CHAPTER 9

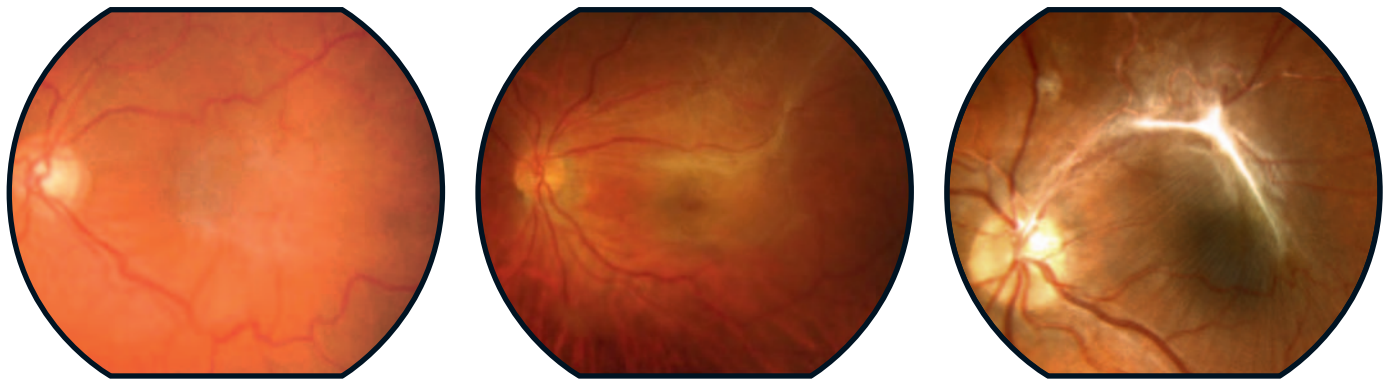
c00009

Vitreomacular Traction, Epiretinal Membranes, and Macular Holes

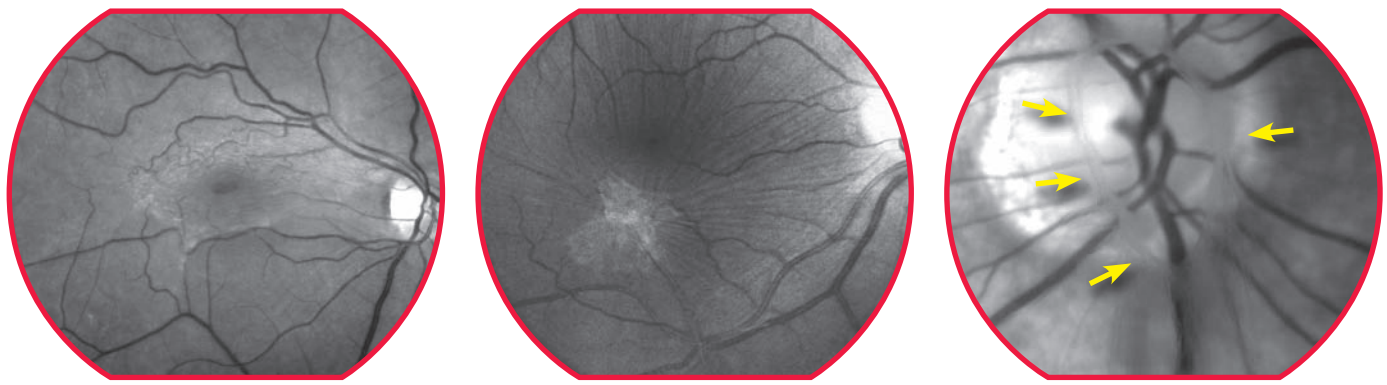
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

Epiretinal Membrane

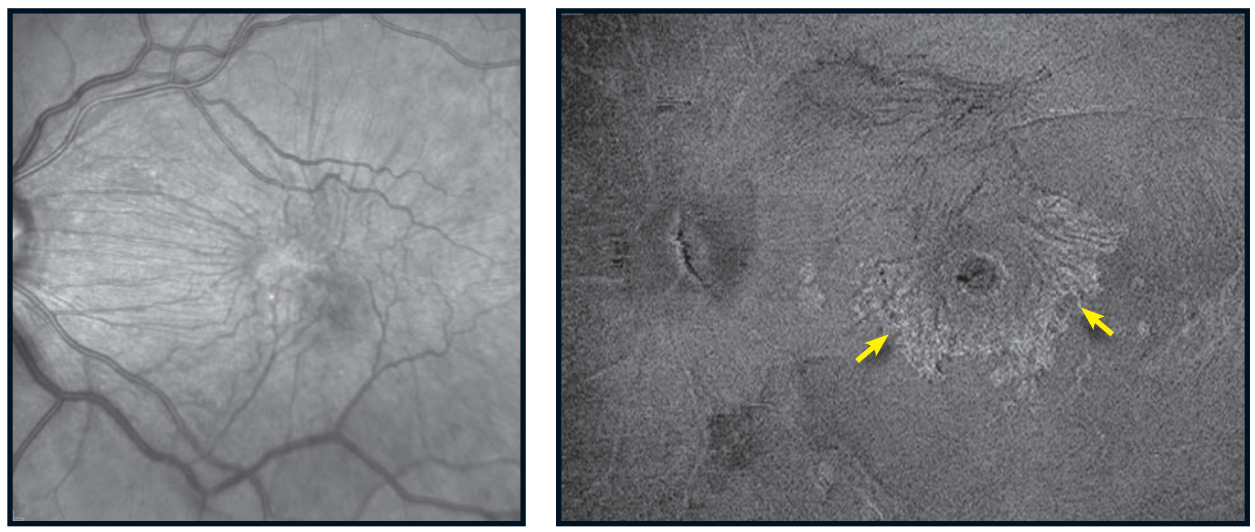
p0040 An epiretinal membrane (ERM) is a fibrocellular proliferation occurring on the retinal surface, most commonly in the macular region. ERMs typically occur following a spontaneous partial or complete posterior vitreous detachment, but secondary causes include intraocular surgery, inflammation, ischemic vascular disease, trauma, retinal tear, rhegmatogenous retinal detachment, and intraocular tumors. Cells thought to contribute to ERMs include retinal pigment epithelium (RPE), fibrocytes, myofibrocytes, and intraretinal glial elements.



f0010 These patients show the variable presentation of ERMs, ranging from semi-translucent gray *(left image)* to opaque white fibrosis *(middle image)*. Some may appear as a fibrotic white band *(right image)*. The appearance during the early stages of an ERM is frequently referred to as "cellophane maculopathy," while prominent surface wrinkling following membrane maturation is referred to as "macular pucker."

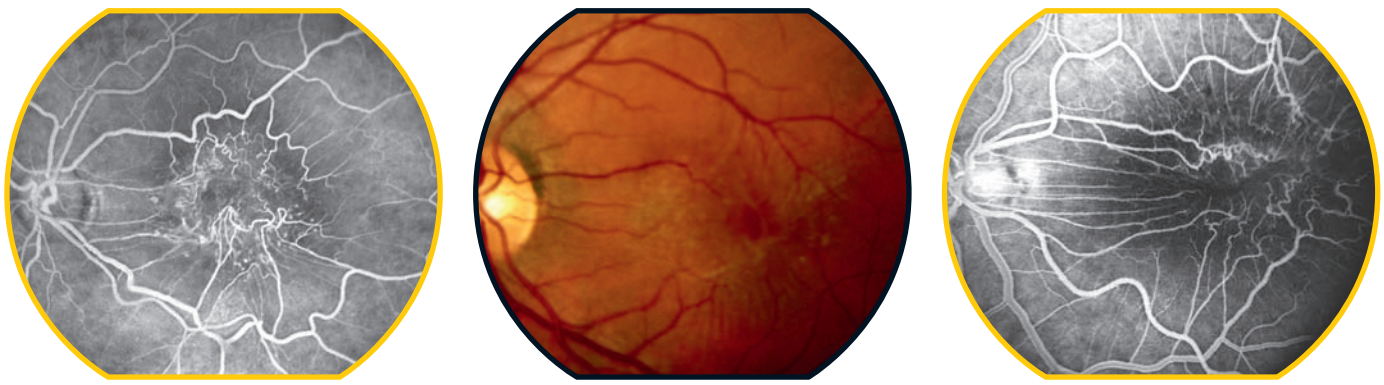


f0015 Monochromatic imaging (red-free) can enhance the details of the vitreoretinal interface. Commonly, there is incomplete detachment of the posterior hyaloid with persistent adherence around the disc in the right image *(arrows)*.



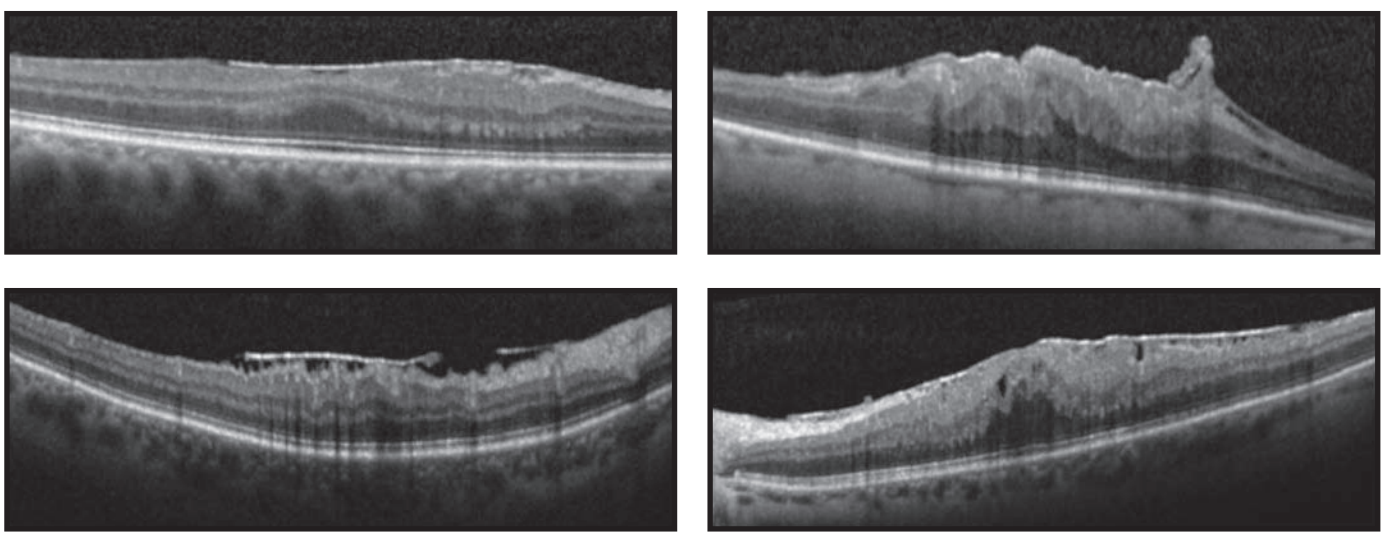
f0020 Infrared reflectance imaging *(left)* and *en face* swept source optical coherence tomography (OCT) imaging *(right)* also provide valuable information about the morphologic and topographic features of ERMs. The margins of the ERM are clearly delineated on swept source images *(arrows)*. *Images courtesy of Dr. Michael Engelbert*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009



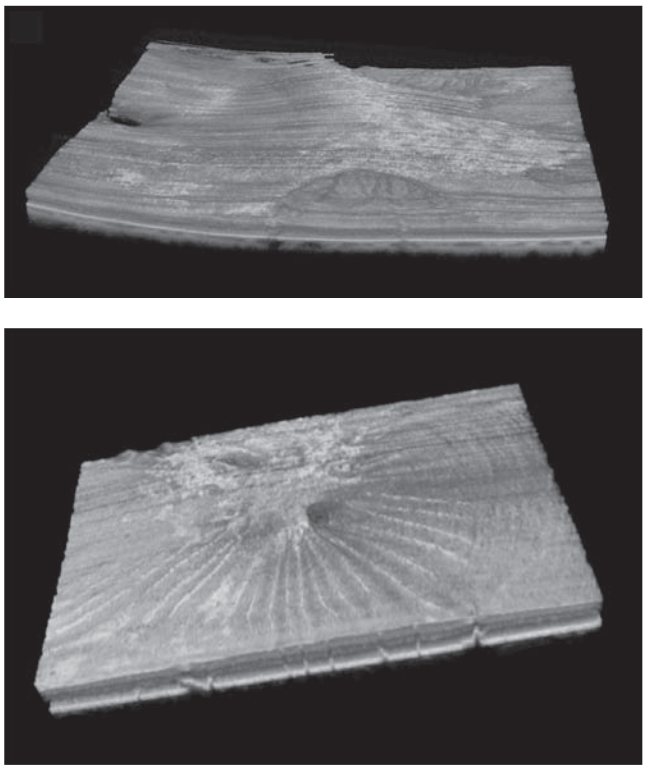
f0025

Fluorescein angiography can aid surgical planning and is useful for identifying complex anatomic relationships between retinal vasculature and ERMs. In the above cases, retinal vasculature is observed to be embedded within the ERM tissue complex. Fluorescein angiography is also useful for excluding secondary causes of ERM, such as retinal vein occlusion.



f0030

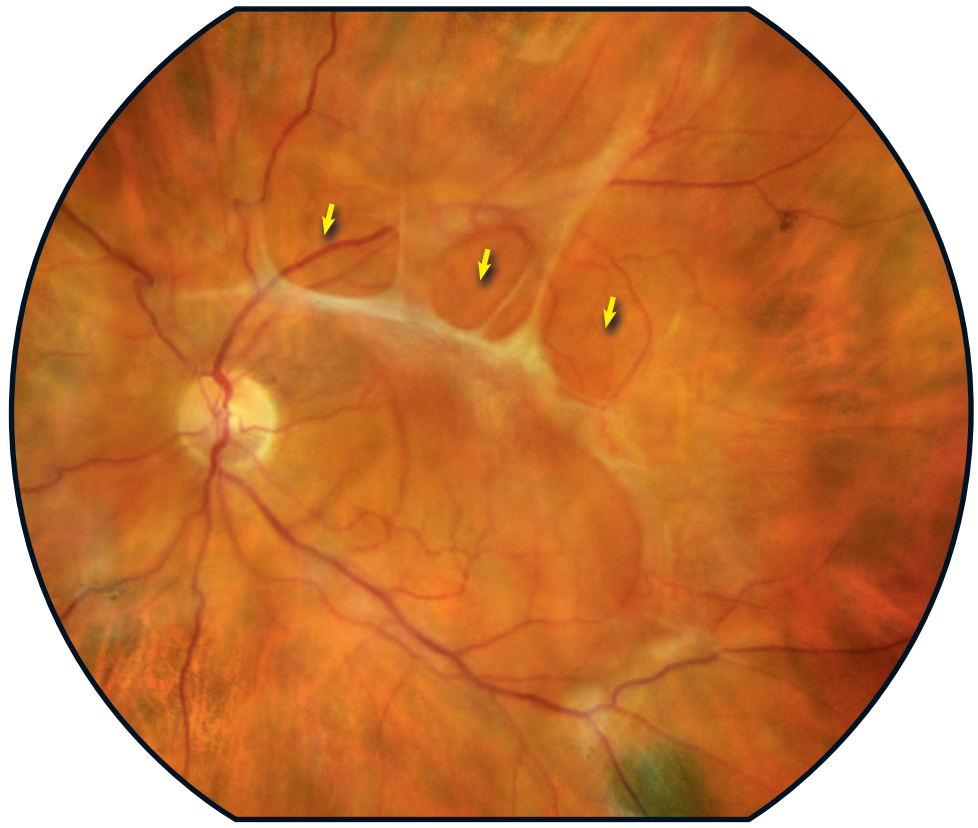
With spectral-domain OCT imaging, an ERM appears as a thin, hyper-reflective line on the inner surface of the retina *(top left image).* Tractional effects of the membrane include retinal thickening *(top right image),* wrinkling of the surface of the retina *(bottom left image),* and intraretinal cystic spaces *(bottom right image).* These tractional effects are responsible for the patient's symptoms of visual loss and metamorphopsia.



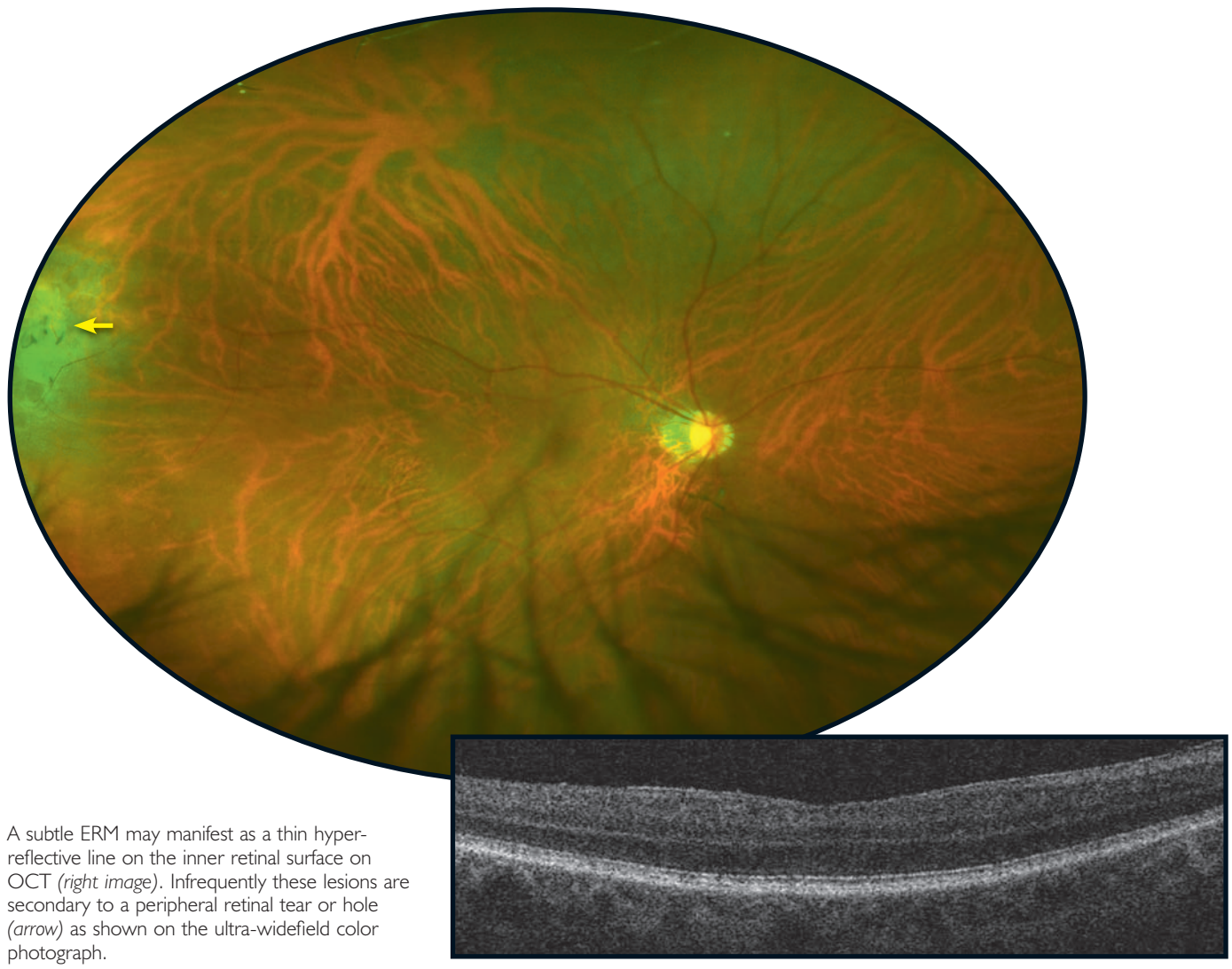
f0035

Three-dimensional volume rendered OCT images demonstrate wrinkling and traction of the inner surface of the retina due to ERM. *Images courtesy of Dr. Richard Spaide*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

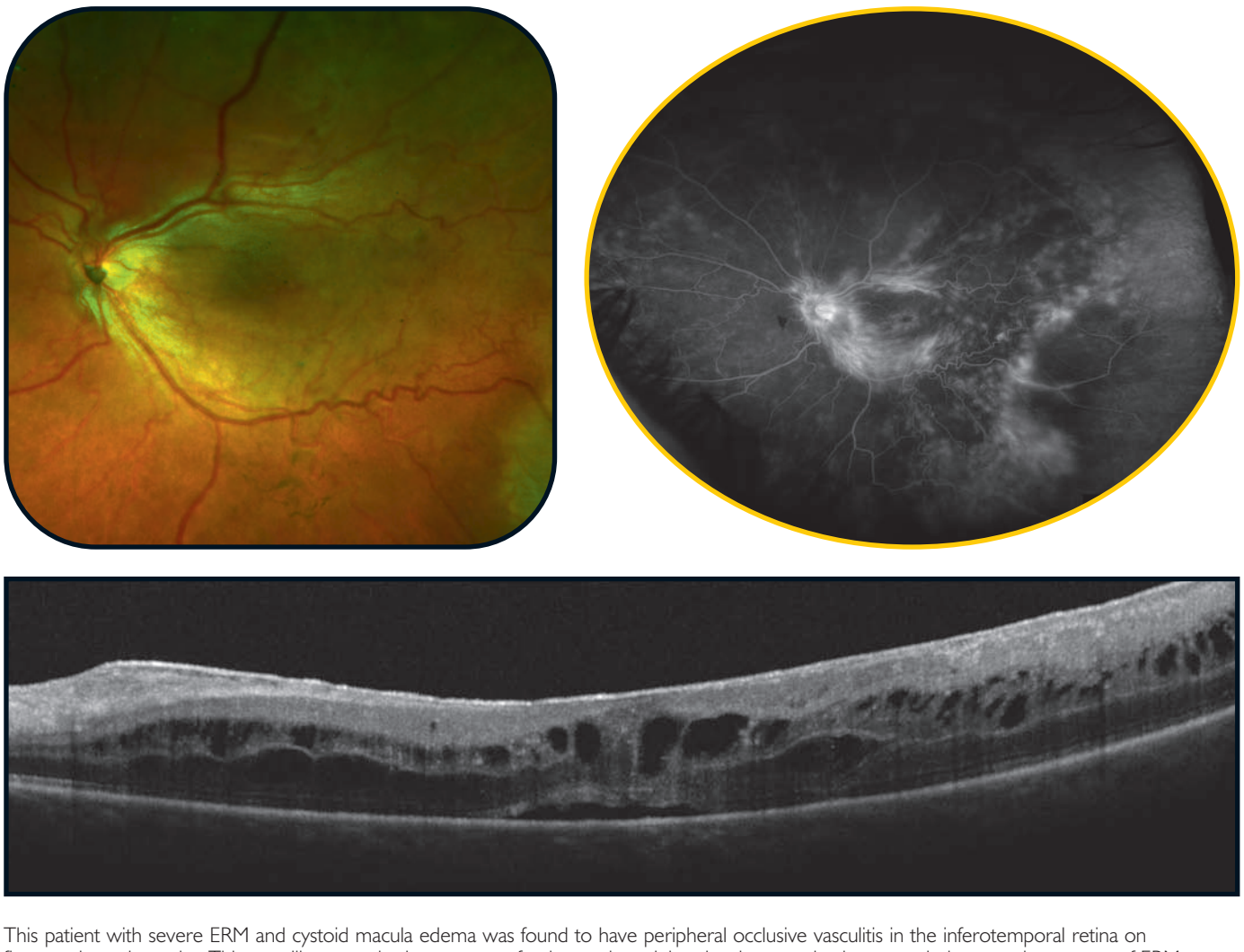


f0040 Note the widespread fibrotic membrane in this patient. There are semilunar, ovoid, and circular defects in the epiretinal tissue *(arrows)*.



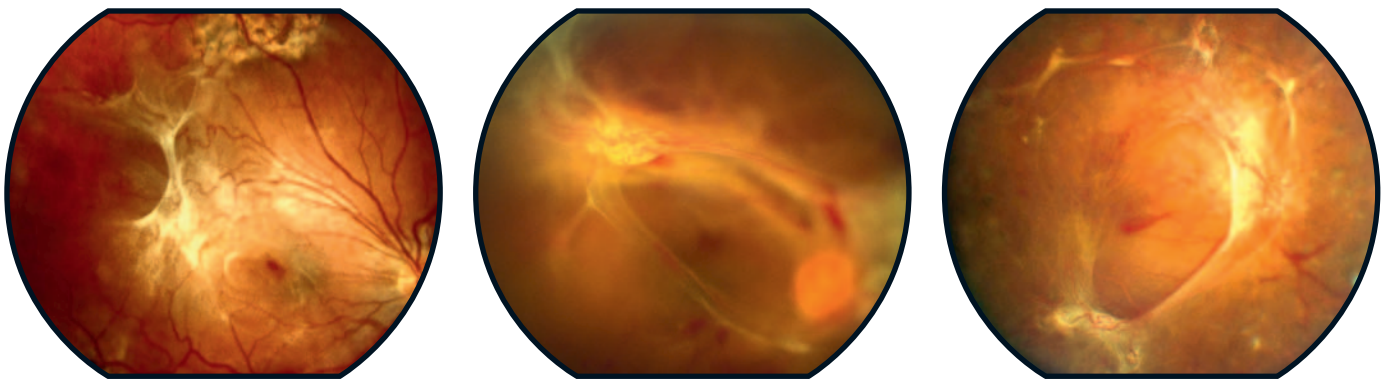
f0045 A subtle ERM may manifest as a thin hyperreflective line on the inner retinal surface on OCT *(right image)*. Infrequently these lesions are secondary to a peripheral retinal tear or hole *(arrow)* as shown on the ultra-widefield color photograph.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009



f0050

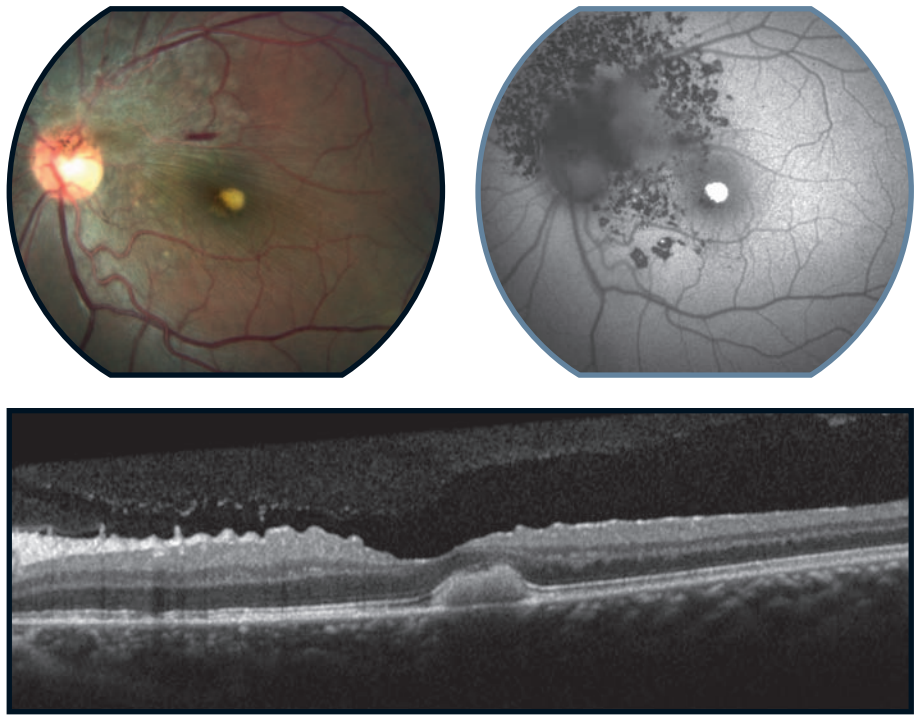
This patient with severe ERM and cystoid macula edema was found to have peripheral occlusive vasculitis in the inferotemporal retina on fluorescein angiography. This case illustrates the importance of a thorough peripheral retina examination to exclude secondary causes of ERM. *Images courtesy of Dr. David Maberley*



f0055

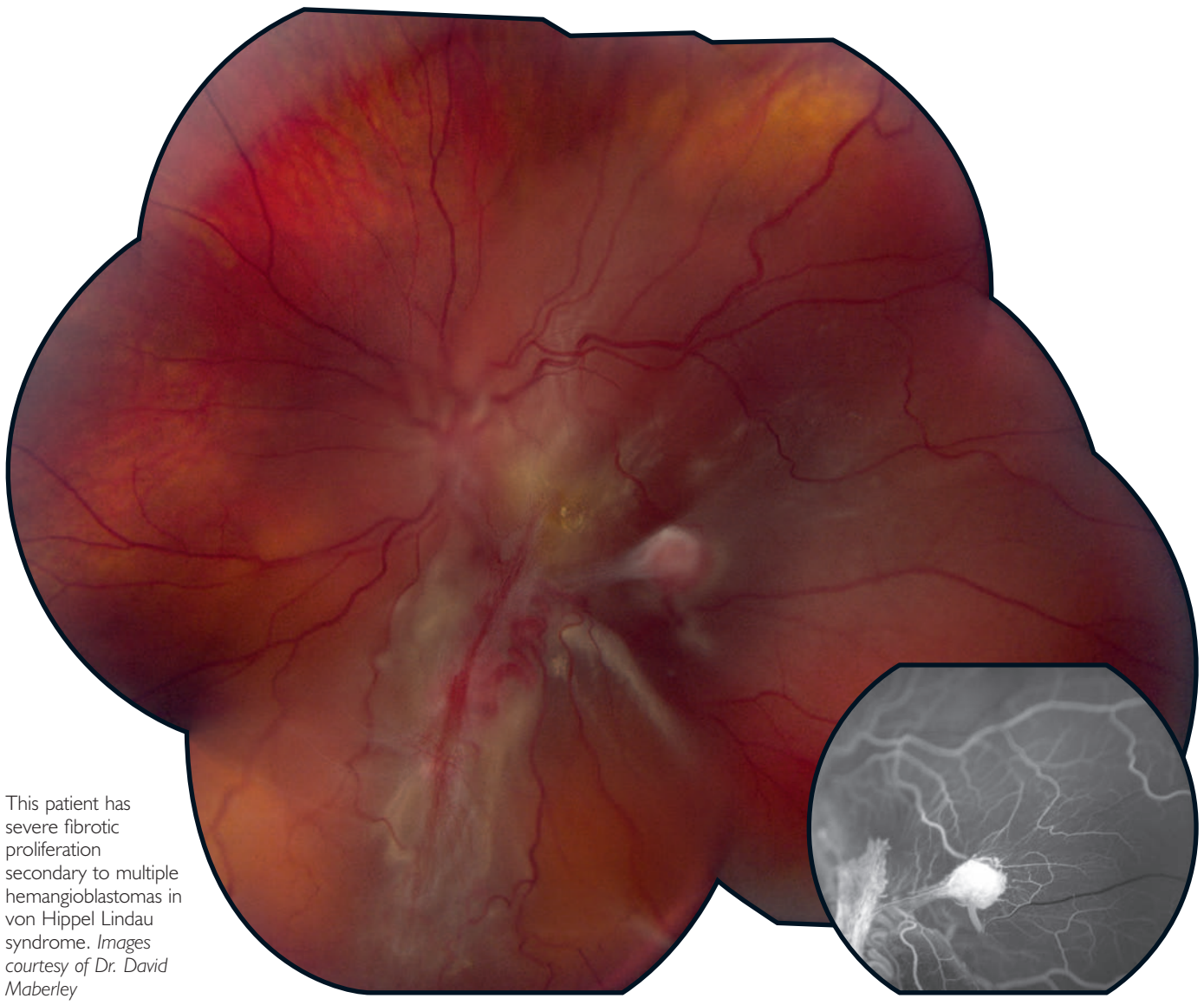
The above patients with proliferative diabetic retinopathy have severe fibrotic proliferation. The fibrovascular tissue usually adopts a curvilinear distribution along the vascular arcades.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009



f0060

This patient has an ERM surrounding the optic disk secondary to a combined hamartoma of the retina and retinal pigment epithelium. The OCT shows a hyper-reflective vitelliform lesion that exhibits hyperautofluorescence at the fovea with fundus autofluorescence imaging. Folds on the surface of the macula are due to tractional forces around the optic disk.



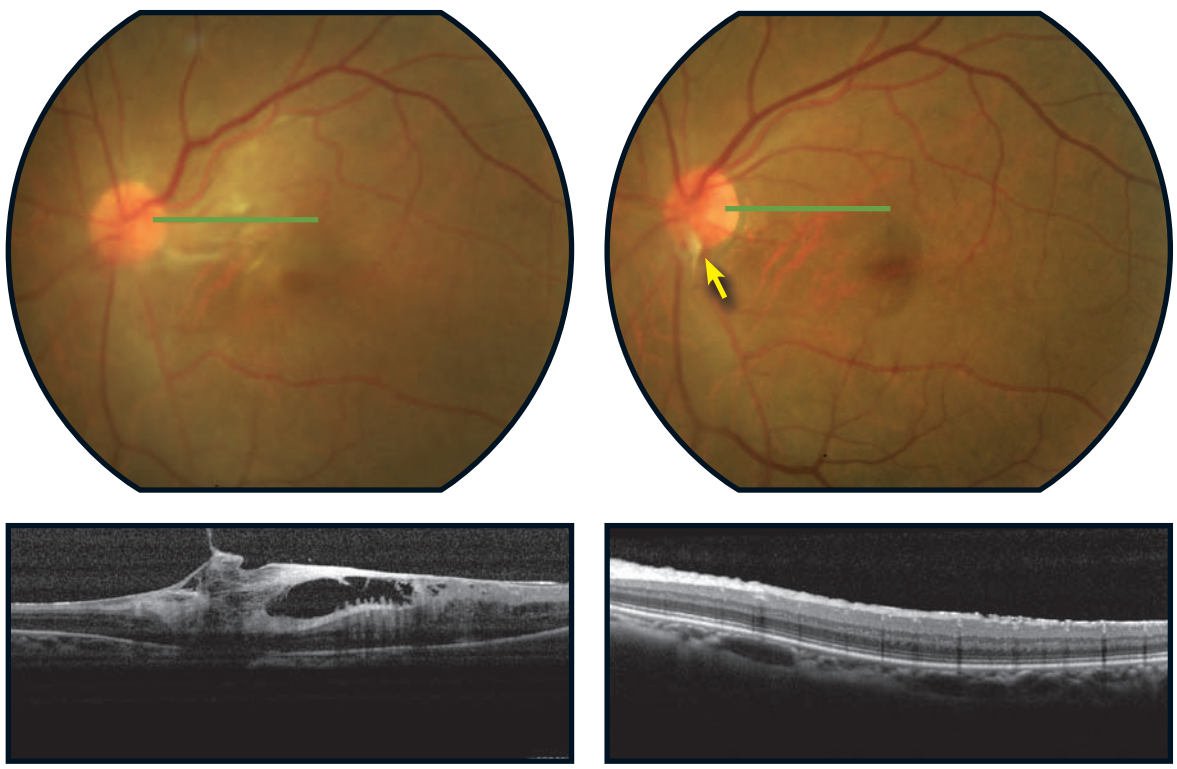
f0065

This patient has severe fibrotic proliferation secondary to multiple hemangioblastomas in von Hippel Lindau syndrome. *Images courtesy of Dr. David Maberley*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

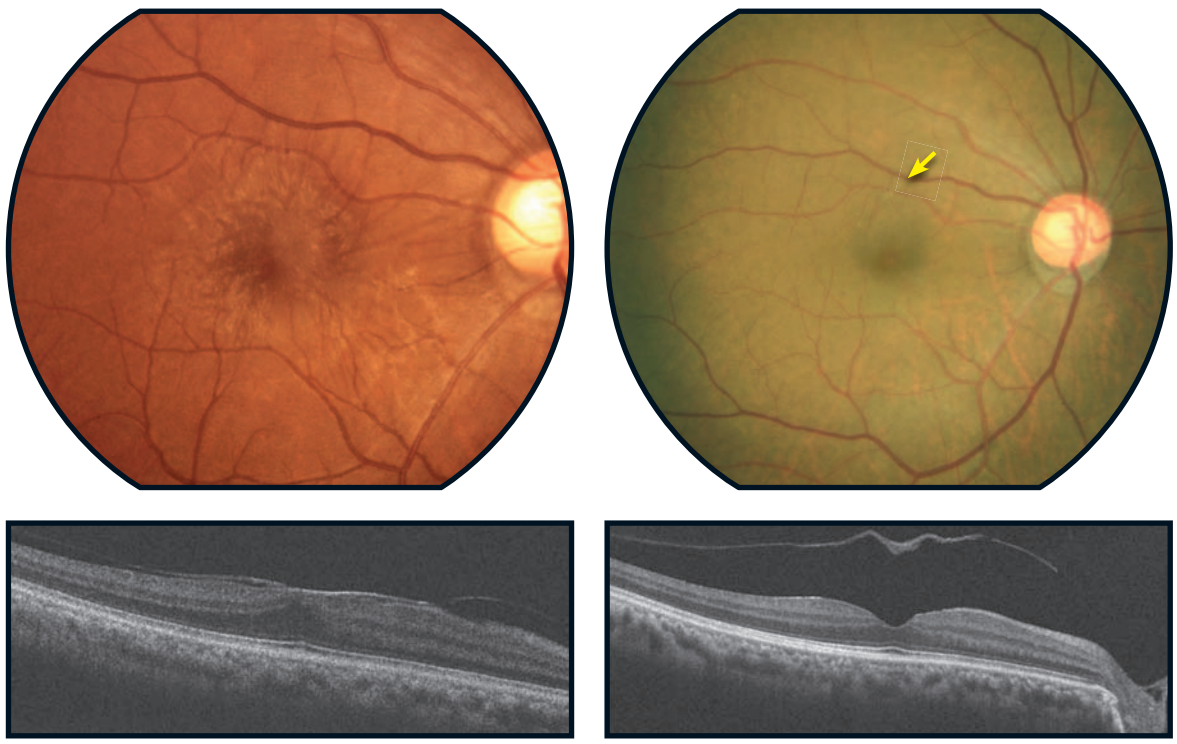
s0015 Spontaneous Release of Epiretinal Membrane

p0045 Cases of spontaneous release of ERMs have been reported. Although uncommon, occurring in approximately 1-3% of cases, it is more frequently observed in young, female, myopic patients.



f0070

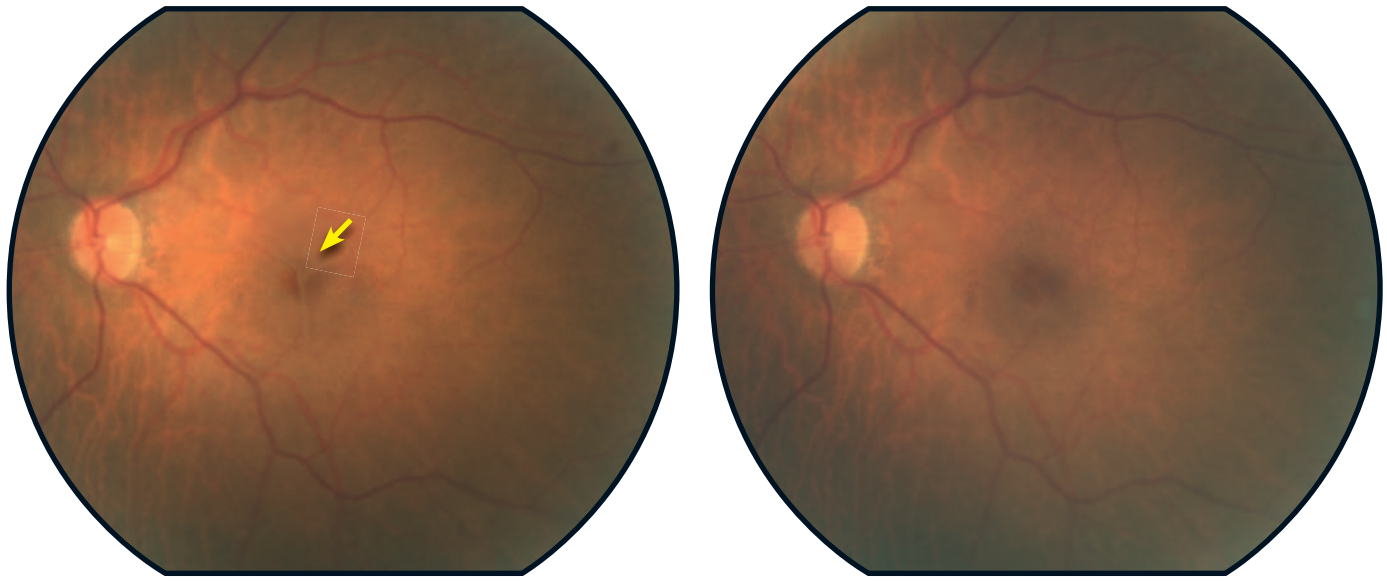
This patient had a thick ERM at the disc and papillomacular bundle *(left)*. The membrane released spontaneously, leaving a legacy of peripapillary fibrous tissue *(arrow)*.



f0075

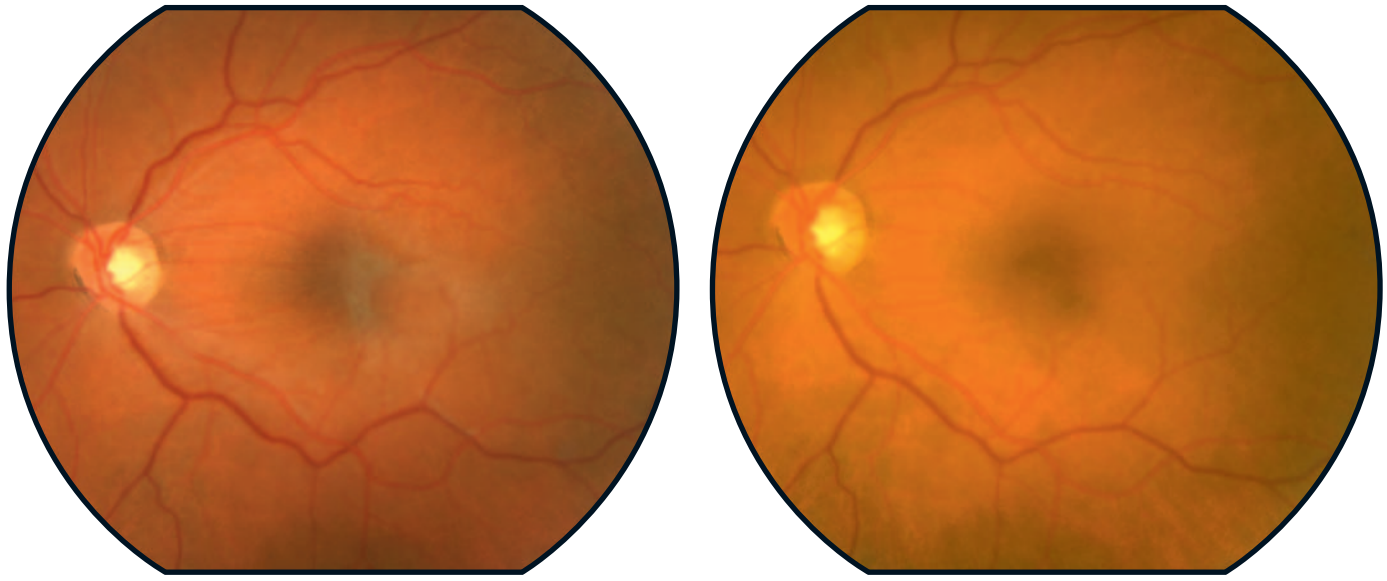
This patient with metamorphopsia due to an ERM experienced spontaneous improvement of symptoms four years later. Color photos and OCT demonstrate spontaneous release of the ERM from the macula. A small remnant of fibrotic tissue is seen along the superior arcade *(arrow)*.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009



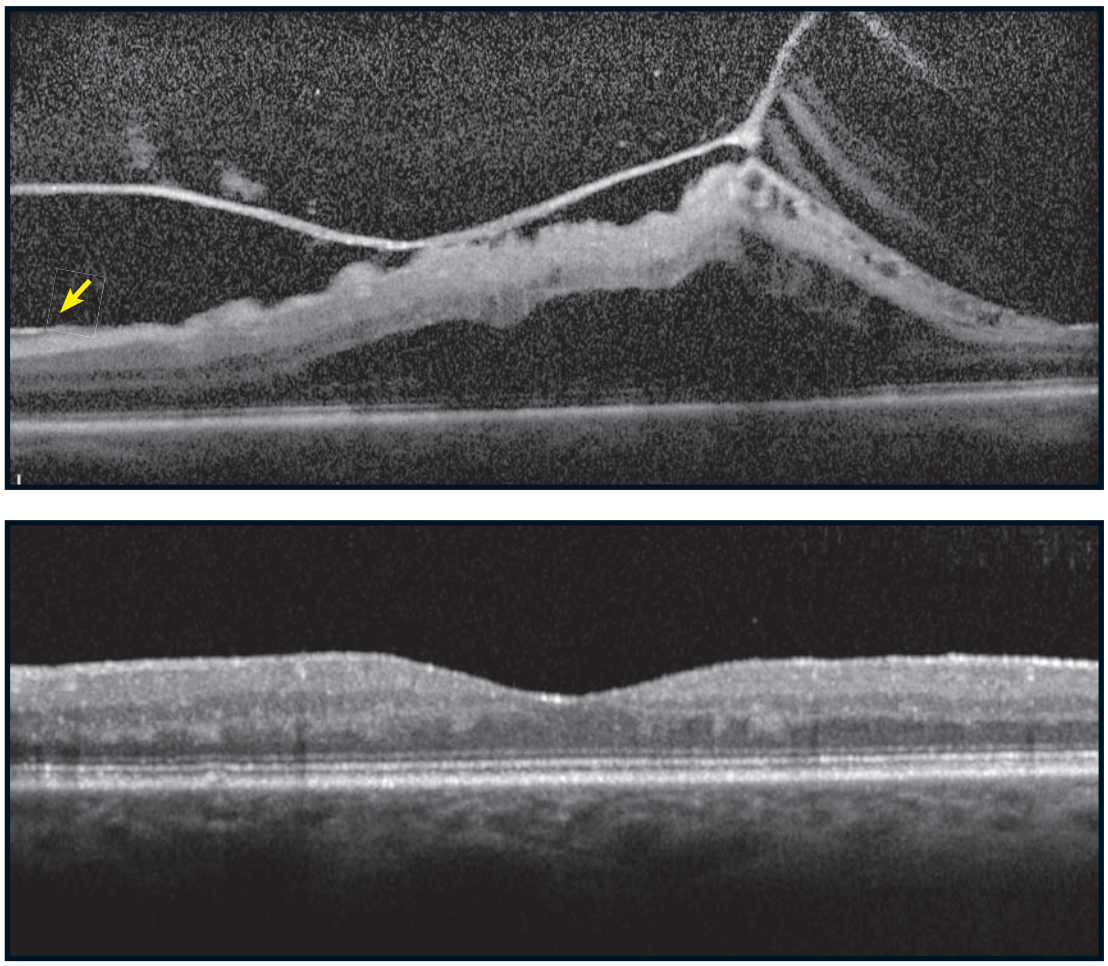
f0080 The above patient experienced subjective improvement in metamorphopsia and was noted to have spontaneous release of a macular ERM *(arrow)* that was present on the initial examination. Image on the right was taken 1 month after the baseline examination. *Images courtesy of Dr. Michael Engelbert*

s0020 Surgical Treatment



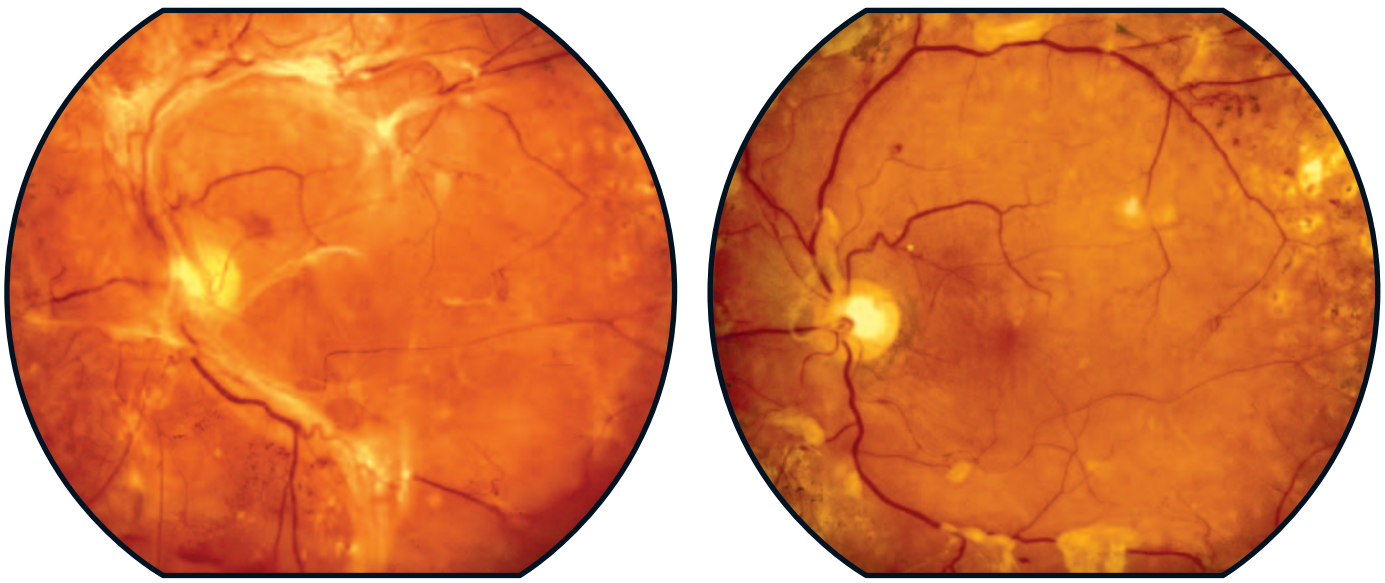
f0085 Patients with visually significant ERMs may be treated with pars plana vitrectomy and membrane peeling. Pre-operative *(left)* and post-operative *(right)* images are provided from a patient that presented with a visually significant ERM. Visual acuity was measured as 20/80 pre-operatively and returned to 20/25 following membrane peeling. *Images courtesy of Dr. Michael Engelbert*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009



f0090

Anatomical outcomes following surgical intervention are best evaluated using OCT. This patient suffered disabling metamorphopsia and visual reduction due to traction from a broad vitreomacular adhesion (VMA). An associated ERM is also seen in the pre-operative OCT *(arrow)* as is frank intraretinal edema. Pre-operative visual acuity was measured as 20/100. Two years following vitrectomy and membrane peeling *(bottom image)*, the normal foveal contour was restored and visual acuity has returned to 20/25. *Images courtesy of Dr. Michael Engelbert*



f0095

Surgical management of tractional membranes due to systemic vascular diseases such as diabetes mellitus can be more challenging. In the above patient, multiple points of macular traction, secondary to proliferative diabetic retinopathy, are evident. The surgical goal in this instance is to release all points of macular traction as illustrated in the post-surgical image on the right. Remnants of fibrous tissue can be seen on the surface of the retina; however, these structures are typically not visually significant. *Images courtesy of Dr. Yale Fisher*

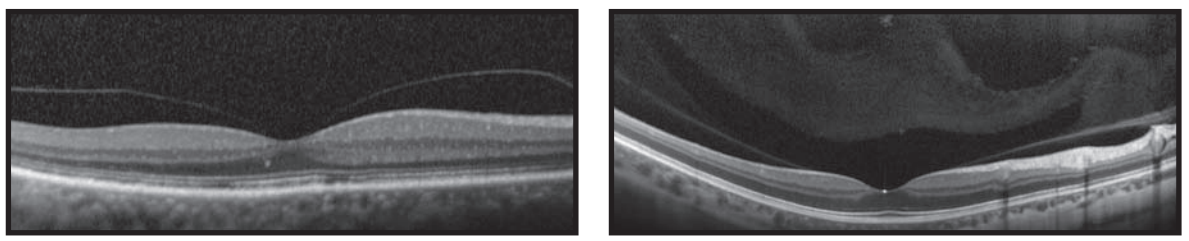
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

Vitreomacular Traction (VMT)

p0055 Vitreomacular traction (VMT) is defined as persistent vitreous attachment to the central macula due to an incomplete posterior vitreous detachment. Histologically, VMT specimens obtained from surgery show a variety of cell types including fibrous astrocytes, myofibroblasts, and fibrocytes, similar to those found in ERMs. In fact, many eyes with VMT have a concurrent ERM and there is considerable overlap between the two entities. With the advent of spectral-domain OCT and vitreolytic agents, an International Vitreomacular Traction Study Classification System for VMA and VMT has been proposed.

Vitreomacular Adhesion (VMA)

p0060 VMA is defined as elevation of the cortical vitreous above the retina surface with the vitreous remaining attached within a 3 mm radius of the fovea. There is no change to the inner retina contour on OCT. VMA can be further subclassified by the size of adhesion into focal or broad.



f0100 These patients have focal VMA ≤1500 μm *(left image)* and broad VMA > 1500 μm *(right image)*.

Vitreomacular Traction (VMT)

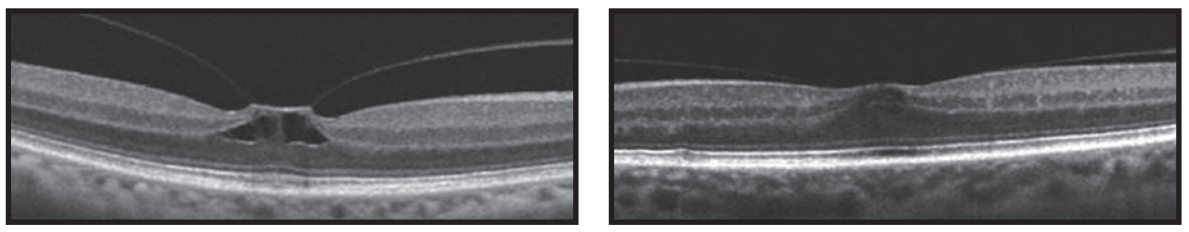
p0065 In VMT, all of the following criteria must exist:

p0040 (1) Perifoveal vitreous cortex detachment from the retinal surface

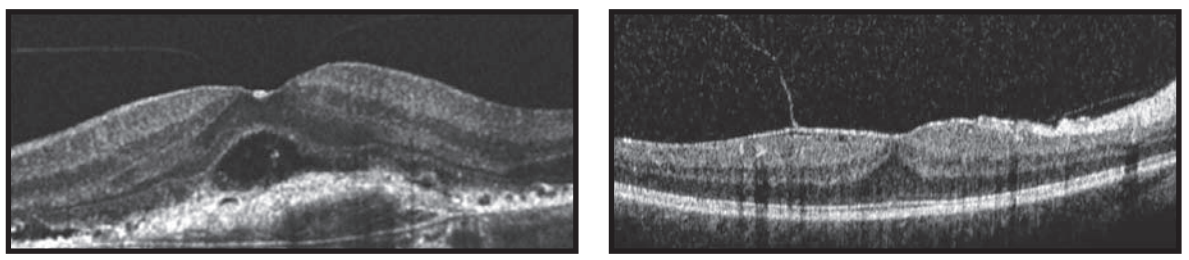
p0045 (2) Macular attachment of the vitreous cortex within a 3 mm radius of the fovea

p0050 (3) Distortion of the foveal surface, intraretinal structural changes, elevation of the fovea above the RPE or a combination thereof, without full-thickness interruption of retinal layers at sites of vitreous adhesion.

Like VMA, VMT can be further subclassified by the size of adhesion into focal or broad.



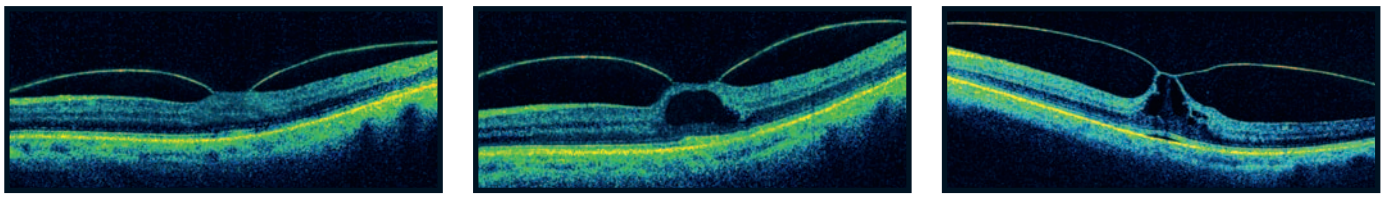
f0105 These patients have focal VMT ≤1500 μm *(left image)* and broad VMT > 1500 μm *(right image). Images courtesy of Dr. Jay Duker*



f0110 VMA and VMT may occur concurrently with other macular abnormalities including age-related macular degeneration *(left image),* retinal vein occlusion, or diabetic macular edema. Eyes with VMT frequently have a concurrent ERM *(right image). Images courtesy of Dr. Jay Duker (left) and Dr. Edwin Ryan (right)*

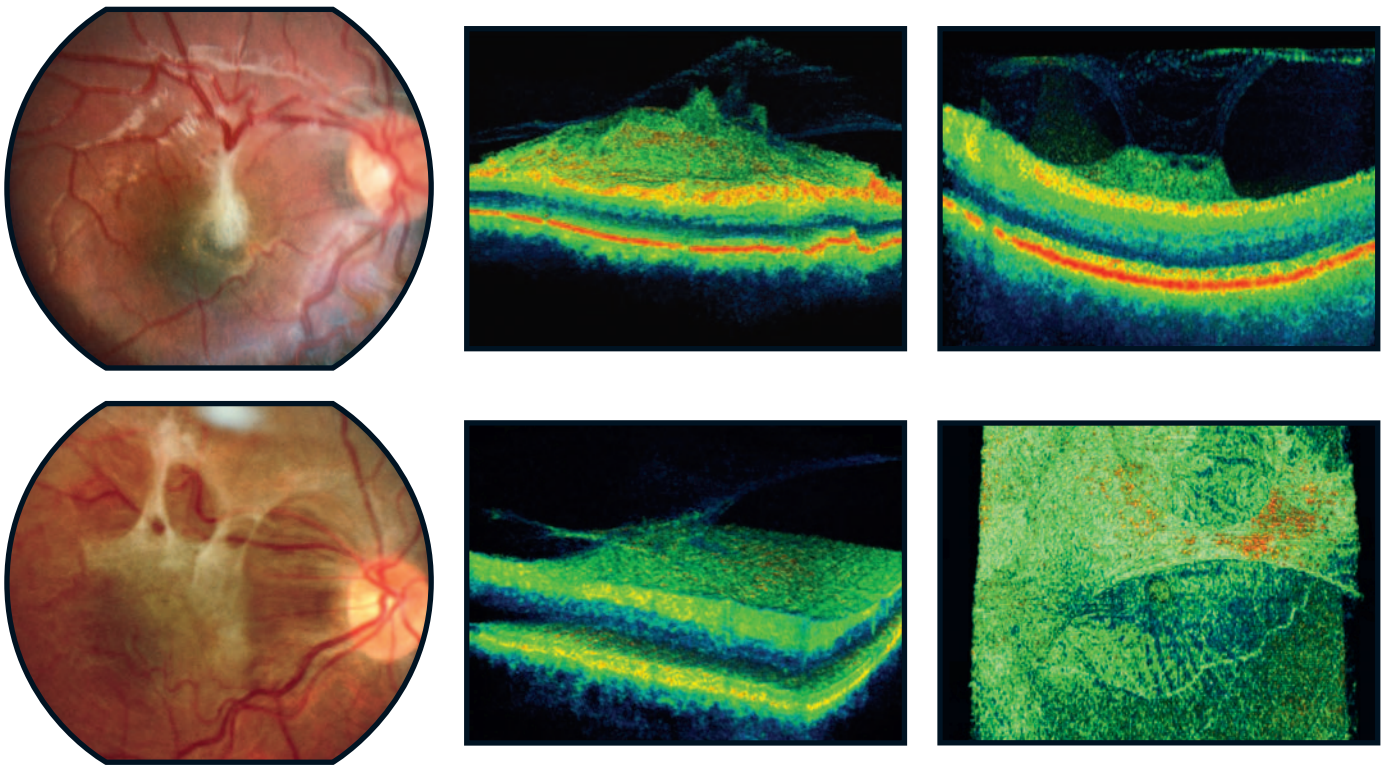
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

f0115



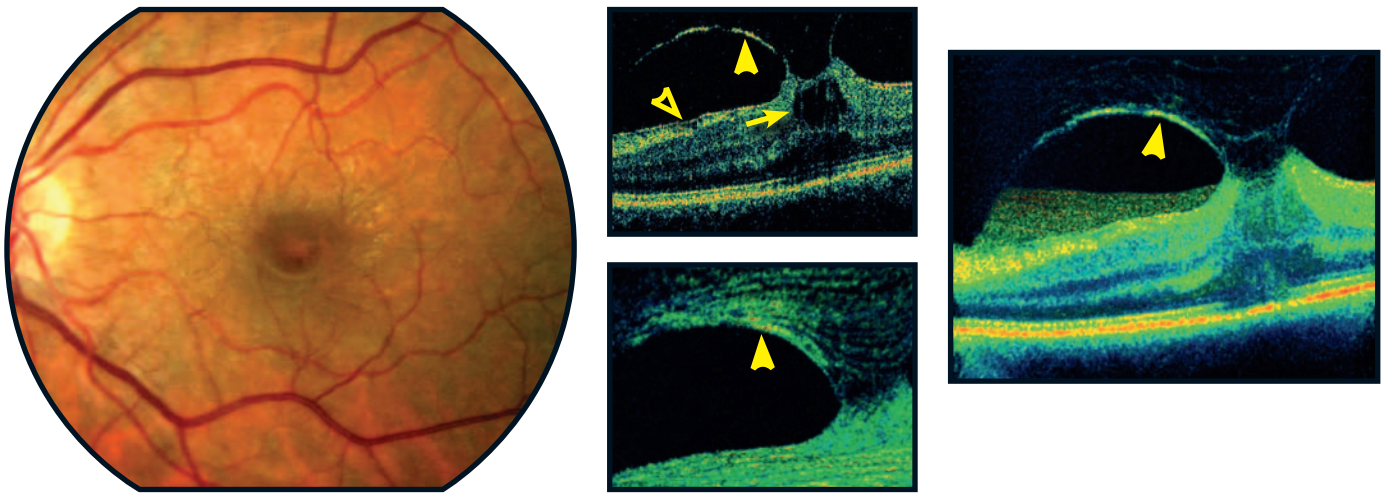
VMT can be graded according to the severity of the tractional effect on the retinal layers. In grade 1, there is elevation of the retina but no split in the retinal layers *(left image)*. In grade 2, there are intraretinal cysts, clefts, or schisis *(middle image)*. In grade 3, there is neurosensory elevation of the retina above the RPE resulting in subretinal fluid *(right image)*. *Images courtesy of Dr. Harry Flynn*

f0120



Three-dimensional OCT images show broad-based VMT with retinal thickening. These images may facilitate surgical planning of membrane peeling by the vitreoretinal surgeon. *Images courtesy of Dr. Hideki Koizumi*

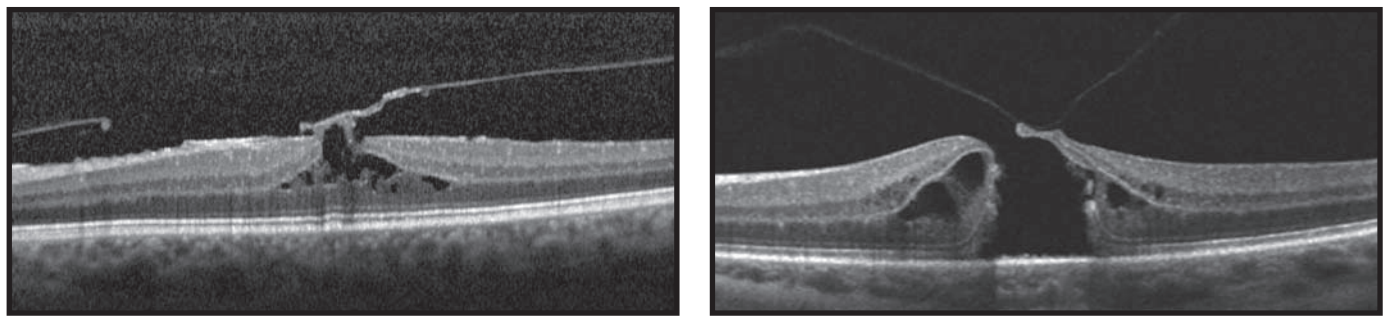
f0125



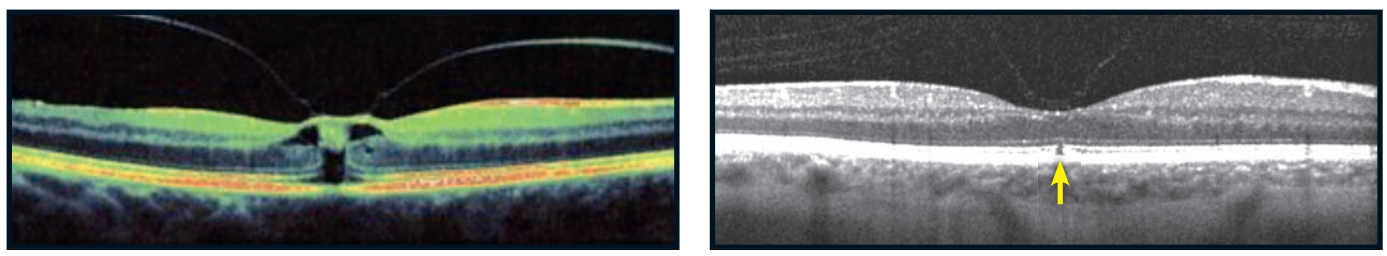
Three-dimensional OCT images show VMT occurring concurrently with an epiretinal membrane. The epiretinal membrane appears as a thin, reflective line above the jagged inner retinal surface *(open arrowhead)*. There is associated retinal thickening, intraretinal cysts *(arrow)*, and subretinal fluid. There are curvilinear bands of fibrous traction or "hourglass" plaques seen with a funnel-like traction on the retina *(arrowheads)*. *Images courtesy of Dr. Hideki Koizumi*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

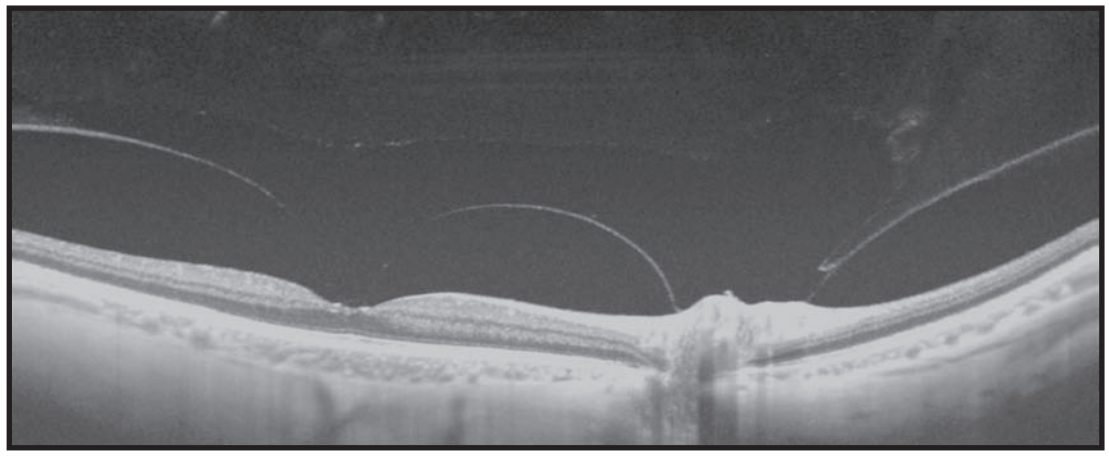
s0040 Natural History

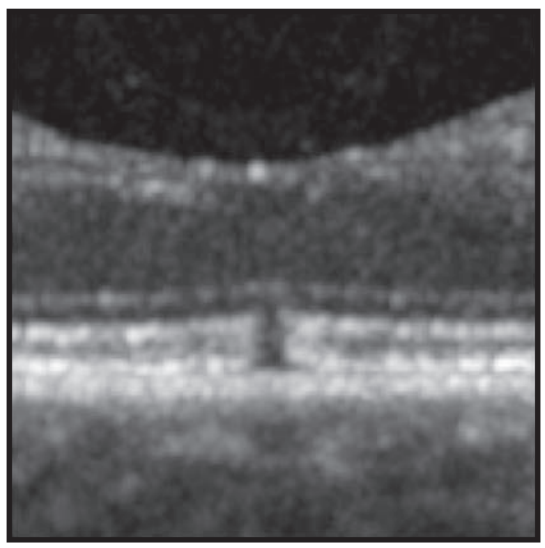


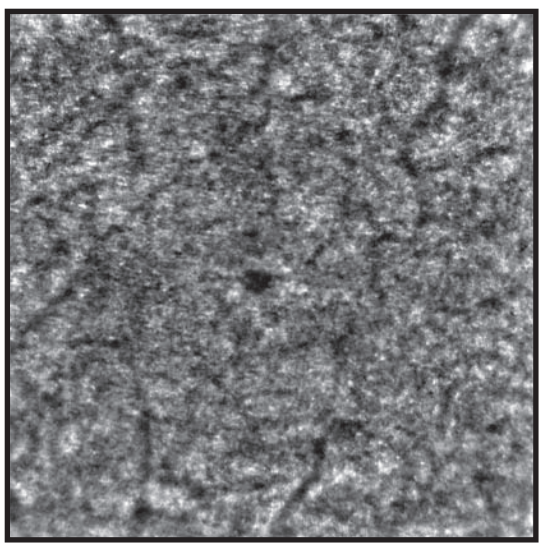
f0130 VMT may show progression to a lamellar macular hole *(left image)* or a full-thickness macular hole (FTMH) *(right image)*.



f0135 Focal VMT may also result in outer retinal defects, also known as macular microholes, outer lamellar macular holes, or foveal photoreceptor defects *(arrow)*.







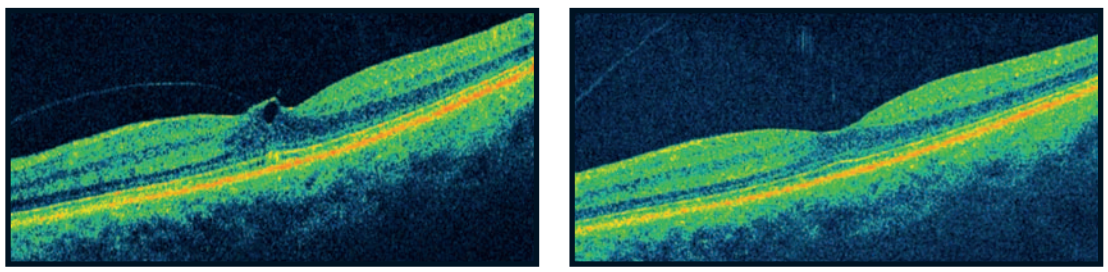
f0140 Patients may complain of a microscotoma due to traction-induced foveal lesions. High resolution swept-source OCT imaging *(top image)* clearly demonstrates the VMA in this case but does not identify the foveal abnormality as the density of the volume scan was inadequate. High density OCT imaging *(bottom left image)* at a subsequent visit reveals the foveal defect, which is also seen as a central loss of cones on adaptive optics imaging *(bottom right image)*.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

s0045 Spontaneous Release of Vitreomacular Traction

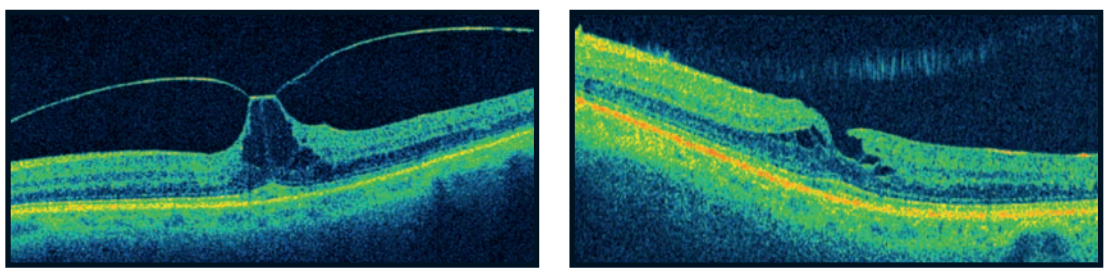
p0090 Spontaneous release of grade 1 or 2 VMT occurs in approximately 30% of eyes and release of grade 3 VMT occurs in almost 60-70% of cases. The mean time to spontaneous release is approximately 16 months.

f0145



Spontaneous release of VMT may occur with resolution of foveal abnormalities. This patient was found to have spontaneous release of VMT after 4 months. Visual acuity was unchanged and remained at 20/25.

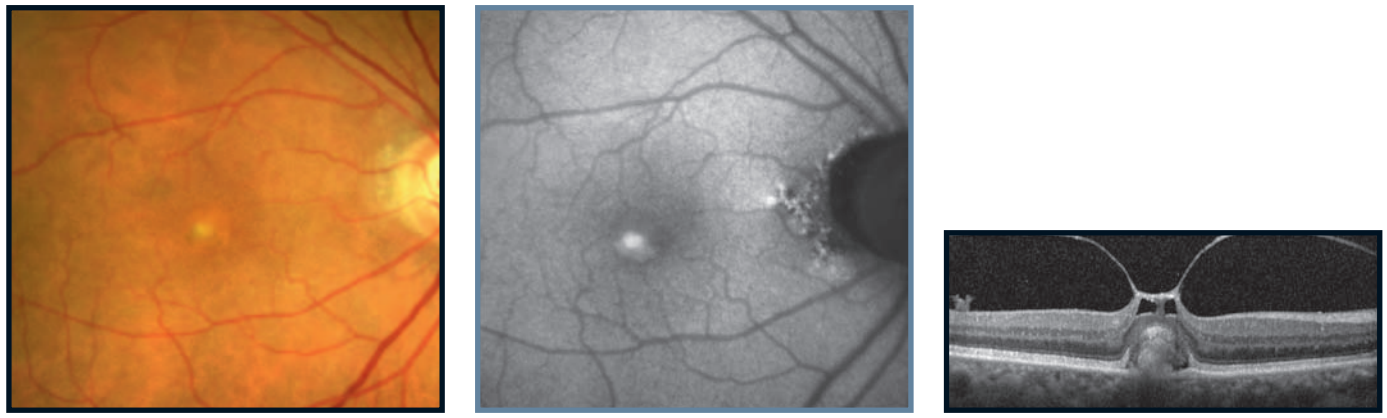
f0150



This patient had spontaneous release of VMT 1 year after initial presentation, with resulting intraretinal schisis at the fovea. Visual acuity was unchanged at 20/50.

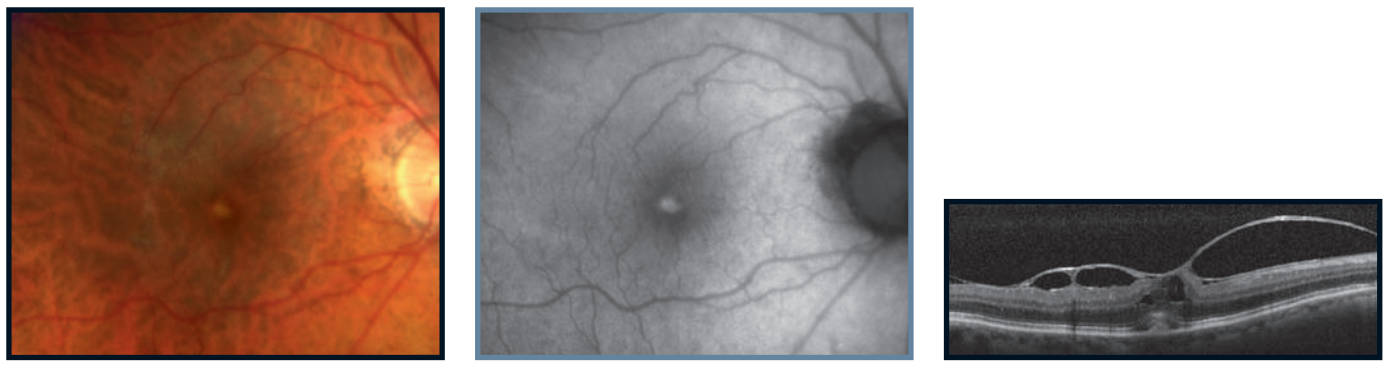
s0050 Vitreomacular Traction and Acquired Vitelliform Lesion

f0155



Focal VMT may be associated with an acquired vitelliform lesion. The vitelliform lesion appears yellow on color fundus photographs and is hyperautofluorescent on fundus autofluorescence imaging. The vitelliform lesion appears as hyper-reflective material in the subretinal space with OCT imaging.

f0160

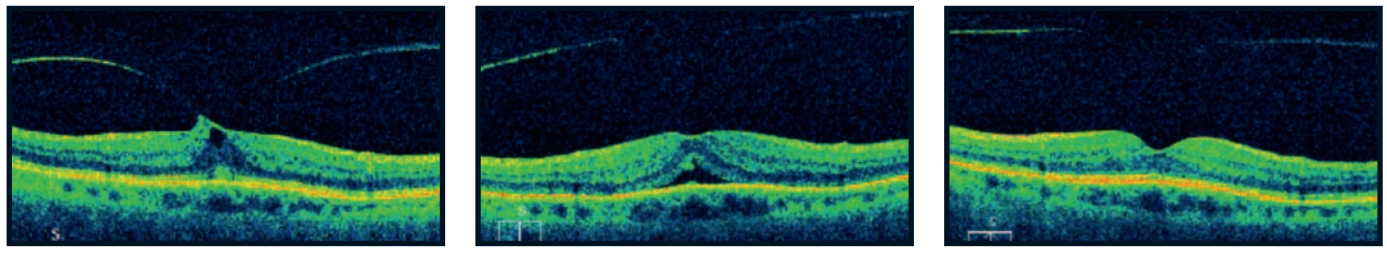


A vitelliform lesion was noted in this patient with broad VMT. *Images courtesy of Dr. Richard Spaide*

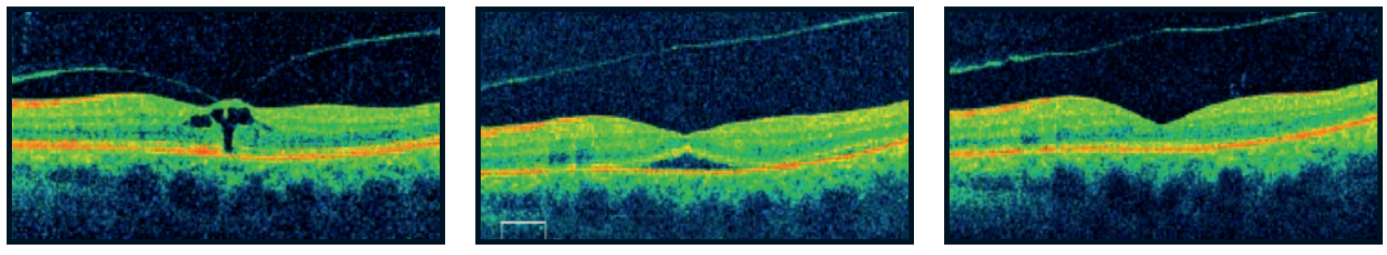
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

s0055 Vitreolytic Treatment

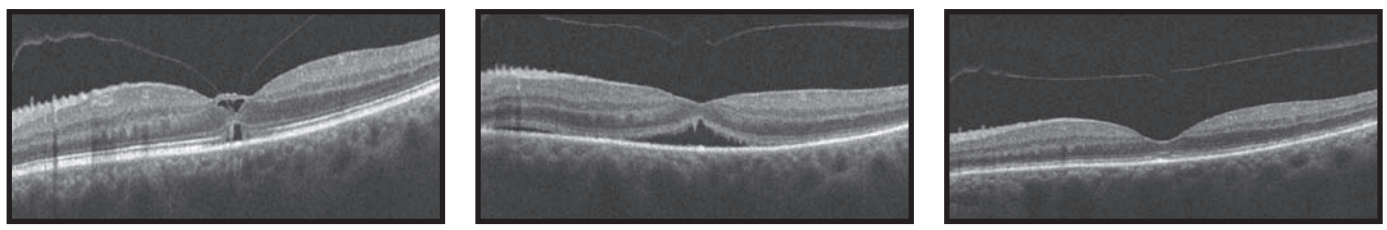
p0100 Recently, ocriplasmin has been employed as an intravitreal vitreolytic agent for focal VMA and focal VMT. Outcomes following treatment with this agent are variable; however, it may be considered as first-line therapy for some symptomatic patients.



f0165 This patient with focal VMT and visual acuity of 20/30 *(left image)* received an intravitreal injection of ocriplasmin, with successful release of the vitreomacular attachment on the first day *(middle image)*. The visual acuity dropped to 20/70 at this time due to subretinal fluid at the fovea. The subretinal fluid resolved by 1 month and the visual acuity improved to 20/20. *Images courtesy of Dr. Rishi Singh*



f0170 This patient is another example of successful release of focal VMT after intravitreal ocriplasmin injection. The initial visual acuity was 20/50. The release was noted 1 week after the injection *(middle image)*. Again, subretinal fluid at the fovea was noted at this time and visual acuity remained unchanged at 20/40. Subretinal fluid resolved by 3 weeks and visual acuity improved to 20/30 *(right image)*. *Images courtesy of Dr. Rishi Singh*



f0175 With high resolution OCT, it is possible to appreciate ellipsoid zone disruptions that are a known complication following ocriplasmin injection *(middle image)*. If ellipsoid zone disruption occurs, it usually recovers spontaneously within 1 to 3 months *(right image)*. *Images courtesy of Dr. Rishi Singh*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

s0060 Full-Thickness Macular Hole

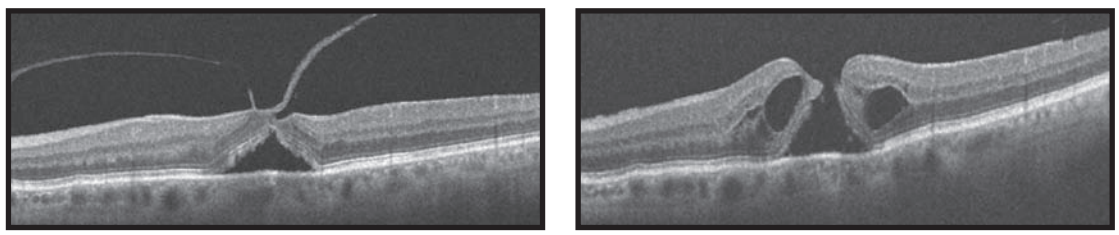
p0105 A full-thickness macular hole (FTMH) is a retinal defect that commences at the level of the internal limiting membrane and extends up to, but not including, the RPE. These defects may arise from a number of retinal insults including traction on the inner retina and/or loss of central neurosensory retinal tissue. Primary FTMHs are due to VMT on the fovea from an anomalous posterior vitreous detachment. Secondary FTMHs are due to a range of causes including trauma, lightning strike, myopia, macular telangiectasia type 2, and age-related macular degeneration. VMT may or may not be a pathogenic factor in the formation of secondary macular holes.

s0065 Classification

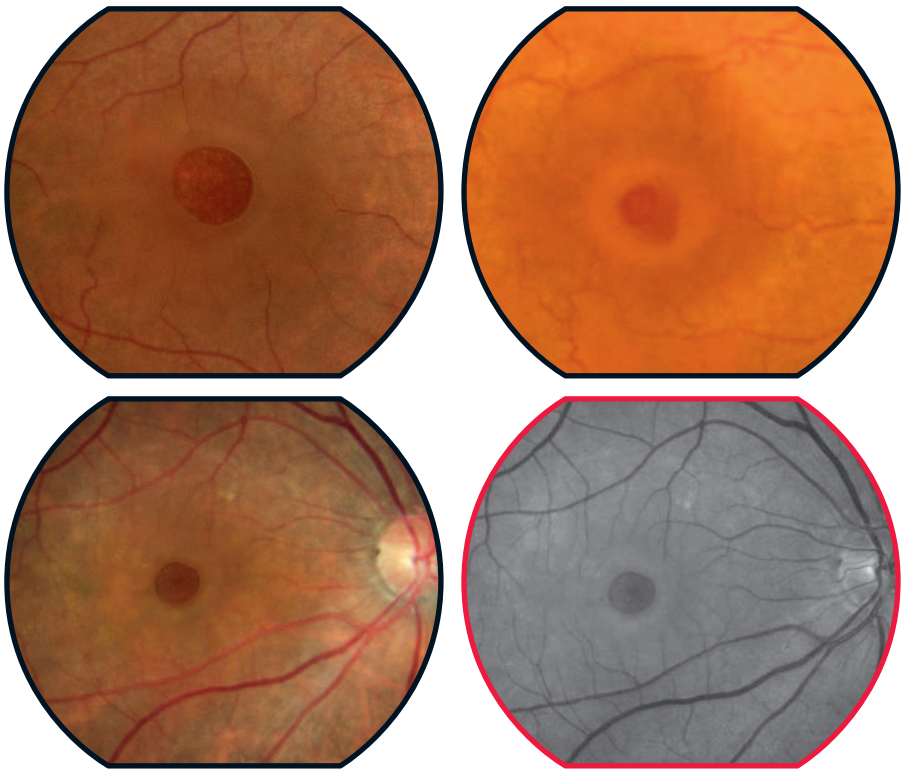
The Gass classification of FTMH comprised 4 stages and was based on clinical examination findings. In contrast, the International Vitreomacular Traction Study Classification of FTMH is based on OCT and describes the size of the hole, and the presence or absence of VMT. Correlation between the two schemes is as follows:

p0110

Gass Classification	International Vitreomacular Traction Study Classification
Stage 1: Impending hole	VMT
Stage 2: FTMH ≤400 μm, no posterior vitreous detachment (PVD)	Small (≤250 μm) or medium (>250-400 μm) FTMH with VMT
Stage 3: FTMH >400 μm, no PVD	Large (>400 μm) FTMH with VMT
Stage 4: FTMH >400 μm with PVD	Any size FTMH without VMT

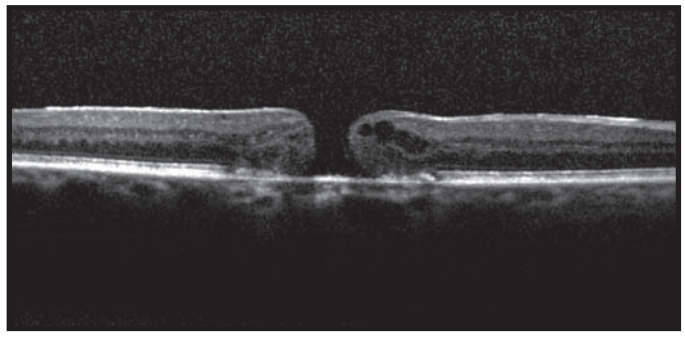


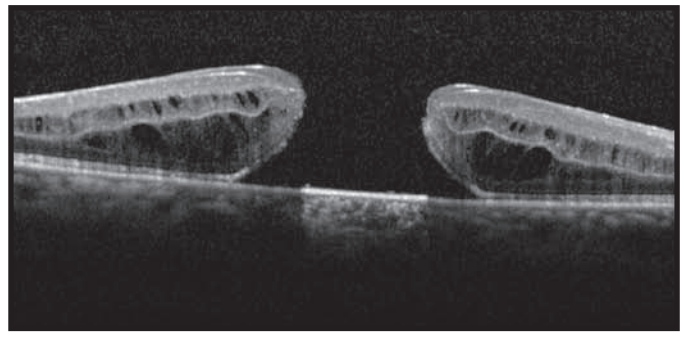
f0180 This patient with VMT developed a FTMH 2 years later. The left image illustrates the OCT features of VMT and the right image illustrates the OCT features of FTMH.



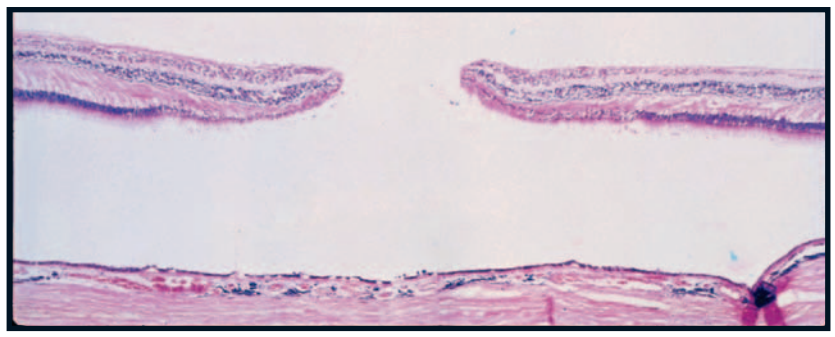
f0185 The characteristic clinical findings of FTMHs are illustrated in these cases. A FTMH is typically delineated by a sharp margin and frequently demonstrates a surrounding cuff of cystic change and subretinal fluid.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

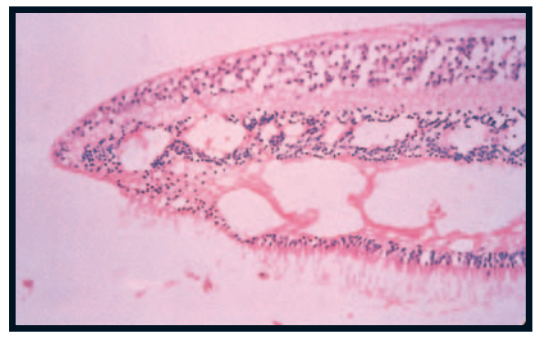




© 418

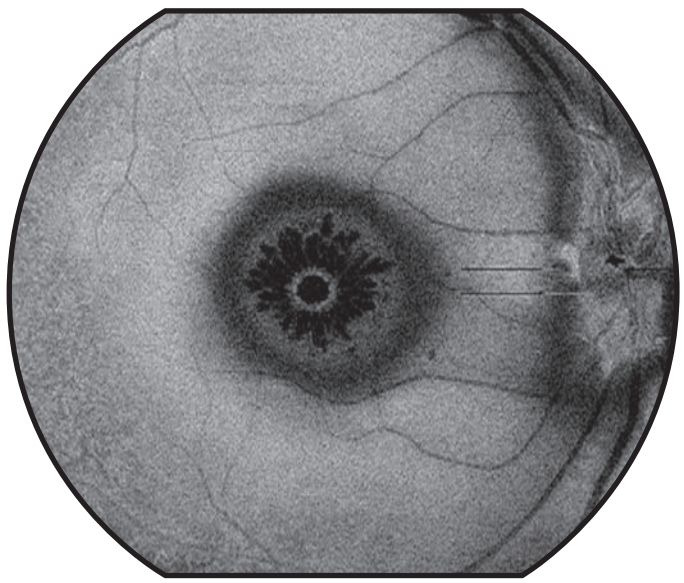


© 419



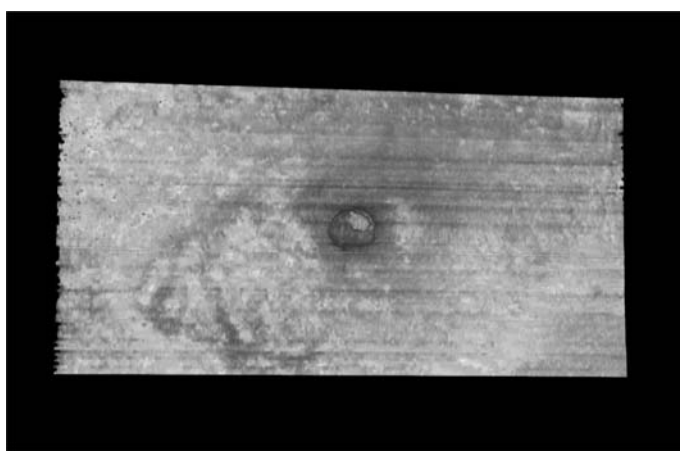
f0190

OCT imaging allows detailed ultrastructural evaluation of macular holes. In the above cases, full-thickness retinal defects are seen as are intraretinal cystic changes at the margins of the hole. Pathology of the margin of the hole shows cystic degeneration of the inner and outer retina and correlates closely to the appearance of macular holes as seen on OCT.



f0195

The petalloid morphology of cystic changes surrounding the margins of macular holes are best appreciated with *en face* imaging techniques, as illustrated in this case, which was evaluated with swept source OCT. *Image courtesy of Dr. Michael Engelbert*

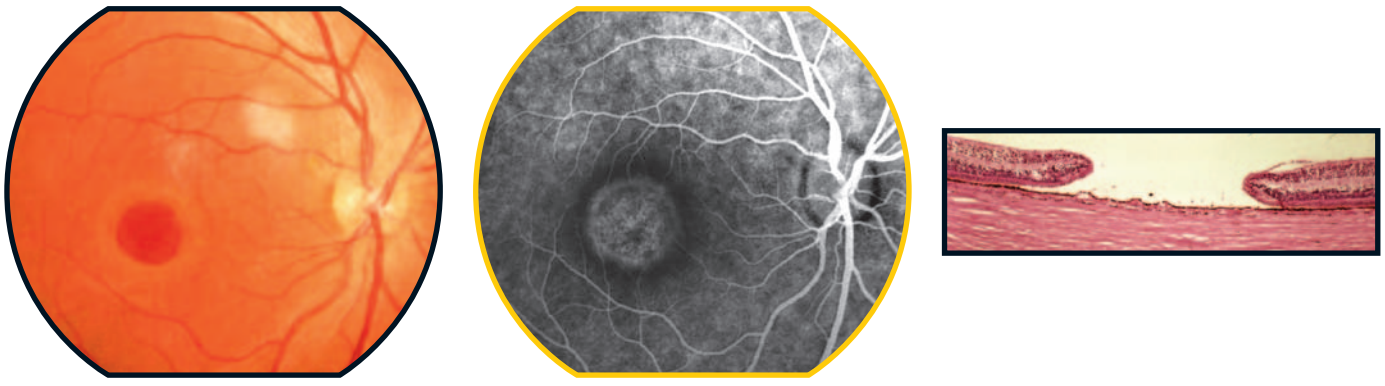


f0200

This volume rendered OCT image shows the FTMH in three dimensions. *Image courtesy of Dr. Richard Spaide*

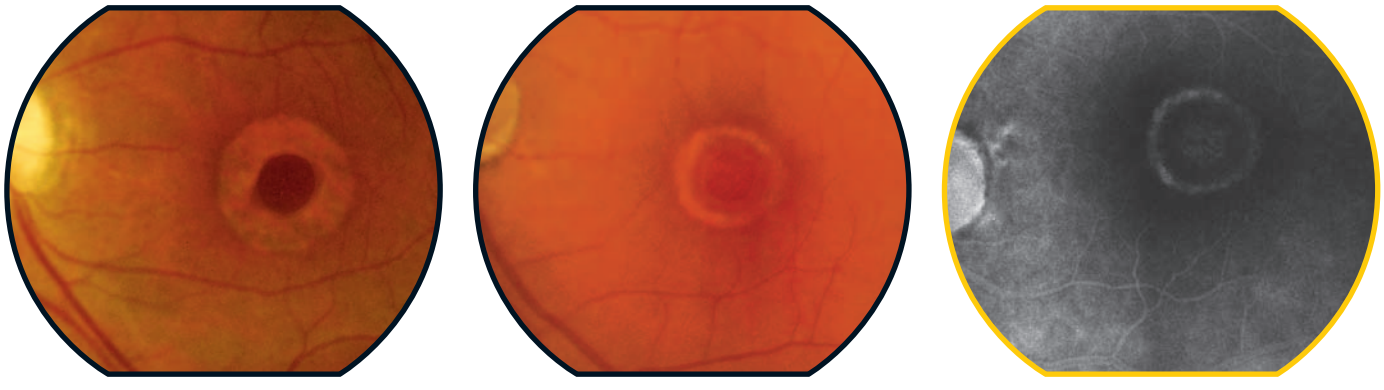
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

f0205



Fluorescein angiography of FTMHs demonstrates hyperfluorescence due to loss of overlying retinal tissue. Attenuation of the RPE at the site of the hole, as seen on the histological specimen *(right image)*, is another reason for this hyperfluorescence.

f0210

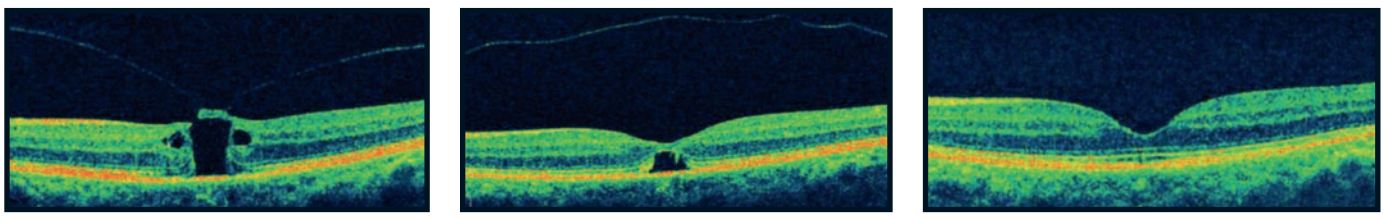


These patients with long-standing macular holes have a demarcation ring that is atrophic in nature. This appears as a hyperfluorescent window defect on fluorescein angiography.

s0070 Spontaneous Closure

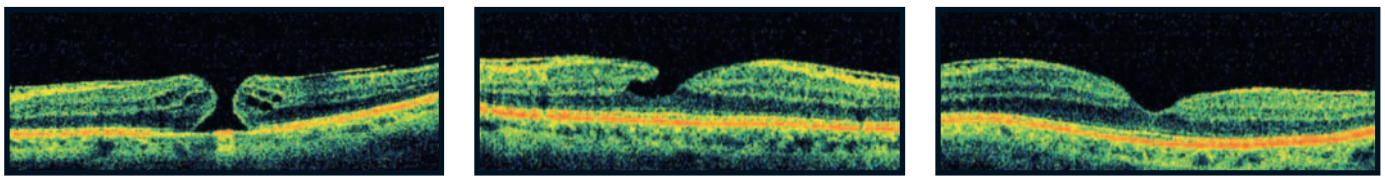
p0115 The rate of spontaneous closure of primary FTMHs has been reported to range from 3% to 6%. The exact mechanism of spontaneous macular hole closure is still unclear, but four different hypotheses have been proposed: (1) complete vitreous detachment over the fovea releasing the tractional forces, (2) formation of an epiretinal membrane resulting in hole shrinkage, (3) glial cell proliferation at the base of the hole, and (4) growth of retinal tissue bridging the hole.

f0215



This patient was noted to have VMT with adhesions of the posterior hyaloid to the roof of the macular hole *(left image)*. One week later, there was spontaneous and complete posterior vitreous separation, with bridging of the retinal tissue at the level of the inner retina. Often, there are outer retinal layer defects, which may either persist or recover with time *(middle image)*. After 1 year, foveal architecture appears normal *(right image)*.

f0220

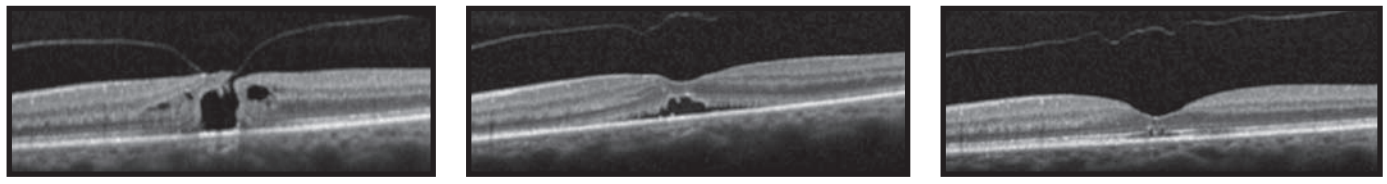


This patient had a FTMH of approximately 200 μm at the narrowest diameter with an epiretinal membrane *(left image)*. Five months later, there was spontaneous apposition of the outer layers of the retina with transformation of the hole into a lamellar configuration *(middle image)*. At 6 months, the foveal architecture appears almost normal *(right image)*. *Images courtesy of Dr. Andrea Scopulo*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

Vitreolytic Treatment

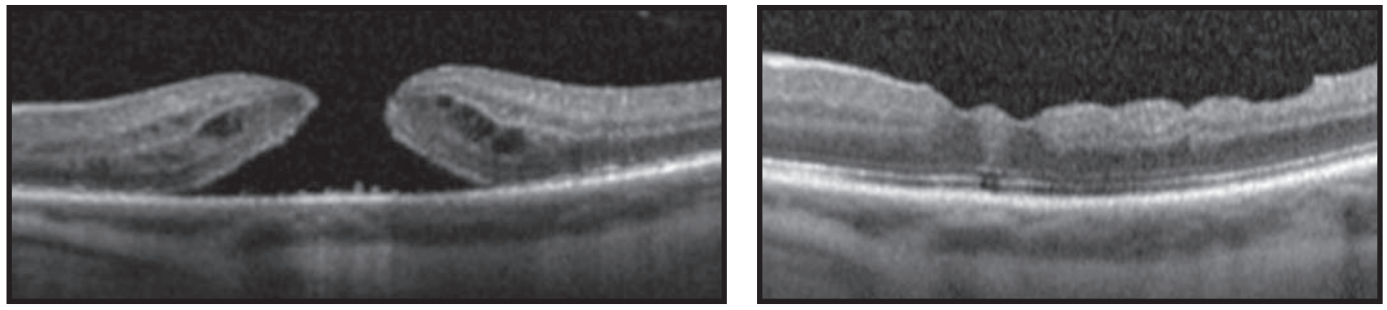
p0120 Intravitreal ocriplasmin therapy has been shown to have a success rate between 10-40% for small stage 2 FTMHs (≤250 μm). Ocriplasmin therapy is used by some surgeons as first-line therapy for carefully selected cases of FTMH. Eyes that fail vitreolytic therapy are managed surgically.



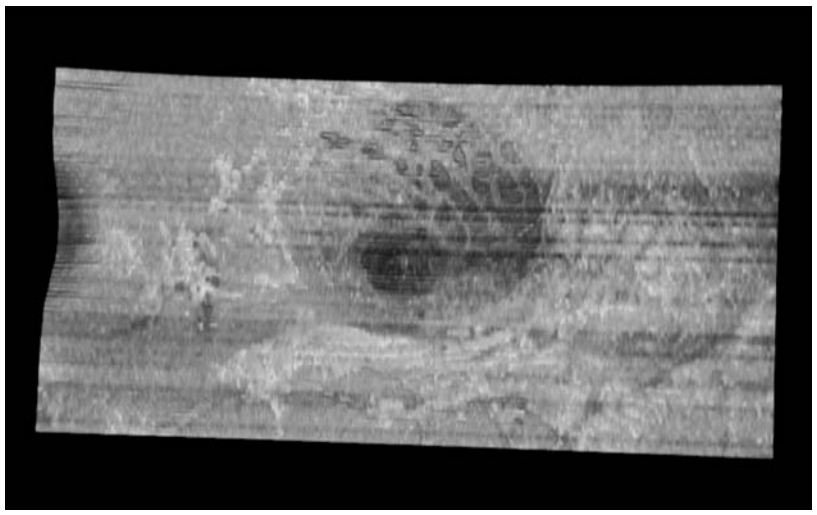
f0225 This patient with a stage 2 FTMH was treated with intravitreal ocriplasmin *(left image)*. One week later, there was separation of the posterior hyaloid and closure of the inner retina *(middle image)*. Six months later, there were persistent outer retinal defects and ellipsoid zone disruptions *(right image)*. *Images courtesy of Dr. John Miller*

Surgical Treatment

p0125 Patients with FTMHs may be treated with pars plana vitrectomy, internal limiting membrane peeling, and gas tamponade. Post-vitrectomy, inner retinal dimpling is seen on the inner retinal surface in approximately a third of cases and typically appears 3 months after surgery. The exact mechanism is unknown, although it has been proposed that the dimples may represent defects in Müller cell regrowth.



f0230 This patient with FTMH underwent pars plana vitrectomy and membrane peeling. Four months after surgery, there is inner retinal dimpling and some disruption at the level of the ellipsoid zone.

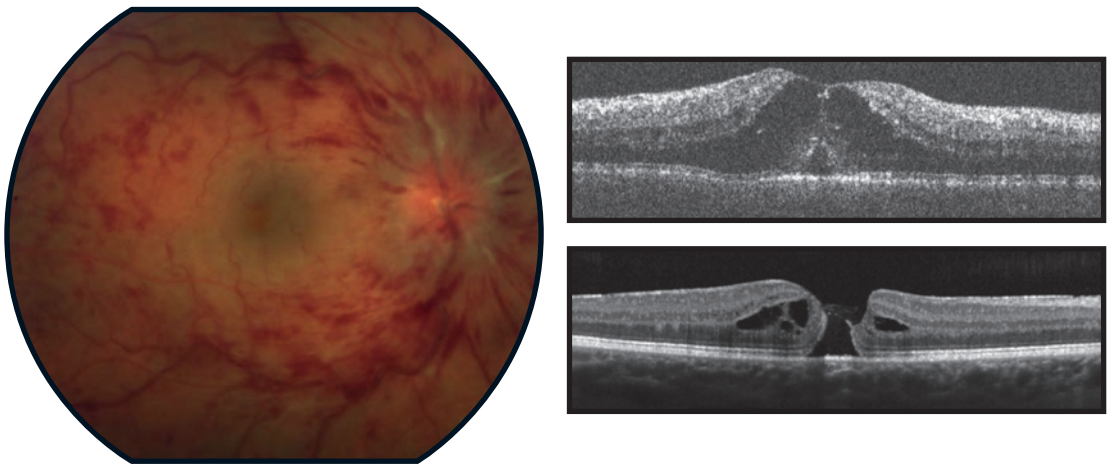


f0235 Three-dimensional volume rendered OCT imaging provides precise spatial visualization of inner retinal dimpling following macular hole surgery. *Image courtesy of Dr. Richard Spaide*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

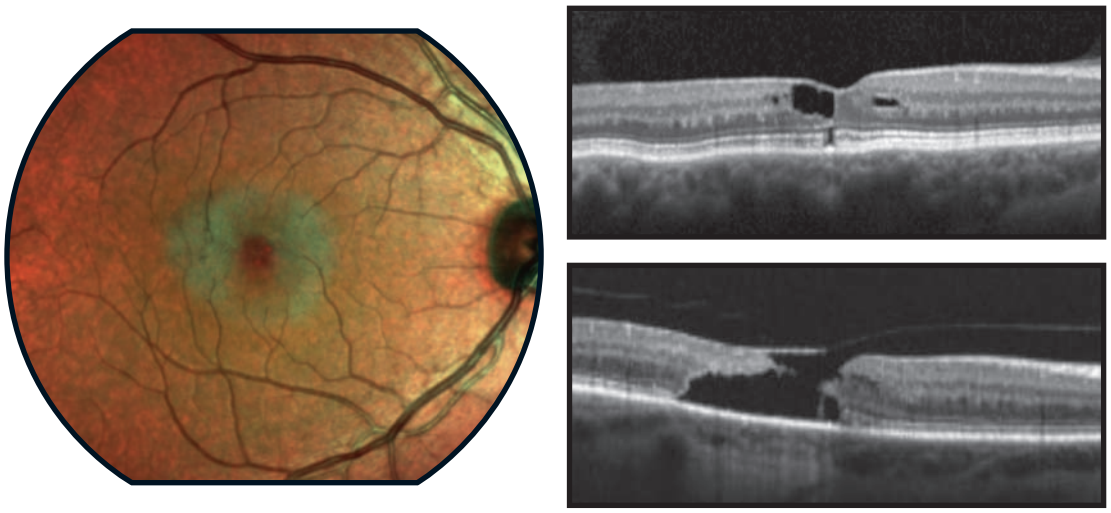
s0085 Secondary Macular Holes

p0130 The pathogenesis of secondary macular holes is different from primary macular holes. Surgical hole closure rates are lower in eyes with secondary macular holes due to the varied pathophysiology.



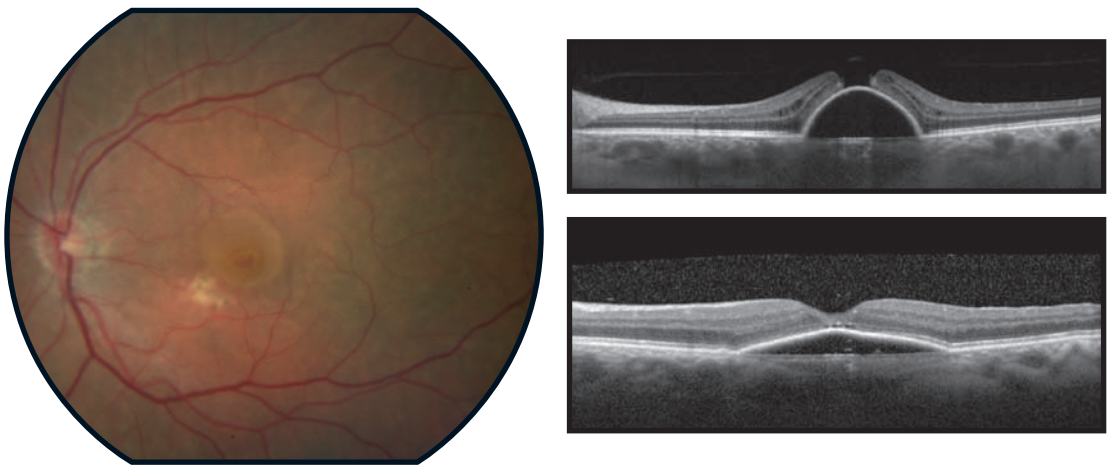
f0240

This patient had a central retinal vein occlusion *(left image)* and cystoid macular edema *(top right image)* that was treated with intravitreal steroids and anti-vascular endothelial growth factor therapy. A FTMH appeared after resolution of the central macular edema *(bottom right image)*. *Images courtesy of Dr. Jay Klancnik*



f0245

This multicolor image highlights the perifoveal lesion that is characteristic of macular telangiectasia type 2 *(left image)*. OCT imaging reveals characteristic atrophic and cavitary retinal defects at the fovea *(top right image)*. With time, a FTMH developed as the area of cavitation and atrophy enlarged *(bottom right image)*.

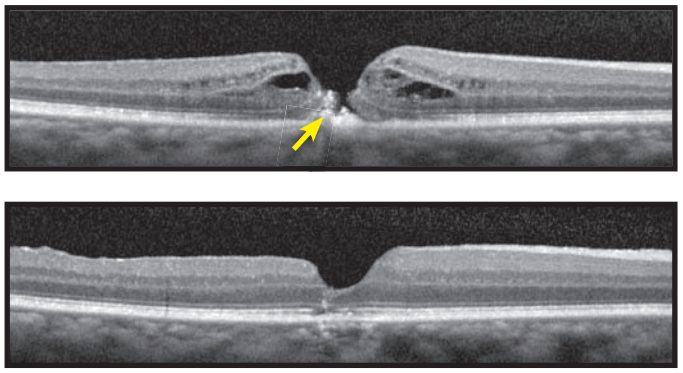


f0250

This patient with a macular hole overlying a serous pigment epithelial detachment was treated with multiple intravitreal anti-vascular endothelial growth factor injections and photodynamic therapy over a period of 1 year to induce flattening of the pigment epithelial detachment. A pars plana vitrectomy with peeling of the internal limiting membrane was performed subsequently with successful closure of the macular hole.

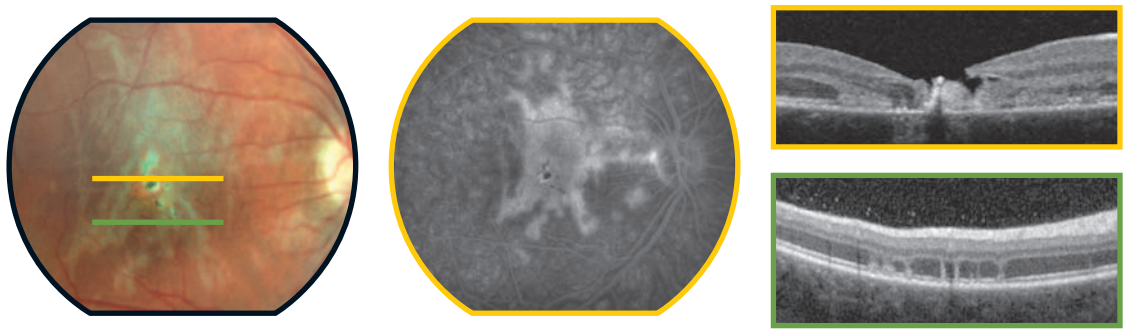
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

f0255



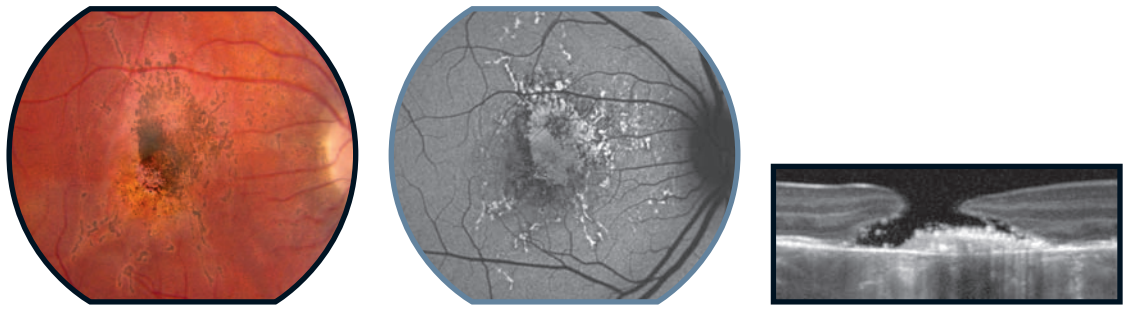
Macular holes may uncommonly develop during the natural course of acquired vitelliform lesions. In this case, a macular hole developed during the process of spontaneous vitelliform reabsorption *(arrow)*. Anatomical closure was achieved following vitrectomy and membrane peeling *(bottom image)*. *Images courtesy of Dr. Michael Engelbert*

f0260



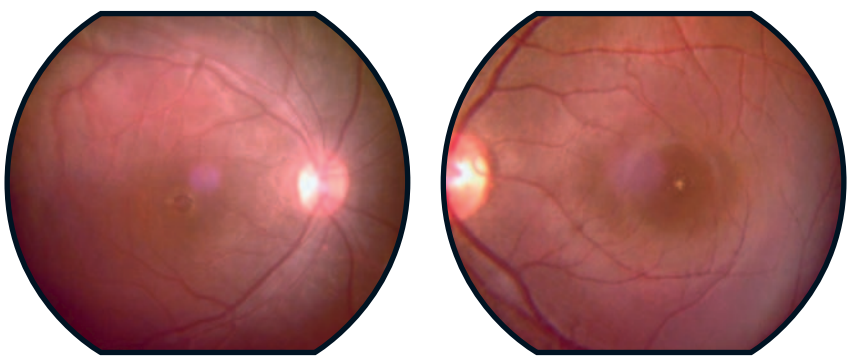
This patient developed a FTMH following self-inflicted injury with a handheld laser pointer. The curvilinear streaks of retinal pigment epithelial damage appear hyperfluorescent on fluorescein angiography. Hyper-reflective vertical steaks in the outer retina, as seen on OCT, characterize the acute stages of laser-induced maculopathy *(bottom right image)*.

f0265



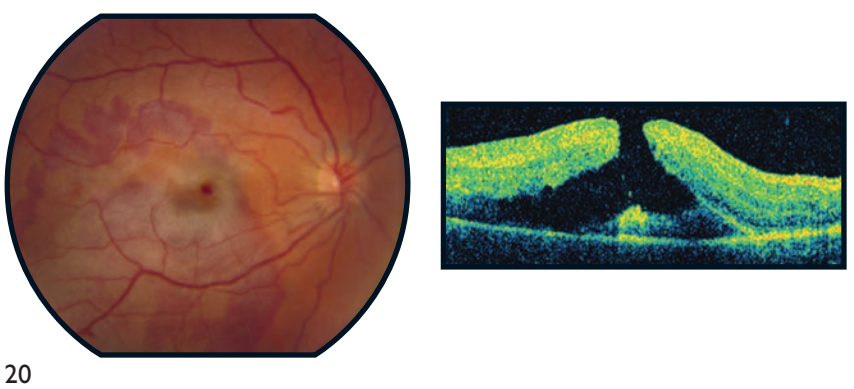
Months later, the patient in the above case developed a pigmented, hyperautofluorescent scar at the site of injury. OCT imaging shows a FTMH at the site of RPE proliferation and scarring.

f0270



Bilateral macular holes may be induced by lightning strike injury as seen in this case. *Images courtesy of Dr. Vikram Jain*

f0275



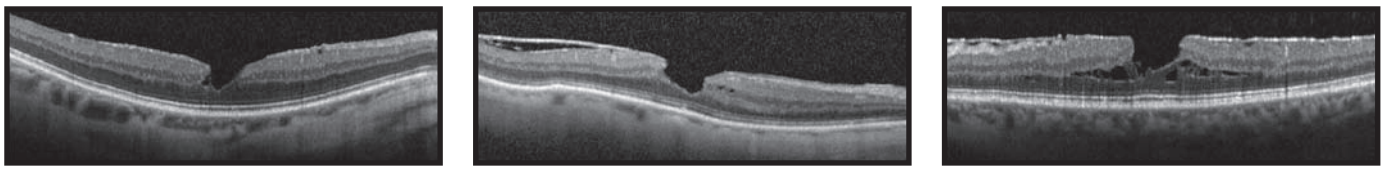
Blunt trauma may also induce an acute FTMH. Blunt injury in this case resulted in a large, subretinal hemorrhage and a FTMH that was clearly seen on OCT. *Images courtesy of Dr. Carmen Puliafito*

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

s0090 Lamellar Macular Hole

p0135 The diagnosis of lamellar macular hole requires the following three criteria to be satisfied: (1) an irregular foveal contour or defect of the inner retina, (2) thinning at the base of the fovea, and (3) absence of a full-thickness defect. Lamellar macular holes may develop following abrupt termination of pathophysiological processes that would otherwise have resulted in a FTMH. Lamellar macular holes may also be due to contraction of an existing perifoveal epiretinal membrane–internal limiting membrane complex. Studies have shown that 80% to 100% of lamellar macular holes are associated with epiretinal membranes. Although lamellar macular holes may progress to FTMHs, approximately 80% were found to be stable, both functionally and morphologically, over time. As such, lamellar macular holes are usually observed and surgical management is considered only if there is visual decline.

f0280

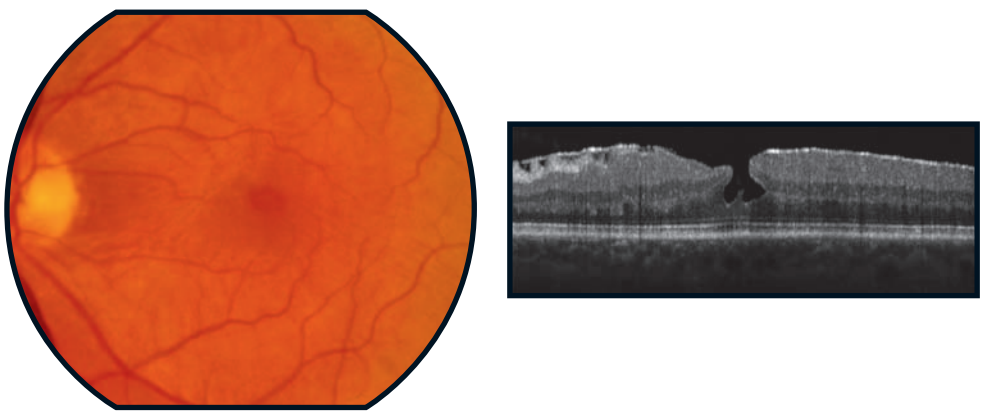


Spectral-domain OCT shows the variable appearance of lamellar macular holes. In these patients, there is an associated epiretinal membrane, an irregularity in the foveal contour, defects in the inner retina, and thinning of the retina at the base of the fovea without a full-thickness defect. There may also be intraretinal schisis due to traction *(right image)*.

s0095 Macular Pseudohole

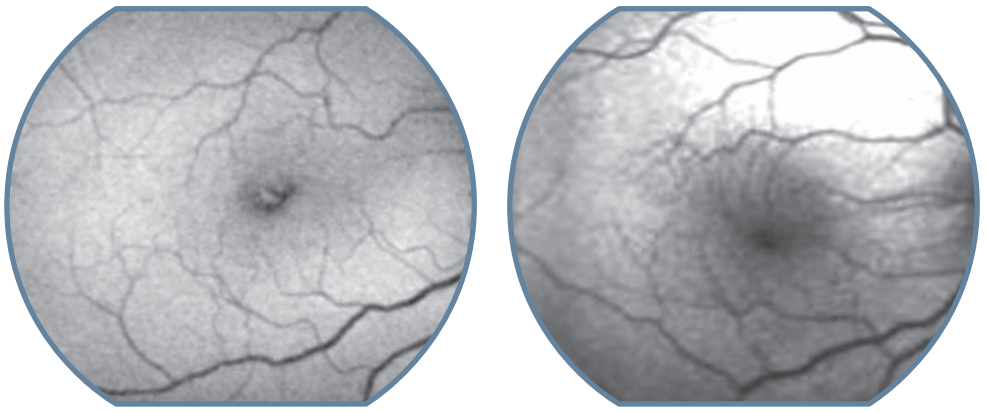
p0140 Macular pseudohole is not a true FTMH and is due to contraction of an epiretinal membrane. With spectral-domain OCT, a macular pseudohole has a very similar appearance to a lamellar macular hole, especially when the foveal tissue loss is subtle. Some clinicians believe that lamellar macular hole and macular pseudohole are two distinct entities, best distinguished with fundus autofluorescence by demonstrating foveal tissue loss in true lamellar macular holes. However, other clinicians believe that the two are very similar in that they both possess a perifoveal epiretinal membrane–internal limiting membrane complex, with lamellar macular holes demonstrating centrifugal contraction and macular pseudoholes demonstrating a centripetal contraction. Management of macular pseudoholes is observation unless there is progressive visual decline that warrants surgery.

f0285



The color fundus photograph shows a macular pseudohole due to an epiretinal membrane *(left image)*. OCT demonstrates an epiretinal membrane with associated tenting of inner retinal tissue due to centripetal contraction *(right image)*.

f0290

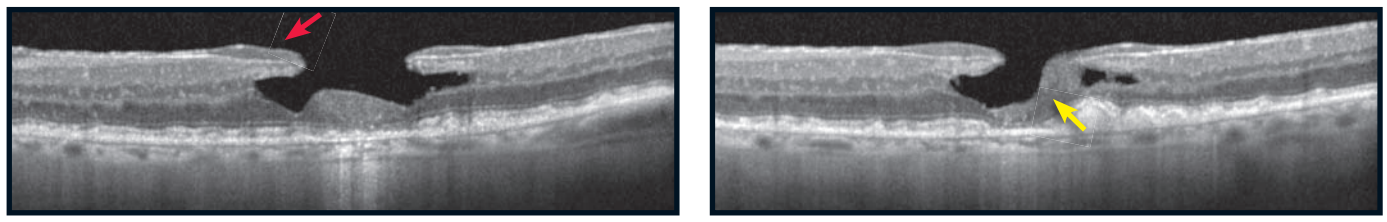


Fundus autofluorescence of a lamellar hole may reveal hyperautofluorescence due to loss of foveal tissue *(left image)* compared to the normal appearance of a macular pseudohole *(right image)*.

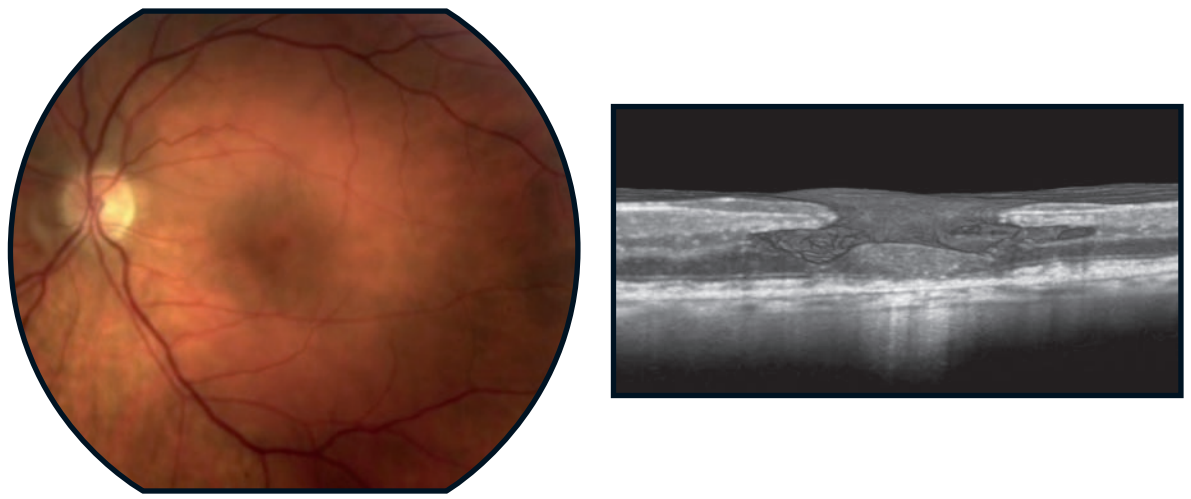
ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

Lamellar Hole-Associated Epiretinal Proliferation

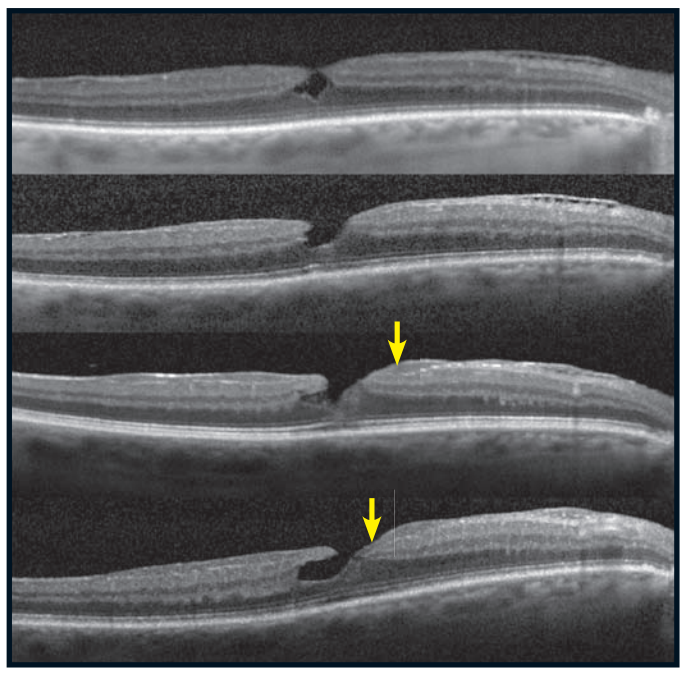
Lamellar hole-associated epiretinal proliferation (LHEP) is an OCT finding that is characterized by homogenous material of varying thickness on the retinal surface that is of medium reflectivity. LHEP has been reported in eyes with inner retinal defects and occurs in approximately one-third of lamellar macular holes. With high density and high resolution OCT scans, LHEP is seen to be contiguous with the middle retinal layers and is postulated to be a proliferation of glial cell tissue from the inner retinal defect onto the epiretinal surface. LHEP is differentiated from an epiretinal membrane by its OCT appearance and its non-contractile nature.



LHEP typically appears as homogenous, medium-reflective material of varying thickness on SD-OCT *(red arrow)*. The LHEP is present on the epiretinal surface adjacent to the lamellar macular hole and is contiguous with the middle retinal layers *(yellow arrow)*.



LHEP is difficult to distinguish on color photography *(left image)*. Volume rendered OCT imaging *(right image)* demonstrates the relationships between LHEP, the middle retinal layers of the retina, and base of the lamellar macular hole. There is an irregular epiretinal surface contour due to the varying thickness of LHEP but no traction or wrinkling of the retina. *Image courtesy of Dr. Richard Spaide*



This serial eye-tracked sequence of OCT scans of the same patient taken over a course of 5 years shows a lamellar macular hole with a thin hyper-reflective epiretinal membrane that develops LHEP over time. There is contiguity between LHEP and the middle retinal layers at the inner retinal defect *(arrow)*.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009

Suggested Reading

Epiretinal Membrane

Appiah, A.P., 1989. Secondary causes of premacular fibrosis. Ophthalmology 96, 389–392.

Koizumi, H., Spaide, R.F., Fisher, Y.L., et al., 2008. Three-dimensional evaluation of vitreomacular traction and epiretinal membrane using spectral-domain optical coherence tomography. Am. J. Ophthalmol. 145 (3), 509–517.

Pang, C.E., Spaide, R.F., Freund, K.B., 2014. Epiretinal proliferation seen in association with lamellar macular holes: a distinct clinical entity. Retina 34 (8), 1513–1523.

Pang, C.E., Spaide, R.F., Freund, K.B., 2015. Comparing functional and morphologic characteristics of lamellar macular holes with and without lamellar hole-associated epiretinal proliferation. Retina 35 (4), 720–726.

Pesin, S.R., Olk, R.G., Grand, M.G., et al., 1991. Vitrectomy for premacular fibroplasia. Prognostic factors, long-term follow-up, and time course of visual improvement. Ophthalmology 98, 1109–1114.

Scheerlinck, L.M., van der Valk, R., van Leeuwen, R., 2015. Predictive factors for postoperative visual acuity in idiopathic epiretinal membrane: a systematic review. Acta Ophthalmol. 93 (3), 203–212.

Smiddy, W.E., Maguire, A.M., Green, W.R., et al., 1989. Idiopathic epiretinal membranes: ultrastructural characteristics and clinicopathologic correlation. Ophthalmology 96, 811–821.

Tari, S.R., Vidne-Hay, O., Greenstein, V.C., et al., 2007. Functional and structural measurements for the assessment of internal limiting membrane peeling in idiopathic macular pucker. Retina 27, 567–572.

Vitreomacular Traction and Macular Hole

Balaratnasingam, C., Dansingani, K., Dhrami-Gavazi, E., et al., 2015. Documentation of spontaneous macular hole closure in macular telangiectasia type 2 using multimodal imaging. Ophthalmic Surg. Lasers Imaging Retina 46 (8), 883–886.

Bhavsar, K.V., Wilson, D., Margolis, R., et al., 2015. Multimodal imaging in handheld laser-induced maculopathy. Am. J. Ophthalmol. 159 (2), 227–231.

Campo, R.V., Lewis, R.S., 1984. Lightning-induced macular hole. Am. J. Ophthalmol. 97, 792–794.

Chang, L.K., Fine, H.F., Spaide, R.F., et al., 2008. Ultrastructural correlation of spectral-domain optical coherence tomographic findings in vitreomacular traction syndrome. Am. J. Ophthalmol. 146 (1), 121–127.

Chew, E.Y., Sperduto, R.D., Hiller, R., et al., 1999. Clinical course of macular holes: the eye disease case-control study. Arch. Ophthalmol. 117, 248–249.

Duker, J.S., Kaiser, P.K., Binder, S., et al., 2013. The International Vitreomacular Traction Study Group classification of vitreomacular adhesion, traction, and macular hole. Ophthalmology 120 (12), 2611–2619.

Fisher, Y.L., Slakter, J.S., Yannuzzi, L.A., et al., 1994. A prospective natural history study and kinetic ultrasound evaluation of idiopathic macular holes. Ophthalmology 101, 5–11.

Goldberg, N., Freund, K.B., 2012. Progression of an acquired vitelliform lesion to a full-thickness macular hole documented by eye-tracked spectral-domain optical coherence tomography. Arch. Ophthalmol. 130 (9), 1221–1223.

Seider, M.I., Lujan, B.J., Gregori, G., et al., 2009. Ultra-high resolution spectral domain optical coherence tomography of traumatic maculopathy. Ophthalmic Surg. Lasers Imaging 40 (5), 516–521.

Stalmans, P., Benz, M.S., Gandorfer, A., et al., MIVI-TRUST Study Group. 2012. Enzymatic vitreolysis with ocriplasmin for vitreomacular traction and macular holes. N. Engl. J. Med. 367 (7), 606–615.

Steel, D.H., Lotery, A.J., 2013. Idiopathic vitreomacular traction and macular hole: a comprehensive review of pathophysiology, diagnosis, and treatment. Eye (Lond) 27 (Suppl. 1), S1–S21.

Tzu, J.H., John, V.J., Flynn, H.W. Jr., et al., 2015. Clinical course of vitreomacular traction managed initially by observation. Ophthalmic Surg. Lasers Imaging Retina 46 (5), 571–576.

ISBN: 978-0-323-28792-0; PII: B978-0-323-28792-0.00009-0; Author: Freund & Sarraf & Mieler & Yannuzzi; 00009