PVD), remnant cortical vitreous layer (after PVD), epiretinal membrane, and intrinsic noncompliance of the ILM. Using electron microscopy, Bando et al. [19] reported that collagen fiber and cell debris were identified on the inner surface of ILM in 70 % of the eyes with MRS, whereas none in ILM from control subjects (idiopathic MH). More fibrous glial cells were found on the inner surface of ILM from the eyes with myopic MRS [19], and they concluded that the cell migration and consequent collagen synthesis on the ILM can be another contributor for developing MRS.

Johnson [17] also suggested retinal arteriolar stiffness as minor mechanism contributing to the MRS development. The OCT examinations of serious sections along the entire posterior vascular arcade showed that the paravascular abnormalities such as paravascular lamellar holes [20], vascular microfolds [20–23], and paravascular retinal cysts [20] are frequently found in eyes with MRS (Figs. 16.5 and 16.6). Following to the formation of paravascular lamellar holes, the glial cells like astrocytes which exist abundantly around the retinal vessels can migrate and proliferate through the paravascular lamellar holes. These cells can produce collagen and facilitate the proliferative and contractile response of ILM. In some OCT sections, we directly can see images suggestive of cell migration toward the vitreous through paravascular lamellar holes (Fig. 16.5a). Hypothetical cartoon of mechanisms of MRS development is shown in Fig. 16.7.

MRS tends to develop in eyes with severe myopic fundus changes (patchy chorioretinal atrophy or bare sclera) more than those with mild myopic fundus changes [2]. Although the reason for this association is not fully clear, the scleral shape alterations are considered to affect the MRS development [24–35]. The slope and shape of posterior staphyloma in highly myopic eyes have long been analyzed by using OCT [27–35]. Smiddy et al. [36] hypothesized that progressive staphyloma formation generated a posteriorly applied force that gives the appearance that there is primary (anterior or tangentially directed) preretinal traction.

Recent studies using swept-source OCT support the association between scleral curvature alterations and MRS [24–26]. Intrachoroidal cavitation (ICC) is yellowish-orange lesion located inferior to the optic disc (see choroid chapter) [37–40]. Ohno-Matsui et al. [25] recently found that ICC was located in the macular area on and around the patchy chorioretinal atrophy. As seen in peripapillary ICC, the sclera in the area of macular ICC was bowed posteriorly (Fig. 16.8). The eyes with macular ICC had retinoschisis around the patchy atrophy significantly more frequently than the eyes without cavitation. Swept-source OCT also showed that the curvature of inner scleral surface of highly myopic eyes could be divided into those whose curvature sloped toward the optic nerve, symmetrical and centered on the fovea, asymmetrical, and irregular [26]. Patients with irregular curvature were significantly older with significantly longer axial lengths than eyes with other curvatures. MRS was present significantly more frequently in the eyes with irregular curvature [26]. These descriptions suggested that a scleral contour might affect the development of MRS. In contrary, some other kinds of scleral contour might act preferably against the MRS development. Dome-shaped macula (DSM) was first described by Gaucher and associates [28] as an unexpected finding in myopic staphyloma and was characterized as an inward convexity of the macula (see sclera chapter).

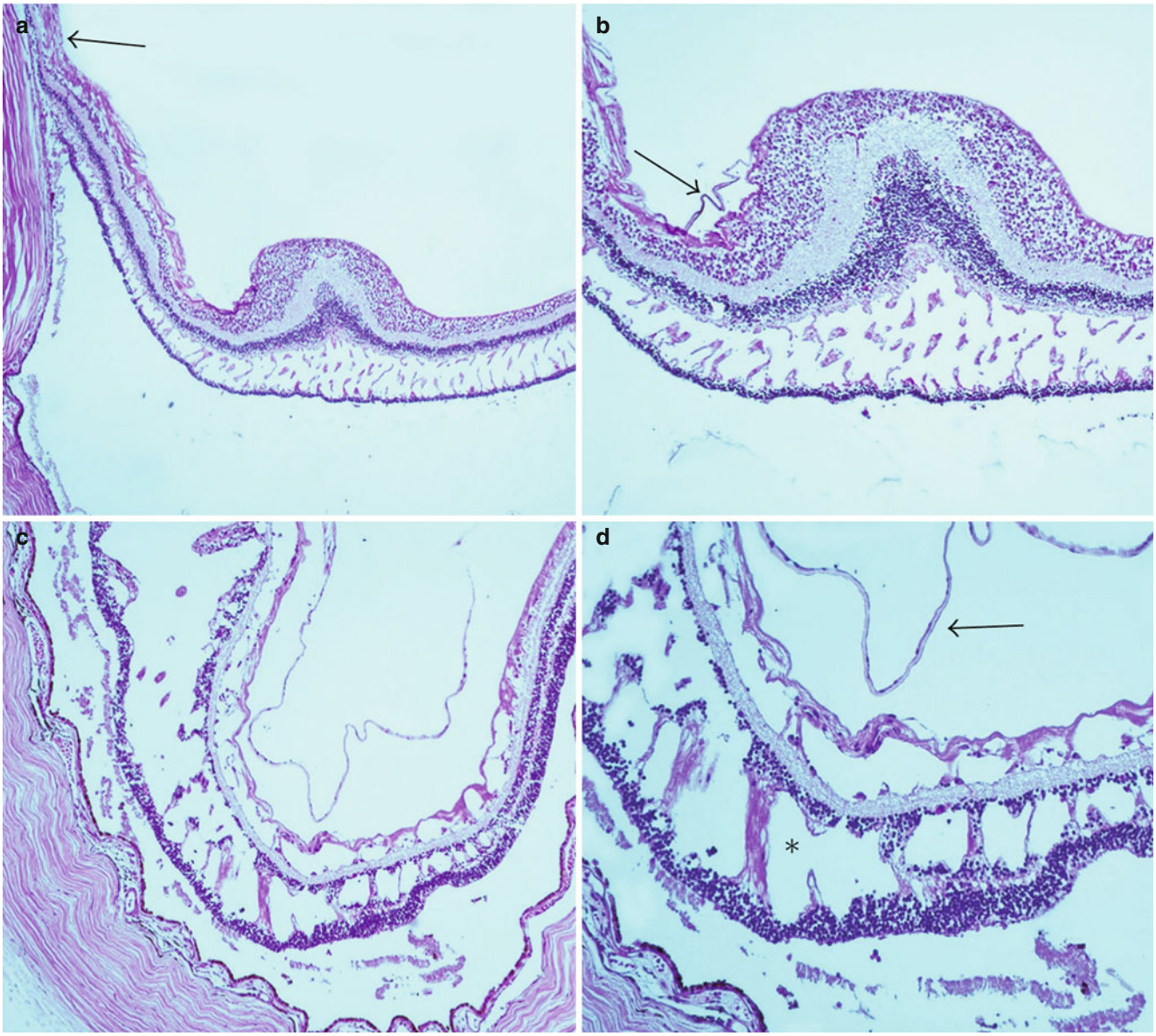


Fig. 16.4 Photomicrograph of the right eye demonstrating areas of macular retinoschisis (MRS). (**a**) Lower magnification showing the tissue splitting in the outer retina. A region containing the staphyloma is also seen (*black arrow*). (**b**) Higher magnification of MRS seen in multiple layers of the retina including the outer plexiform layer, inner plexiform layer, nerve fiber layer, and the outer plexiform layer in the perifoveal region. A thin fibrous preretinal membrane is seen (*black arrow*) (Hematoxylin and eosin, original magnification, (**a**) ×50; (**b**) ×100.) (**c**) Photomicrograph of the left eye demonstrating MRS in the outer plexiform layer, ganglion cell layer, and nerve fiber layer. (**d**) Higher magnification demonstrating neuronal bridges between both nuclear layers (*asterisk*). A fibrous preretinal membrane is seen (*black arrow*) (Hematoxylin and eosin, original magnification, (**c**) ×50; (**d**) ×100)

By using EDI-OCT, Imamura and Spaide [30] reported that the DSM is the result of a localized variation in thickness of the sclera in the macular area. Ellabban and associates [41] recently found that among 9 eyes with extrafoveal retinoschisis, only 1 eye had foveal schisis without either foveal RD or MH formation. They suggested that the bulge in eyes with a DSM may act as a macular buckle-like mechanism and thus may prevent or alleviate tractional forces over the fovea, thereby preventing schisis or detachment.

Progression of MRS into foveal RD after IVB against myopic CNV has been reported [42]. In eyes with a myopic CNV, the neural retina is pushed vitreally by the protrusion of the CNV. In addition, there is a tractional force of the ILM on the retina in eyes with a retinoschisis. IVB causes a rapid shrinkage of the CNV accompanied by an absorption of the subretinal hemorrhage and exudated fluid. Under these conditions, the continuous inward tractional force by the ILM enhanced by the rapid contraction

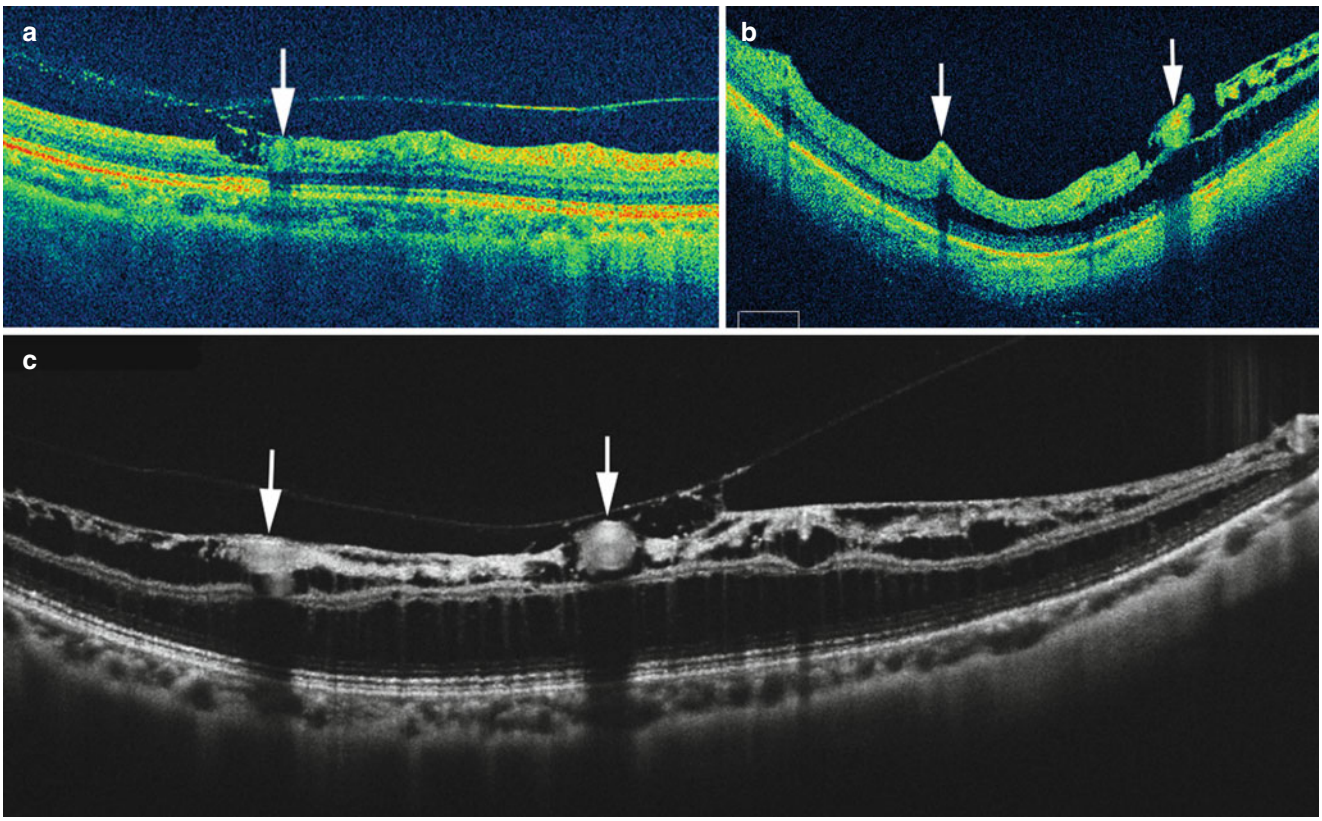


Fig. 16.5 Paravascular abnormalities seen in eyes with myopic macular retinoschisis (MRS). In (**a**–**c**), *arrows* indicate the retinal vessels. (**a**) The upper roof of paravascular retinal cyst is avulsed by the detached posterior hyaloid and is observed as paravascular lamellar hole. Through the paravascular lamellar hole, it seems that the cells migrate toward the vitreous, shown as many granular hyperreflective dots. (**b**) Paravascular lamellar holes are observed along both sides of the retinal vessel (shown by a *right arrow*). Retinal vessels seem to protrude toward the vitreous and are observed as retinal vascular microfolds. (**c**) Paravascular retinal cysts are observed along the retinal vessels. A wide MRS is also noted in the inner as well as outer retina

of the CNV leads to further splitting of the retina and finally to an RD.

16.6 Natural Course

Earlier studies have reported the progression of an MRS into more serious complications such as foveal RD or full-thickness MH during its natural course [9, 11, 36, 43–46]. Benhamou et al. [9] reported that one of the 21 highly myopic eyes (4.8 %) with MRS without MH evolved into a full-thickness MH, and this eye had a vitreous traction on the fovea. Fujimoto and associates [5] reported that a foveal RD developed in 6 of 21 eyes (28.6 %) with myopic MRS and FTMH developed in 2 of the 21 eyes (9.5 %) with MRS during a follow-up. Gaucher and associates [45] reported that 6 of 18 eyes (33.3 %) with MRS that did not undergo surgical intervention developed full-thickness MH during a mean follow-up of 34.7 months (range, 12–60 months). Shimada et al. [44] also reported that four of eight eyes (50.0 %) with MRS progressed to foveal RD or full-thickness MH in a follow-up period of more than 2 years. Sun et al. [46] reported five eyes of five patients with myopic MRS who developed full-thickness MH. The natural evolution from MRS to full-thickness MH was classified into two patterns by OCT findings. In full-thickness MH formation pattern 1, a focal area of the external retinal layer was elevated and followed by the development of a small outer lamellar MH and retinal detachment (RD). The outer lamellar MH and RD were then enlarged horizontally and elevated vertically until the outer lamellar MH was attached to the overlying retinal layer. A full-thickness MH finally developed when the roof of RD opened. In full-thickness MH formation pattern 2, the opening of the roof of MRS or cystoid space caused an inner lamellar MH. The MRS was then gradually resolved except the residual MRS beneath the inner lamellar MH, and the inner lamellar MH would finally proceed into a full-thickness MH. As a mechanism of progression from MRS to foveal RD, Shimada et al. [11] investigated five eyes of five consecutive patients with myopic MRS who developed an RD during the follow-up period. The results showed that the progression from MRS to foveal RD passed through four stages: (1) a focal irregularity of the thickness of external retina, (2) an outer lamellar MH development within the

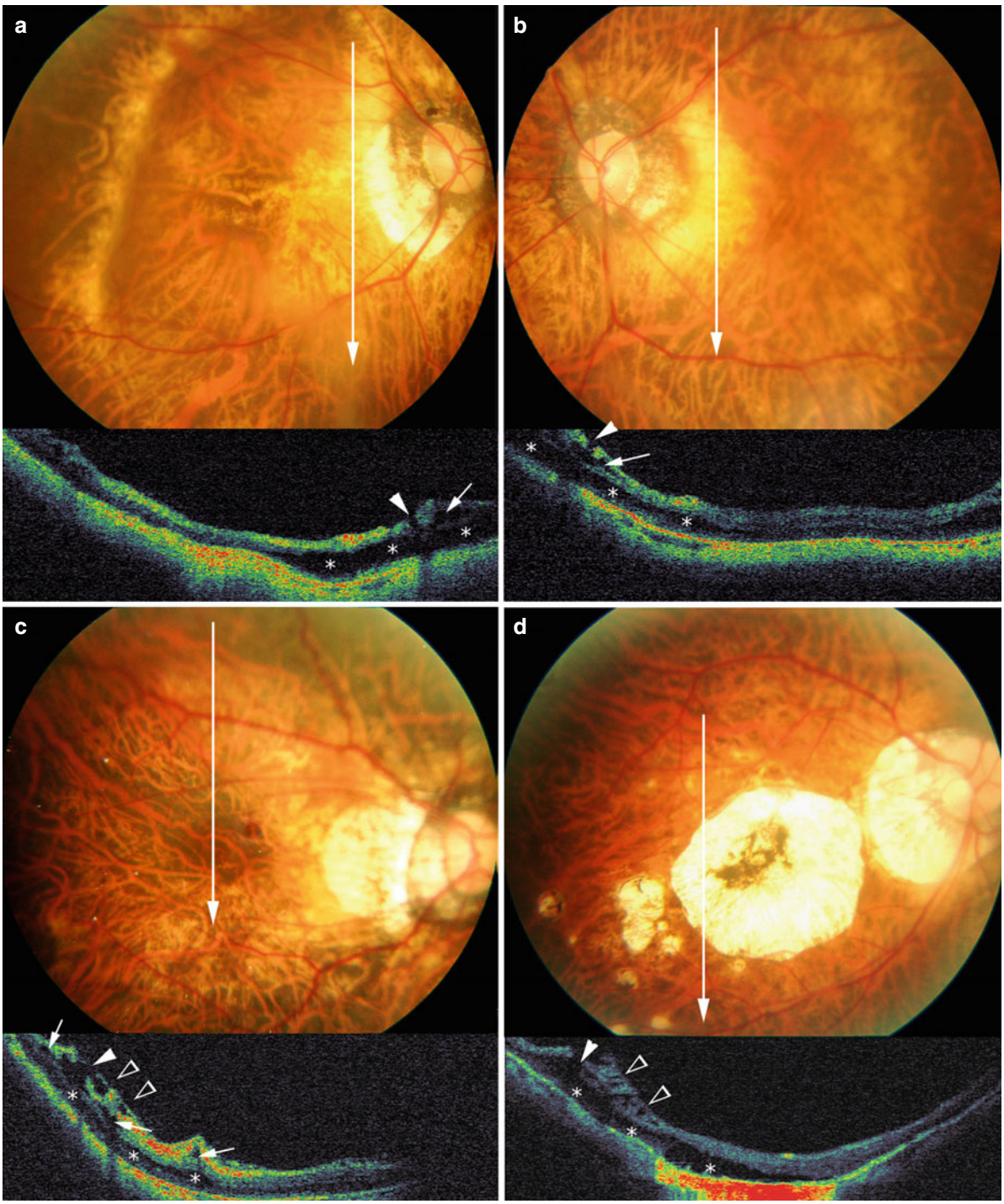


Fig. 16.6 Cases with paravascular retinal holes and retinoschisis. (**a**) Right fundus photograph and optical coherence tomography (OCT) image of a 70-year-old woman with a refractive error of −15.0 diopters (*D*) and an axial length of 28.6 mm. Vertical OCT scan shows a paravascular retinal hole (*arrowhead*). Microfolds around the lower temporal retinal vessel can be seen, and a paravascular retinal cyst is also observed (*arrow*). (**b**) Left fundus photograph and OCT image of a 70-year-old woman with a refractive error of −14.5 D and an axial length of 28.4 mm. Vertical OCT scan shows a paravascular retinal hole (*arrowhead*). Paravascular retinal cyst (*arrow*) is also observed around the retinal hole. (**c**) Right fundus photograph and OCT image of a 56-year-old woman with a refractive error of −16.0 D and an axial length of 29.8 mm. Vertical OCT scan shows a paravascular retinal hole (*arrowhead*). Paravascular retinal cyst is also observed (*arrows*). Internal limiting membrane detachment is observed (*open arrowheads*). (**d**) Right fundus photograph and OCT image of a 60-year-old man with a refractive error of −12.0 D and an axial length of 28.5 mm. Vertical OCT scan shows a paravascular retinal hole (*arrowhead*). Internal limiting membrane detachment is observed (*open arrowheads*). (**e**) Right fundus photograph and OCT image of 54-year-old man with a refractive error of −14.0 D and an axial length of 28.3 mm. Vertical OCT scan shows a paravascular retinal hole (*arrowhead*). Paravascular retinal cyst is also observed (*arrows*). Internal limiting membrane detachment is observed (*open arrowheads*). (**f**) Right fundus photograph and OCT image of a 76-year-old woman with a refractive error of −15.0 D and an axial length of 27.5 mm. The OCT scan shows a paravascular retinal hole (*arrowhead*). *Outer retinoschisis

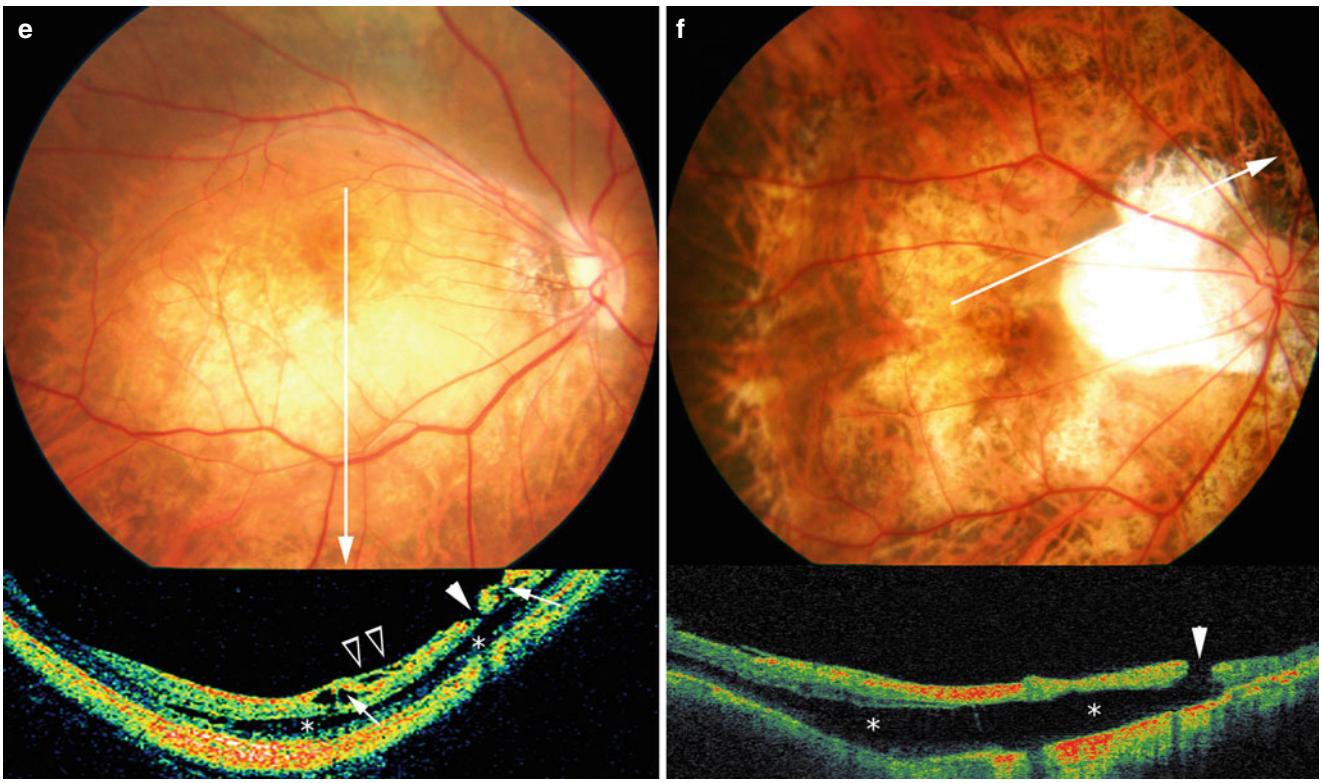


Fig. 16.6 (continued)

thickened area and subsequent development of small RD, (3) horizontal separation of the column-like structures overlying the outer lamellar hole and vertical enlargement of outer lamellar hole, and (4) the elevation of the upper edge of the external retina and the attachment to the upper part of retinoschisis layer accompanying with further enlargement of RD (Fig. 16.9). The interval from stage 1–3 was relatively short (mean, 4.5 months) indicating that we should be very cautious about the progression to RD when the findings in stage 1 are observed in OCT images. Once the outer lamellar hole develops, an RD will develop in a short time. Sayanagi and Ikuno [47] reported a case who showed a spontaneous resolution of MRS and a consequent development of foveal RD.

Shimada et al. [48] recently analyzed the natural course in as many as 207 eyes with MRS with a follow-up ≥24 months. Shimada et al. have classified MRS according to its extent and location from S_0 through S_4 (Fig. 16.10): no MRS (S_0), extrafoveal MRS (S_1), foveal only MRS (S_2), foveal but not entire macular area MRS (S_3), and entire macular area MRS (S_4). The progression of MRS was defined as (1) an increase of the extent or height of MRS (an increase of the height means the change >100 μm) and (2) new development of lamellar MH, foveal RD, or FTMH. The results showed that the progression was found in 26 of 207 eyes (12.6 %) (Fig. 16.11). According to the extent of MRS at the initial examination, the progression was found in 6.2 % of the eyes with S_0, in 3.6 % of the eyes with S_1, in 8.9 % of the eyes with S_2, in 13.0 % of the eyes with S_3, and in 42.9 % of the eyes with S_4. The eyes with S_4 MRS had significantly more frequently showed a progression of MRS than the eyes with S_0–S_3. In the eyes with S_0 and S_1, a development or increase of MRS was found. In the eyes with S_2, a development of full-thickness MH was a main progression pattern. In the eyes with S_4, a development of foveal RD was a main progression pattern. Figure 16.11 shows the representative cases with progression.

For the natural course of the lesions associated with MRS, Tanaka et al. [49] reported that a lamellar MH might be a relatively stable condition in highly myopic eyes, as we observe for lamellar MH in non-myopic eyes. Twenty-three of 24 eyes (95.8 %) with a lamellar MH did not show any changes in the OCT images during a mean follow-up of 19.2 ± 10.2 months, although the remaining eye progressed to a full-thickness MH.

Spontaneous resolution of MRS has also been reported. Polito et al. [50] reported a case whose foveal RD as well as MRS spontaneously disappeared in the follow-up by developing spontaneous posterior vitreous detachment (PVD). Using the MRS classification into S_0–S_4, Shimada et al. [48]

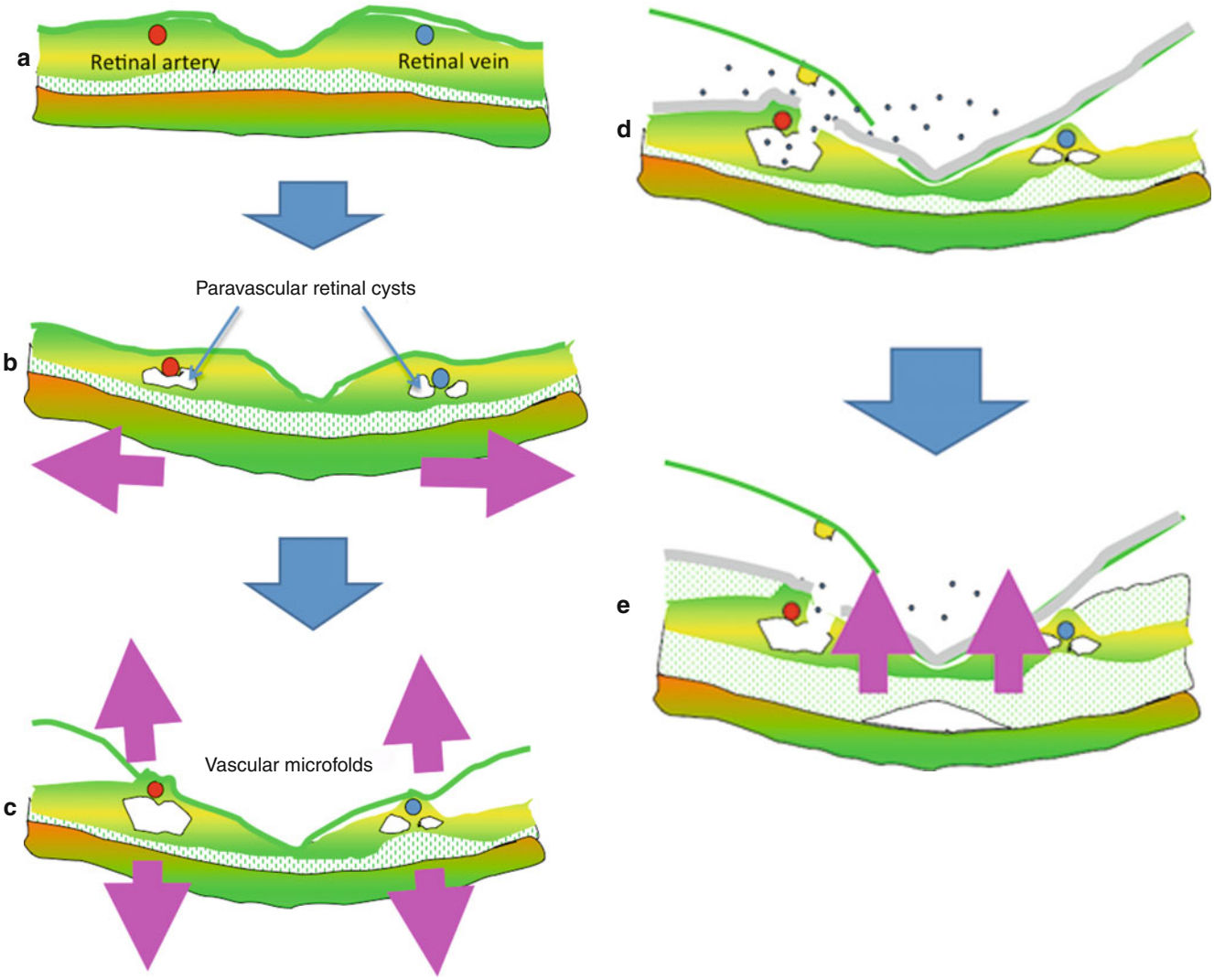


Fig. 16.7 Cartoons showing hypothetical mechanism of developing myopic macular retinoschisis (MRS). Cross-sectional image of normal macula (**a**). In response to uniform expansion of the globe due to increased axial length, paravascular retinal cysts develop along the retinal vessels because of the difference of expansion between retinal vessels and other parts of the retina (**b**). Secondary to posterior staphyloma, retinal vascular microfolds develop and paravascular retinal cysts enlarge and fuse with one another (**c**). With the development of posterior vitreous detachment, the inner wall of paravascular retinal cysts is pulled away, and inner lamellar holes develop (**d**). Through the paravascular lamellar holes, the glial cells migrate and produce collagen, enhancing the proliferative and contractile property of the inner internal limiting membrane (ILM) (**e**). Due to increased contractility of ILM, MRS finally develops

also analyzed the spontaneous resolution of MRS. The improvement of MRS was defined as a decrease of the extent or height of MRS without developing new lamellar MH, RD, or full-thickness MH. Interestingly, 8 among a total of 175 eyes showed a resolution of MRS, a decrease of MRS in two eyes, and complete resolution of MRS in six eyes (Fig. 16.12). Resolution of MRS was seen in three eyes with each of S_3 and S_4 and in one eye of each S_1 and S_2. Six of the eight eyes with improvement of MRS showed a release of retinal traction before resolution of MRS. PVD developed in four eyes and spontaneous disruption of ILM was developed in two eyes before resolution of MRS. Figure 16.9 shows the representative cases with spontaneous resolution. Although the number of the patients is limited, it suggests that a spontaneous disruption of ILM occurs and it causes a subsequent resolution of MRS.

16.7 Treatment for MRS

The literature on treatment options for MRS and its complications is limited by a lack of prospective data and small numbers in treated series. However, the usefulness of pars plana vitrectomy (PPV) in resolving the foveal RD and MRS

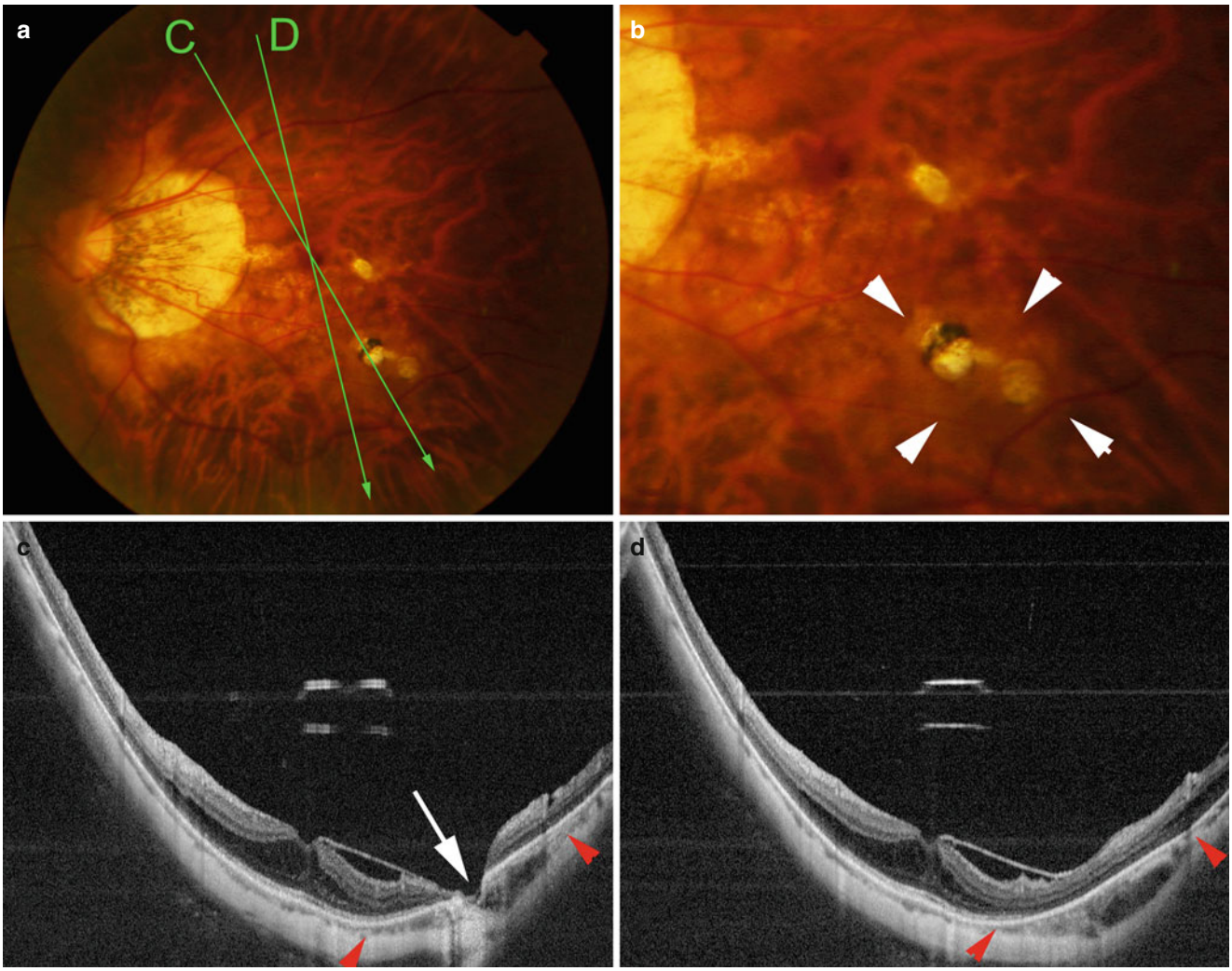


Fig. 16.8 Macular retinoschisis (MRS) seen in the area of macular intrachoroidal cavitation (macular ICC). (**a**) Photograph of the left fundus of a 60-year-old woman showing three areas of patchy chorioretinal atrophy temporal to and inferotemporal to the fovea. (**b**) Magnified image of top left image shows three areas of patchy chorioretinal atrophy. The area around the patchy atrophy is orange (*arrowheads*). (**c**) B-scan swept-source optical coherence tomographic (OCT) image of *line C* in (**a**) shows that the sclera is bowed posteriorly (between *arrowheads*) compared to neighboring sclera beyond the retinal pigment epithelium (RPE) defects. The choroid seems to be thickened in the area and the retina is caved into the intrachoroidal cavitation (*arrow*). Inner retinoschisis is observed around the intrachoroidal cavitation. (**d**) In the section shown by *line D* in *top left* image, a bowing of the sclera is observed (between *arrowheads*). The hyporeflective space suggests the presence of fluid in the space of the intrachoroidal cavitation

has been reported in many studies [51–57]. The functional and anatomical outcome of vitrectomy in earlier studies is summarized in Table 16.1.

The indication of PPV for MRS without full-thickness MH has not been consistent. It is generally considered that we should perform vitrectomy for the eyes which developed foveal RD in addition to MRS. However, we are not certain what the indications are and when the optimal time is for surgery especially for myopic MRS without RD. The majority of eyes with MRS without foveal RD retain relatively good vision. Based on the four distinct stages from MRS to foveal RD, Shimada et al. [11] recommend that we had to better consider surgical treatment between stage 3 (development of outer lamellar MH and small RD around it) and 4 (the upper edge of outer retina is attached to the upper part of the retinoschisis layer), because the vitrectomy at stage 4 has an increased risk of developing full-thickness MH postoperatively.

ILM peeling was first reported by Kuhn [58] to treat macular retinal detachment without macular hole in a highly myopic patient, although OCT was not performed in this report. The need for ILM peeling during vitrectomy remains a controversy; however, it is necessary when apparent ILM traction is recognized on preoperative OCT images. The cases with myopic MRS and foveal RD who were successfully treated with vitrectomy without ILM peeling were

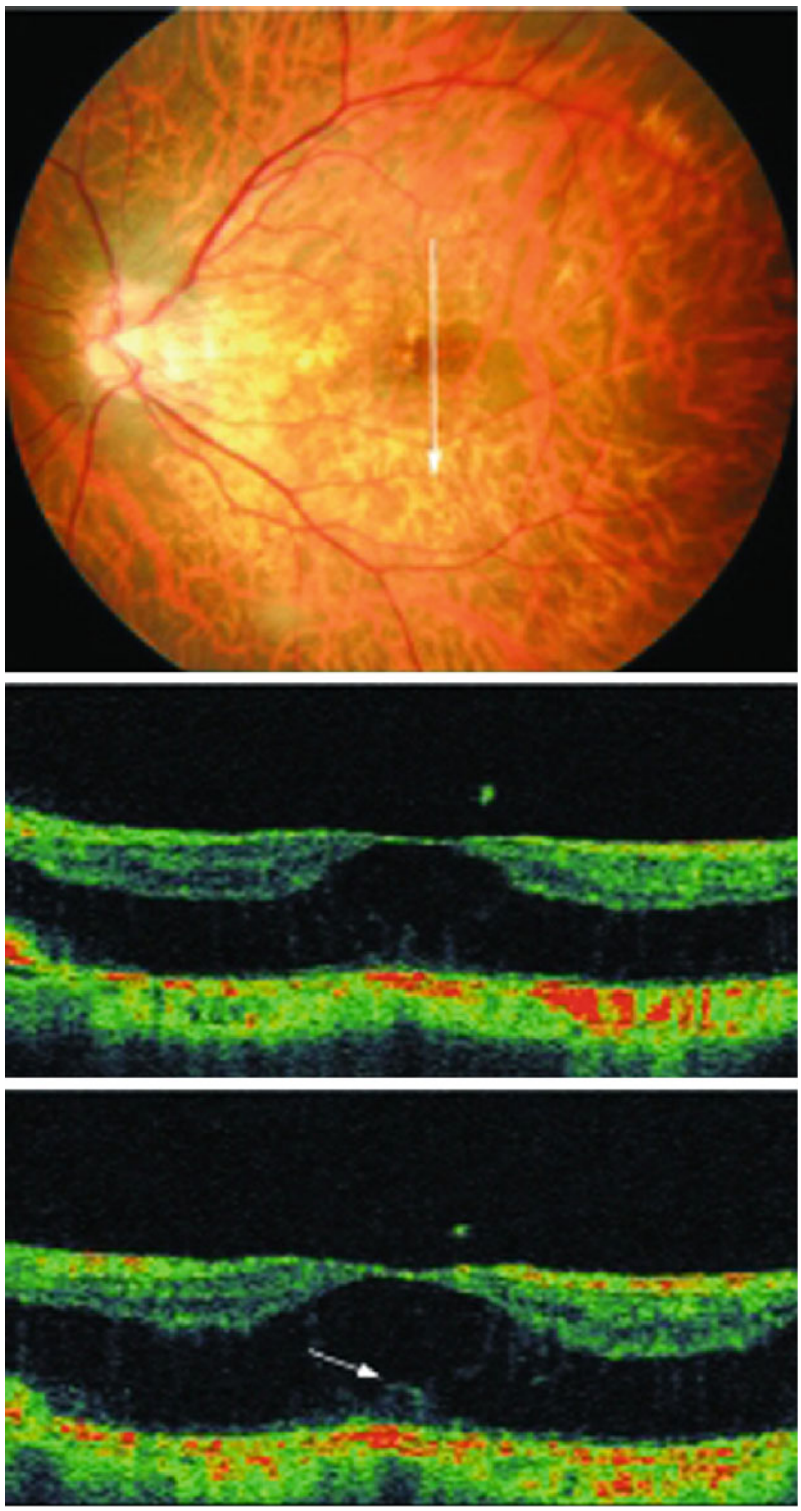
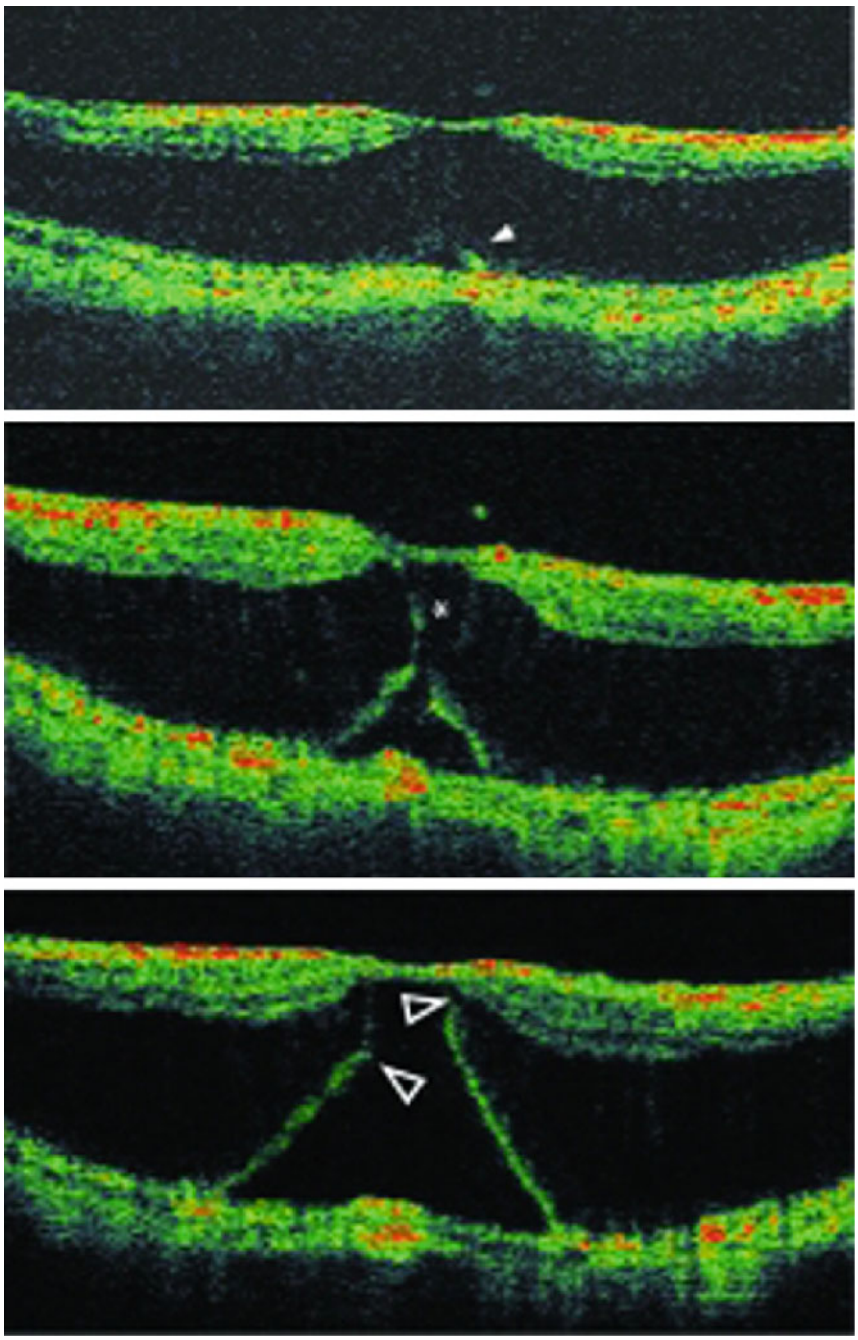


Fig. 16.9 Different stages in the progression from myopic macular retinoschisis to early retinal detachment. *Top row*: posterior fundus photographs at the initial examination. *Second to fifth rows*: optical coherence tomographic (OCT) images at the initial examination and at stages 1, 2, 3, and 4. At the initial examination, the OCT images show macular retinoschisis without a retinal detachment. The outer retinal layer appears to be normal. At stage 1, the OCT images show a focal thickening of the outer retinal layers (*arrow*), and at stage 2, a lamellar hole (*arrowhead*) is present beneath the thickened area. At stage 3, the retinoschisis layer overlying the outer lamellar hole is separated horizontally (*asterisks*), and the outer lamellar hole appears enlarged. At stage 4, the upper edge of the external retina (*open arrowhead*) is attached to the upper part of retinoschisis layer. The RD is larger and is accompanied by the resolution of the retinoschisis

Fig. 16.9 (continued)



reported [57]; however, Futagami and Hirakata [59] reported a case whose MRS recurred 3 years after the vitrectomy without ILM peeling and was successfully treated by the second surgery involving the ILM peeling.

Besides the dispute whether we should perform ILM peeling or not to treat MRS, another option would be to remove ILM completely in the macular area or outside the foveal area only. The development of a full-thickness MH is a serious complication in highly myopic eyes with a foveal RD during and after vitrectomy [55]. This is important because a full-thickness MH is associated not only with reduced vision but also with a risk of developing an MH retinal detachment in highly myopic eyes [60–63]. It is difficult to obtain a closure of full-thickness MH in highly myopic eyes [64, 65].

The mechanisms of why and how full-thickness MHs develop postoperatively in eyes with foveal RD have not been fully determined; however, it is hypothesized that ILM peeling itself will increase the risk of developing a full-thickness MH. The mechanical traction on such a thinned

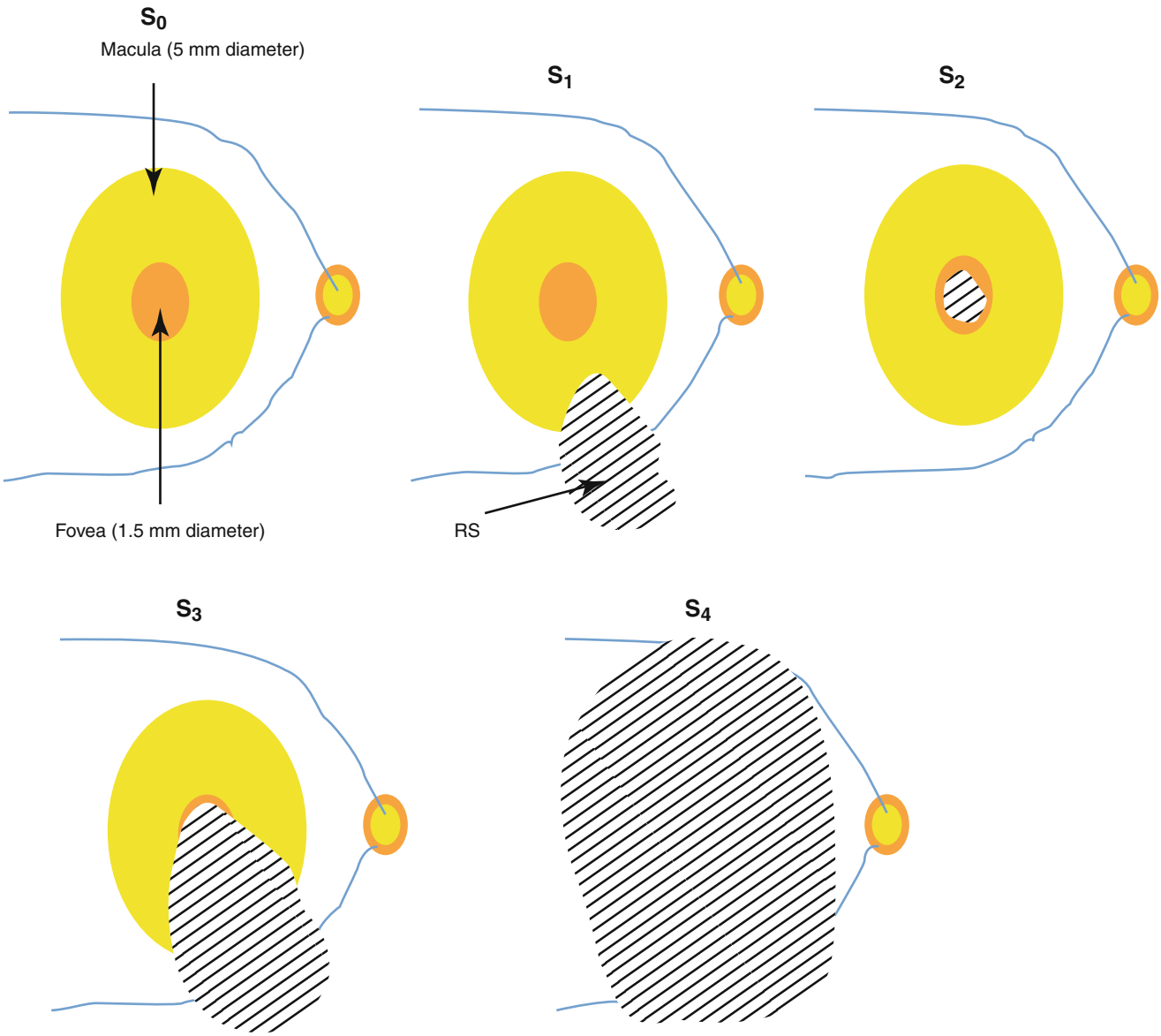


Fig. 16.10 Schematic illustration of classification of myopic macular retinoschisis (MRS) according to the area. S_0 no MRS, S_1 extrafoveal, S_2 foveal, S_3 includes both fovea and extrafoveal but does not involve the entire macula, S_4 entire macula

central fovea by peeling the ILM off of the fovea could induce a break of the central foveal tissue. Another possibility is that when the retina moves backwards to match the contour of the staphyloma after vitrectomy, the removal of ILM may reduce the structural strength of the fovea. Based on these concerns, Ho et al. [66] and Shimada et al. [67] reported the "foveola nonpeeling technique" or "fovea-sparing ILM peeling," respectively. For fovea-sparing ILM peeling, the ILM was grasped with an ILM forceps and peeled off in a circular fashion (Fig. 16.13), but the ILM was not completely removed and was left attached to the fovea. After the ILM was peeled from the entire macula area except the foveal area (in a circular area with a diameter approximately that of the vertical extent of the optic disc), the peeled ILM was trimmed with a vitreous cutter. Shimada et al. [67] reported that at around 3 months after fovea-sparing ILM peeling, a contraction of the remaining ILM at the fovea was observed as an irregular thickening of the retinal surface, and the outer lamellar hole became smaller or indistinct (Fig. 16.14). No further contraction of the remaining ILM was obvious after 3 months. None of the eyes with the fovea-sparing ILM peeling developed full-thickness MH [66, 67]. Although the longer follow-up in a large number of the patients as well as appropriate controls is necessary, these results are promising for the prevention of postoperative full-thickness MH formation. The combined release of macular

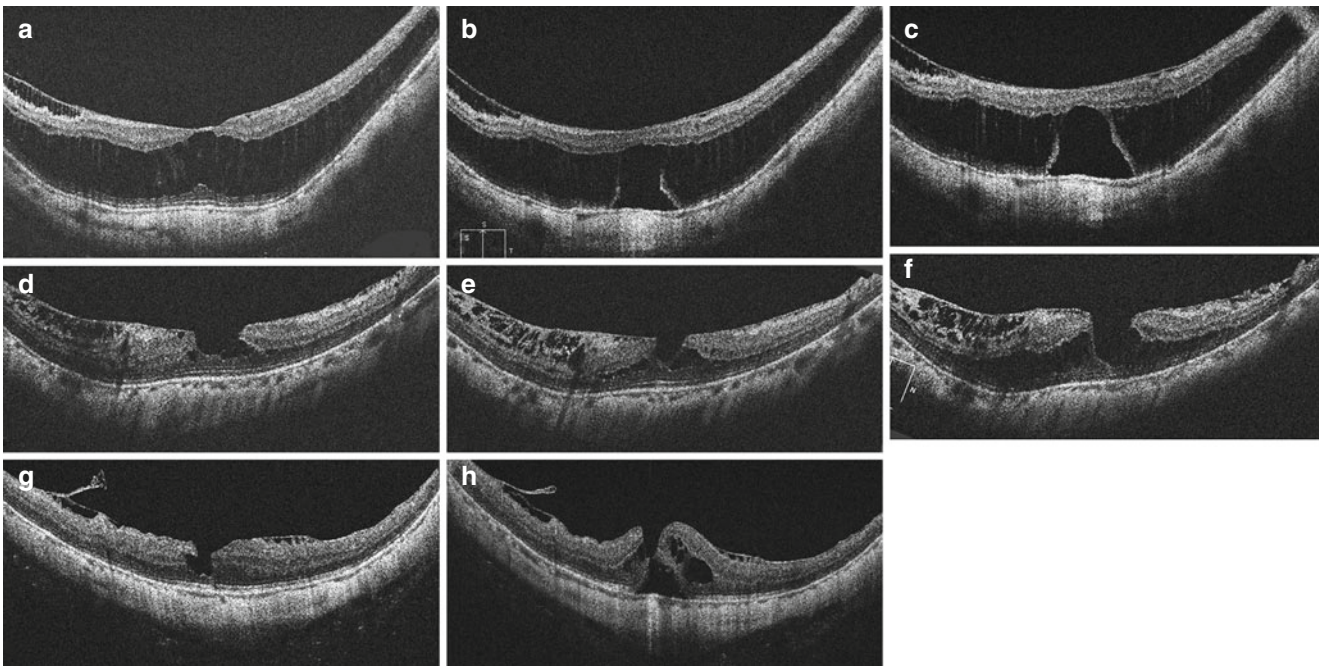


Fig. 16.11 Cases who show progression of myopic traction maculopathy (MTM). (**a**–**c**) Progression from macular retinoschisis (MRS) to foveal retinal detachment (RD). Left eye of a 69-year-old man with an axial length of 30.9 mm. (**a**) Vertical OCT scan across the fovea shows a wide MRS in the macular area. (**b**) Fifteen months later, a foveal RD has developed. An outer lamellar macular hole is also formed. (**c**) At 20 months after the initial examination, the foveal RD is seen. (**d**–**f**) An increased extent of MRS. A right eye of a 52-year-old man with an axial length of 30.1 mm. (**d**) Vertical OCT section across the fovea shows a limited MRS on the fovea. Inner lamellar macular hole is also seen. ILM detachment is seen inferior to the fovea. (**e**) Nine months later, the MRS area has slightly enlarged. (**f**) Two more months later, the MRS area has further enlarged. (**g**, **h**) Development of full-thickness macular hole in the left eye of a 62-year-old man with an axial length of 31.9 mm. (**g**) Vertical OCT section across the fovea shows an inner lamellar macular hole. ILM detachment is observed around the fovea. (**h**) Eight months later, a full-thickness macular hole has developed

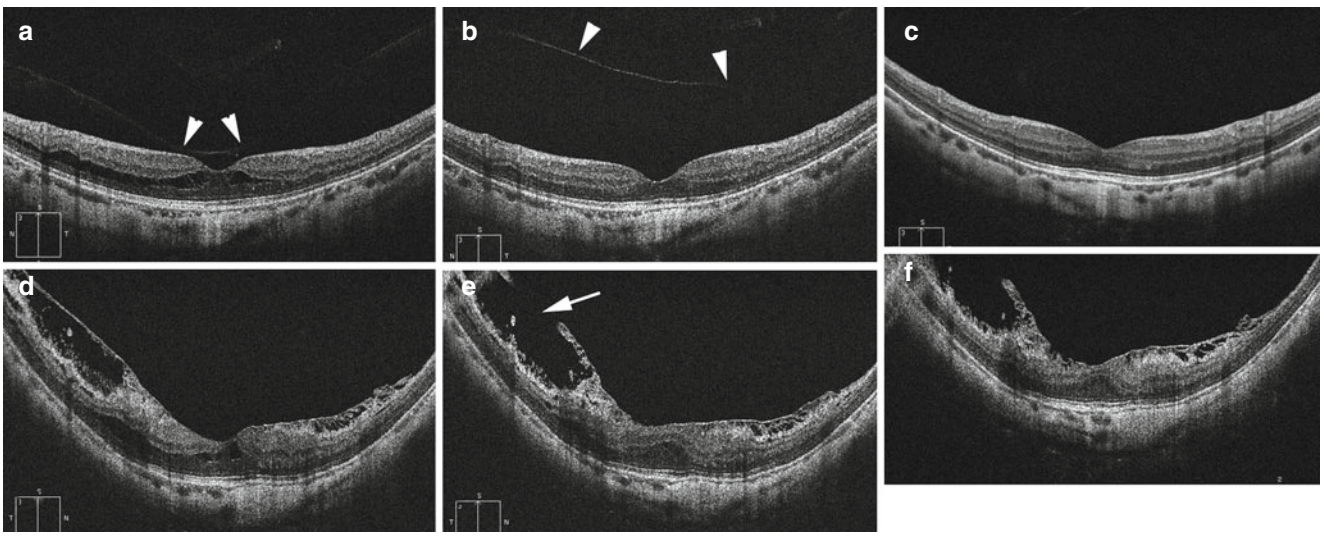


Fig. 16.12 Cases who show a resolution of myopic traction maculopathy (MTM). (**a**–**c**) Complete resolution of macular retinoschisis (MRS) subsequent to a development of posterior vitreous detachment (PVD). Left eye of a 46-year-old woman with a refractive error (spherical equivalent) of −16.0 D and an axial length of 29.3 mm. (**a**) Vertical OCT scan across the fovea at the initial examination shows a shallow MRS on and around the fovea. Partial PVD is seen (*arrowheads*). (**b**) One month later, a complete PVD has occurred (*arrowheads*) and MRS has almost disappeared. (**c**) Fifteen months after the initial visit. MRS has been completely resolved. (**d**–**f**) Resolution of macular retinoschisis (MRS) secondary to spontaneous disruption of internal limiting membrane (ILM). Right eye of a 52-year-old man with an axial length of 30.9 mm. (**d**) Vertical OCT scan across the fovea at the initial examination shows a shallow MRS on and upper to the fovea. ILM detachment is also noted. (**e**) Fourteen months later, a spontaneous disruption of ILM has occurred upper to the fovea (*arrow*). MRS appears to decrease. (**f**) Twenty-four months after the initial visit, MRS has been completely resolved

Table 16.1 Summary of earlier studies reporting the functional and anatomical outcome of vitreous surgery against myopic traction maculopathy

					Preoperative OCT findings			Vitreous surgery		Postoperative OCT findings				Mean BCVA		
Year	Author(s)	No. of eyes (patients)	Age (years) (mean) (range)	Axial length (mm) (mean) (range)	MRS	Foveal RD	FTMH	ILM peeling	Gas tamponade	Complete resolution of RD and MRS	Partial resolution of RD and MRS	Unchanged	Complications	Before surgery	After surgery	Mean follow-up period (months)
2003	Kobayashi and Kishi	9 (7)	54.7 (36–74)	27.5 (26.5–28.5)	9/9 eyes	9/9 eyes	–	+	+	8/9 eyes	1/9 eyes	–	FTMH in 1 eye	0.17 (0.02–0.4)	0.48 (0.4–0.6)[a]	20.4
2003	Kanda	2 (2)	52 and 84	N/A	2/2 eyes	1/2 eyes	–	+	1/2 eyes	1/2 eyes	1/2 eyes	–	–	N/A	N/A	8 and 12
2004	Ikuno	6 (5)	59.5 (51–63)	29.2 (27.9–29.9)	6/6 eyes	6/6 eyes	–	+	+	5/6 eyes	1/6 eyes	–	–	N/A	N/A	14
2005	Spaide	6 (5)	61	N/A	4/6 eyes?	4/6 eyes?	–	–	+	6/6 eyes	–	–	–	20/100	20/60	19.1
2005	Kwok	9 (8)	52.7 (40–65)	29.0 (26.3–32.1)	9/9 eyes	9/9 eyes	–	+	+	7/9 eyes	2/9 eyes	–	–	20/80	20/50	17.2
2006	Hirakata	16 (14)	64.9 (53–77)	28.0 (24.9–30.2)	16/16 eyes	11/16 eyes	2/16 eyes	6/16 eyes	12/16 eyes	16/16 eyes	–	–	FTMH in 5 eyes	N/A	N/A	25.6
2006	Scott	3 (3)	53, 31, 69	32.6 mm in 1 case	3/3 eyes	2/3 eyes	–	2/3 eyes	3/3 eyes	3/3 eyes	–	–	–	N/A	N/A	8, 7, 1
2007	Pannozzo	24	58 (32–79)	N/A	24/24 eyes	5/24 eyes	–	24/24 eyes	–	23/24 eyes	–	1/24 eyes	FTMH in 5 eyes	logMAR 0.6 (1.1–0.2)	logMAR 0.43 (1.1 to –0.1)	29.6
2007	Gaucher	11	55 (43–70)	N/A	11/11 eyes	5/11 eyes	–	1/11 eyes	6/11 eyes	4/11 eyes	4/11 eyes	–	FTMH in 3 eyes	logMAR 0.97	logMAR 0.63	26.9
2008	Yeh	3 (3)	61, 62, 52	30.1, 28.8, 31.1	3/3 eyes	3/3 eyes	–	–	+	1/2 eyes	2/2 eyes	–	RRD and retinal breaks in one eye each	N/A	N/A	12
2008	Ikuno	44 (42)	63.3 (43–79)	29.1 (24.4–34.6)	16/44 eyes	17/44 eyes	11/44 eyes	+	+	44/44 eyes	–	–	FTMH in 2 eyes	N/A	N/A	12
2009	Fang	6 (6)	53.1	29	6/6 eyes	6/6 eyes	–	–	+	4/6 eyes	2/6 eyes	–	–	20/400	20/160	9.8
2010	Kumagai	39 (39)	66.3 ± 8.3 (44–80)	28.6 ± 2.3 (24.2–34.7)	39/39 eyes	27/39 eyes	–	+	34/39 eyes	39/39 eyes	–	–	–	logMAR 0.79 ± 0.60	logMAR 0.54 ± 0.60	6
2011	Zhang	18 (17)	51.3 ± 13.7 (25–78)	29.7 ± 2.1 (26.8–34.1)	18/18 eyes	12/18 eyes	–	+	11/18 eyes	18/18 eyes	–	–	–	logMAR 0.94 (2–0.15)	logMAR 0.49 (1.3–0.15)	17.5
2012	Kim	17 (17)	61.9 (44–78)	29.75 (27.80–32.95)	17/17 eyes	9/17 eyes	–	+	9/17 eyes	12/17 eyes	2/17 eyes	3/17 eyes	FTMH in 2 eyes	logMAR 0.81–0.83	logMAR 0.56	13 or 15.3
2012	Shin	38 (36)	63.5 ± 9.5 (32–84)	29.16 ± 1.92 (26.61–36.17)	38/38 eyes	7/38 eyes	2/38 eyes	+	+	34/38 eyes	3/38 eyes	–	FTMH in 1 eye	logMAR 0.841 ± 0.534	logMAR 0.532 ± 0.536	6

OCT optical coherence tomography, *MRS* macular retinoschisis, *RD* retinal detachment, *FTMH* full-thickness macular hole, *ILM* inner limiting membrane, *BCVA* best-corrected visual acuity, *logMAR* logarithm of minimum angle of resolution, *N/A* not applicable, *RRD* rhegmatogenous retinal detachment

[a]In the eight eyes without developing FTMH during surgery

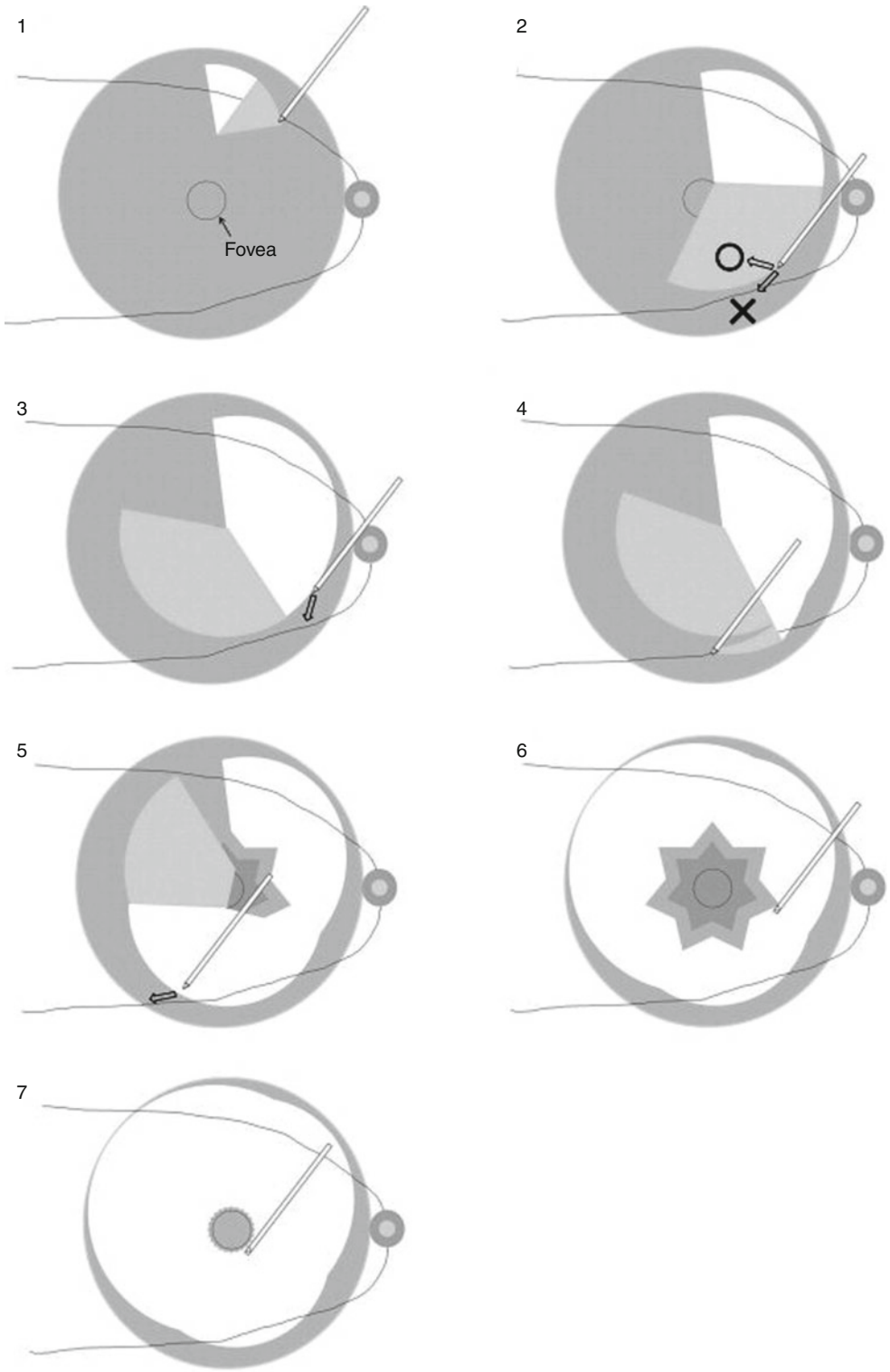


Fig. 16.13 Schematic drawings of fovea-sparing internal limiting membrane (ILM) peeling. *1* Start ILM peeling away from the central fovea. *2* Proceed with the ILM peeling. *3* When the peeled ILM flap comes close to the central fovea, stop and start ILM peeling from a new site. *4* Proceed with ILM peeling from the new site with special attention not to peel the ILM around the central fovea. *5* Start the ILM peeling from several new sites and proceed to peel ILM from the entire macular area away from the central fovea. *6* Trim the ILM that remains on and around the fovea with a vitreous cutter. *7* Completed fovea-sparing ILM peeling

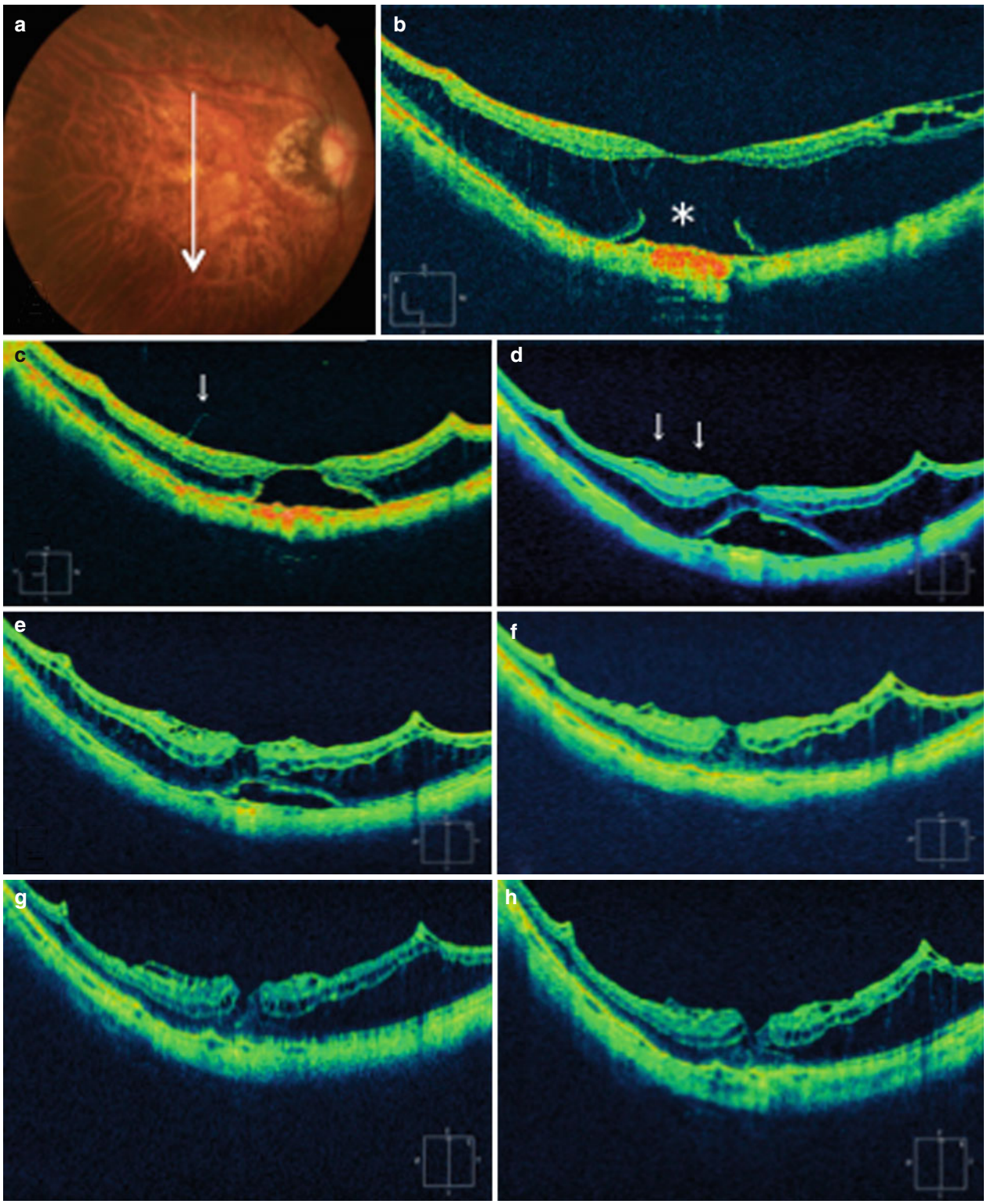


Fig. 16.14 Changes in optical coherence tomographic (OCT) findings after vitrectomy with fovea-sparing internal limiting membrane peeling and gas tamponade to treat myopic foveal retinal detachment (RD). (**a**) Preoperative fundus photograph. An *arrow* indicates the OCT scan line. (**b**) Preoperative OCT image of the same eye. Foveal RD with large outer lamellar macular hole (*asterisk*) and macular retinoschisis can be seen. (**c**) At 1 month after fovea-sparing ILM peeling, the retinoschisis is decreased along with the intraocular gas absorption. The foveal RD is still present although reduced. The rolled edges of the ILM (*arrow*) can be seen. (**d**) At 3 months after surgery, the residual ILM (between *arrowheads*) appears to have contracted and thickened. The retinoschisis is slightly increased and foveal RD is still present. However, the outer lamellar hole has become small. (**e**) At 6 months after surgery, the retinoschisis and foveal RD are still present but decreased. (**f**) At 12 months after surgery, the foveal RD is completely resolved and the retinoschisis is also decreased except at the lower macular area around the retinal artery which is observed as a retinal vascular microfold. (**g**, **h**) At 18 months (*left*) and 24 months (*right*) after surgery, the retinoschisis has been absorbed

traction and less surgical trauma to the central fovea led to a centripetal contraction of the remaining ILM, which is most likely why full-thickness MH did not develop.

The usefulness of scleral buckling with macular plombe for eyes with MRS and foveal RD without MH has also been reported [68–71]. However, complications including chorioretinal atrophy development and preexisting fibrovascular proliferation were found [69], probably due to mechanical pressure and stretching of the retina-choroid by the protrusion of macular plombe. In addition, the posterior scleral reinforcement surgery [72, 73], an intraocular expansible gas, and prone posturing [74, 75] have been reported to treat MRS.

Recently, it has been reported that an intravitreal injection of the vitreolytic agent ocriplasmin resolved vitreomacular traction and closed macular holes [76]. The usefulness of ocriplasmin for an MRS should be investigated in the future.

Also, a new approach involves suprachoroidal buckling using a catheter to deliver long-acting hyaluronic acid into the suprachoroidal space, which creates a choroidal indentation, thereby supporting the macula in the area of the posterior staphyloma. In a study of highly myopic eyes (five with MRS and seven with MHRD) treated with this technique, all patients with MRS achieved anatomical improvement and 4 out of 5 improved vision by at least one line [77]. Among the eyes with MHRD, 57 % showed improvement in visual acuity with no recurrence of RD at 1 year. The long-term outcomes of this approach are currently unknown. The scleral shortening in addition to vitrectomy might also be useful for difficult cases.

16.8 Other Types of Macular Retinal Detachments in Pathologic Myopia

16.8.1 Macular Hole Retinal Detachment (MHRD)

RD resulting from full-thickness MH occurs most commonly in highly myopic eyes [60, 63, 78]. Ripandelli et al. [43] reviewed OCT findings of 214 eyes with pathologic myopia (axial length >30 mm and posterior staphyloma) and found full-thickness MH in 18 eyes (8.4 %). FTMH in highly myopic eyes are sometimes asymptomatic. Coppe et al. [79] examined 373 highly myopic patients with no visual disturbance and found FTMH in 24 eyes (6.26 %). The absence of symptoms could be related to the localization of the hole in a juxtafoveal area. Akiba et al. [63] retrospectively analyzed 52 consecutive eyes with MH and severe myopia and found that an extensive RD was observed in 37 eyes (71 %). Morita et al. [61] found that the incidences of MHRD were 97.6 % in myopia over −8.25 D, 67.7 % in myopia between −8.0 and −3.25 D, and 1.1 % in eyes under −3.0 D; 100 % in widespread chorioretinal atrophy, 90.6 % in spotty or linear chorioretinal atrophy, 64.3 % in myopic tigroid fundus, and 0 % in eyes without myopic tigroid or atrophy; and 96.0 % in eyes with posterior staphyloma and 8.2 % in eyes without staphyloma. Oie and Ikuno [80] reported that among various types of staphyloma by Curtin, the rate of type II staphyloma was significantly higher in the Japanese patients with MHRD. The fellow eyes of MHRD are reportedly at high risk of MHRD [81–83]. Oie and Emi [82] analyzed the fellow eyes of MHRD and found that the incidence of MHRD among the highly myopic fellow eyes was 12.8 %.

Despite the surgical interventions, MHRD is still one of the most difficult types of RD to treat, with poor visual prognosis [84]. Nonclosure or reopening of the MH and RD may still develop, and in some patients, anatomical success may require multiple procedures.

MHRD in highly myopic eyes has been reported to develop following to some medical interventions like hypotony after trabeculectomy [85], after YAG laser capsulotomy [86], after cataract surgery [87], after clear lens extraction [88], and after LASIK [89, 90]. Shimada et al. [12] reported that a macular hole was detected by OCT in 14 % of the eyes with CNV and large chorioretinal atrophy (>1 disc area). The hole always existed at the border between an old CNV and the surrounding chorioretinal atrophy. RD developed in 89 % of eyes with complete PVD; thus, it is suggested that posterior staphyloma rather than anteroposterior vitreomacular traction may contribute to the development of RD associated with MH in severely myopic eyes.

Surgical treatments for MHRD have been reported in many studies. The most common procedures appear to include the vitrectomy, removal of adherent vitreous cortex, removal of ERM, fluid-gas exchange, and intraocular gas (or silicone oil, if necessary) tamponade [78, 87, 91–98], although some studies reported the successful reattachment of MHRD without removal of ILM. Nakanishi et al. [99] analyzed prognostic factors in PPV for initial reattachment of MHRD and found that an axial length was the only significant prognostic factor for initial reattachment after PPV with gas tamponade for MHRD in high myopia. Jo et al. [31] reported that the presence of MRS negatively impacts the visual and anatomical prognosis of vitrectomy for full-thickness MH in highly myopic patients. The effectiveness of macular buckling has also been reported [100]. The inverted ILM flap technique was originally reported for idiopathic macular hole by Michalewska et al. [101]. In this technique, the ILM was not removed completely from the retina during vitrectomy but was left attached to the edge of the MH. The ILM was then massaged gently over the MH so that the MH was covered with the inverted ILM flap. Recently, Kuriyama et al. [102] reported that was effective for a closure of myopic macular hole. Further studies are expected to confirm the effectiveness of this new technique.

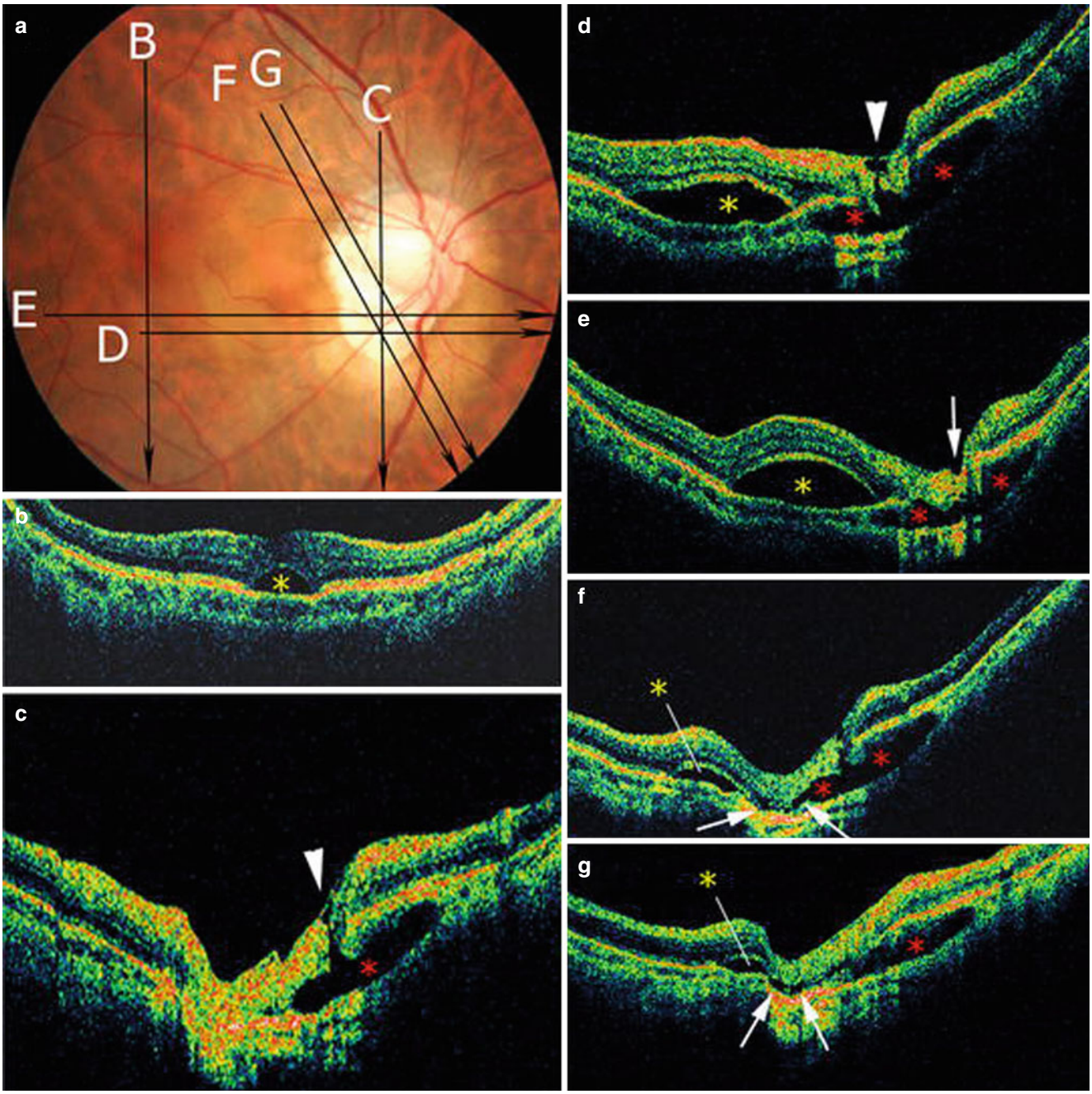


Fig. 16.15 Optical coherence tomographic images of the macular retinal detachment and intrachoroidal cavitation (ICC). (**a**) Each *line* shows an optical coherence tomography (OCT) scan for the images shown in (*B–F*) (scan length, 10 mm). (**b**) Vertical OCT section across the central fovea showing a retinal detachment (*asterisk*). (**c**). Vertical OCT section through the hole-like lesion within the area of chorioretinal atrophy showing a full-thickness defect at the hole-like lesion (*arrowhead*). The vitreous cavity is connected to the intrachoroidal cavity through this defect, suggestive of ICC (*red asterisk*). (**d**) Horizontal OCT section through the hole-like lesion within the area of chorioretinal atrophy shows an empty space at this lesion (*arrowhead*). The vitreous cavity is connected to the intrachoroidal cavity through this defect, suggestive of ICC (*red asterisks*). Retinal detachment is also detected (*yellow asterisk*) and is separated from ICC by the retinal pigment epithelial (RPE) layer. (**e**) Horizontal OCT section across the hole-like lesion at the border between myopic conus and chorioretinal atrophy shows a small hollow of the retina at the site (*arrow*). A full-thickness retinal defect is not observed in any adjacent sections. Intrachoroidal cavity suggestive of ICC (*red asterisks*) and retinal detachment (*yellow asterisk*) can also be seen. (**f**) Oblique OCT section through the hole-like lesion within the chorioretinal atrophy shows that the ICC (*red asterisks*) seems continuous with the retinal detachment (*yellow asterisk*) through the outer retinal schisis-like path at the conus area (*arrows*). (**g**) In the adjacent section of (**h**) the continuity of ICC with macular RD through the outer retinal schisis-like path is clearly observed

16.8.2 Macular RD Associated with Peripapillary Intrachoroidal Cavitation (ICC)

ICC is a yellowish-orange lesion inferior to the optic disc seen in 4.9 % of highly myopic eyes [37–40]. Spaide et al. [40] demonstrated the posterior deformation of the sclera in the area of ICC (see "choroid" chapter). Shimada et al. [103] reported a case with high myopia in which a macular RD was accompanied with peripapillary ICC. In their patient, OCT examination revealed that the vitreous cavity was connected to the ICC space through a full-thickness tissue defect in the retina overlying the ICC, and the ICC was continuous with the RD through the subretinal path at the conus area (Fig. 16.15). This suggested that the eyes with peripapillary ICC might be at risk of developing macular RD. Akimoto et al. [104] reported a similar case with macular RD and peripapillary ICC and without high myopia. Recently, Yeh and colleagues [105] analyzed 122 eyes with peripapillary ICC and found that 26.2 % of the eyes with ICC were not highly myopic (<−6 D). These suggest that peripapillary ICC was not exclusive to highly myopic eyes, and peripapillary structural alterations like disc tilting might relate to the ICC development and subsequent formation of ICC-related macular RD.

16.8.3 RD Caused by a Retinal Break in and Along the Macular Atrophy or Patchy Atrophy

In addition to the MH, a retinal break reportedly develops within or along the margin of macular atrophy [106, 107]. Macular atrophy develops around the regressed CNV in the atrophic stage of myopic CNV [108, 109]; thus, these suggest that myopic CNV acts on the development of MRS and RD in various manner and in various stages of CNV. Chen et al. [107] reported cases which developed RD caused by paravascular linear retinal breaks over areas of patchy atrophy. Considering that patchy chorioretinal atrophy contributes on the development of MRS by forming macular ICC, patchy atrophy also acts on the development of MRS and RD in various manners, as seen in macular atrophy.

16.9 Closing Remarks

MRS is the macular lesion which was most recently identified by new imaging modalities in highly myopic eyes. However, after its discovery, the pathologies, the pathogenesis, and treatment options have been greatly investigated due to the advance of OCT technologies as well as the surgical techniques. It is certain that there will be a great improvement for understanding this pathology and preventing a vision loss due to MRS in the near future.

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