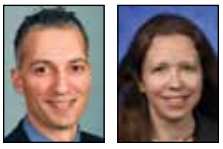
Modified Vitrectomy for Lamellar Macular Hole

With this approach, epiretinal tissue containing macular pigment and glial cells is preserved.

BY FUMIO SHIRAGA, MD, PhD; YUKI MORIZANE, MD, PhD; AND SHUHEI KIMURA, MD, PhD



In this issue of Retina Today, *Fumio Shiraga, MD, PhD; Yuki Morizane, MD, PhD; and Shuhei Kimura, MD, PhD, describe a new surgical technique for vitrectomy in patients with lamellar macular hole.*

We extend an invitation to readers to submit pearls for publication in Retina Today. *Please send submissions for consideration to Dean Eliott, MD (dean_eliott@meei.harvard.edu); or Ingrid U. Scott, MD, MPH (iscott@hmc.psu.edu). We look forward to hearing from you.*

— Dean Eliott, MD; and Ingrid U. Scott, MD, MPH

Lamellar macular hole (LMH) was first described by Gass[1] in 1975 as an abortive process of full-thickness macular hole formation that resulted from cystoid macular edema. In contrast, macular pseudoholes (MPH) were attributed to centripetal contraction of epiretinal membrane (ERM).[2] Michalewski et al[3] demonstrated that MPH may progress to LMH, and, because it is an advanced stage of the same non–full-thickness macular disorder, progression of ERM may be the cause of both MPH and LMH. Chen et al[4] hypothesized that both entities may be different manifestations of the same disease.

ERM has been shown to coexist in most patients with LMH. When ERM coexists with macular edema associated with branch retinal vein occlusion or diabetic retinopathy, or when it occurs after cataract surgery, LMH is secondary. In contrast, when no causative retinal diseases are present, LMH should be referred to as idiopathic.

Although the natural prognosis for idiopathic LMH is usually good, some patients exhibit a decline in visual acuity that may be amenable to surgical treatment. Because LMH is regularly accompanied by typical ERM, surgical treatment often includes ERM removal and internal limiting membrane (ILM) peeling with or without gas tamponade. In our surgical experience, LMH is usually accompanied by epiretinal tissue containing macular pigment and glial cells,[5] and the epiretinal tissue appears to originate from inside the LMH. Thus, we have developed a new surgical technique for vitrectomy in patients with LMH, in which epiretinal tissue containing macular pigment and glial cells is preserved but not removed.[6]

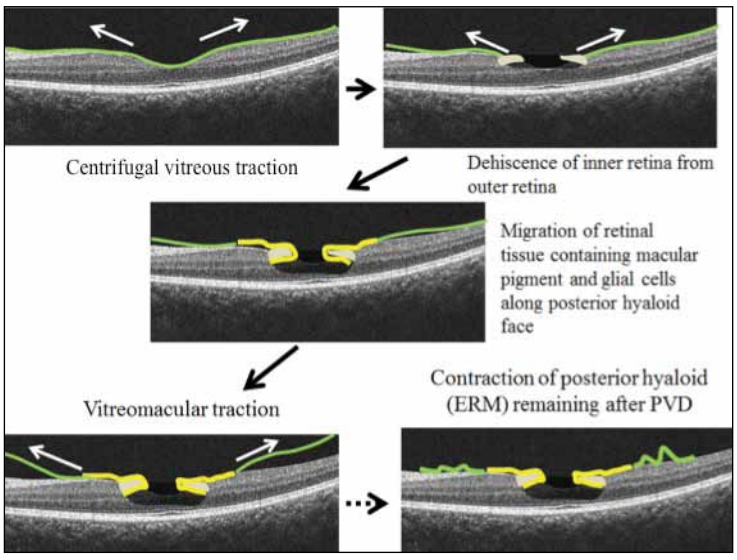


Figure 1. Mechanism of development of LMH, part 1.

MECHANISM OF LMH DEVELOPMENT

We speculate that there are 2 mechanisms of LMH development. First, after a dehiscence of the inner retina from the outer retinal layers caused by vitreous traction, some of the

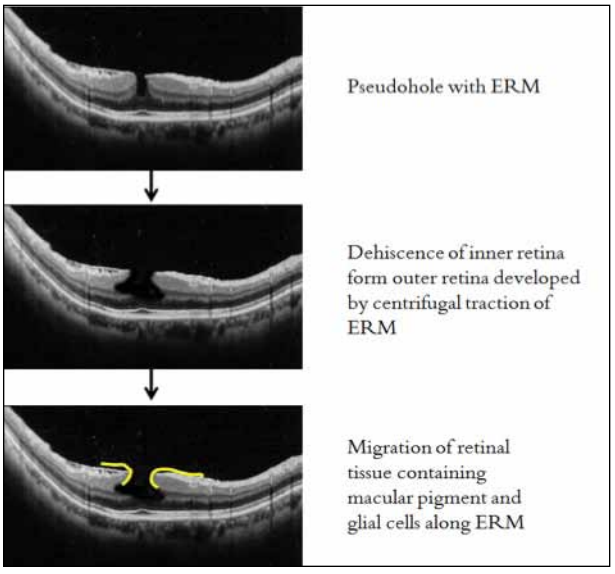


Figure 2. Mechanism of development of LMH, part 2.

dehisced retinal tissue with macular pigment and glial cells migrates along the posterior hyaloid face (Figure 1). When vitreomacular traction or contraction of the remnants of the posterior hyaloid membrane after posterior vitreous detachment (PVD) exists, patients may note decreased and/or distorted vision. In such cases, surgical treatment may be considered.

With the second mechanism of LMH development, MPH is caused by centripetal traction of ERM. However, in some patients with MPH, centrifugal traction of ERM exists. In such cases, a dehiscence of the inner retina from the outer retina may develop, resulting in visual acuity loss (Figure 2).

SURGICAL TECHNIQUE

In eyes with LMH, we perform 25-gauge, transconjunctival, sutureless microincisional vitrectomy surgery (MIVS). If PVD does not develop, triamcinolone-assisted PVD creation is performed with a vitreous cutter. In 6 of 22 consecutive eyes (27.3%) in our series, PVD was not present at the time of surgery. After PVD induction and removal of the posterior hyaloid, epiretinal tissues with macular pigment are centripetally peeled off from the retina using microforceps, and the epiretinal tissue is left attached to the edge of the LMH (Figure 3A-C).

In some cases, a peripheral lucent portion of peeled epiretinal tissue, which does not contain any macular pigment, is trimmed using the vitreous cutter. As a consequence, only epiretinal tissue that contains macular pigment remains and surrounds the LMH. The epiretinal tissue is gently massaged centripetally over the LMH using a backflush instrument with a brush needle

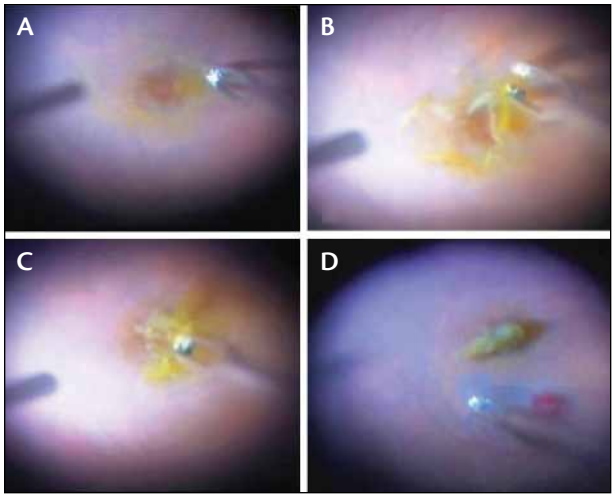


Figure 3. Surgical photographs. (A, B, C) Yellowish ERM is being peeled centripetally using microforceps. (D) After leaving the ERM attached to the edge of an LMH, brilliant blue G-assisted ILM peeling is performed around the LMH in a circumferential manner.

(Shiraga Backflush Instrument, DORC) so that the LMH is covered with the inverted epiretinal tissue pieces.

Next, brilliant blue G-assisted ILM peeling is performed around the LMH in a circumferential manner (Figure 3D). During the ILM peeling, the ERM tissue over the LMH is left untouched. At the end of surgery, an air-fluid exchange is performed. After surgery, patients are instructed to maintain a facedown position for 24 hours.

SURGICAL OUTCOMES

Functional Results

In our series of 22 eyes with a mean postoperative follow-up duration of 8.2 months (median, 8 months; range, 3–17 months), mean best corrected visual acuity (BCVA) improved significantly from 0.329 ±0.188 logMAR (geometric mean BCVA, 20/43) preoperatively to 0.047 ±0.167 logMAR (geometric mean BCVA, 20/22) at the final postoperative visit (paired *t*-test; $P < .001$). BCVA improved by 0.2 logMAR in 19 eyes (86%) and stabilized in 3 eyes (14%) at the final visit after the surgery. BCVA was 20/20 or better at the final postoperative visit in 11 of 22 eyes (50%).

Microstructural Results

Recovery of a smooth, normal-appearing foveal contour, assessed by spectral-domain optical coherence tomography (SD-OCT), occurred in 17 of 22 consecutive eyes (77%), while the foveal contour was irregular in 4 eyes (18%). In the other eye, a lamellar defect

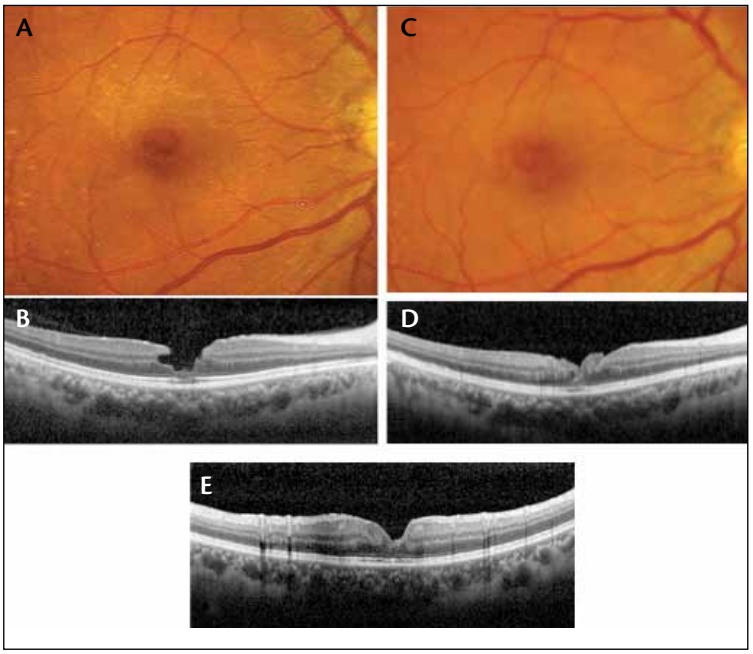


Figure 4. (A) A preoperative color photograph shows an LMH. BCVA is 20/25. (B) Preoperative SD-OCT shows a lamellar defect. (C) Seven days after surgery, a color photograph does not show any apparent LMH. (D) Seven days after surgery, SD-OCT shows filling of embedded epiretinal tissue containing macular pigment. (E) Four weeks after surgery, SD-ODT shows a regular foveal configuration. BCVA improved to 20/13.

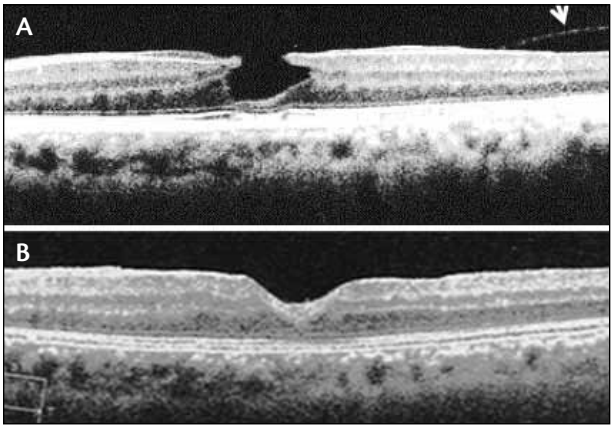


Figure 5. (A) SD-OCT shows thick ERMs (arrows) containing macular pigment before surgery. An arrow indicates the posterior hyaloid face. (B) The thick ERMs are not observed after surgery, and continuity of the IS/OS junction line is restored.

remained. One week after surgery, SD-OCT showed hyperreflective tissue infilling the dehiscence space that had been seen prior to the surgery between the inner and outer retina in all eyes (Figure 4). Subsequently, the foveal configuration appeared to be recovering to the normal state (Figure 5).

SD-OCT demonstrated discontinuity of the inner segment/outer segment (IS/OS) junction line before surgery in 8 of 20 eyes (35%). In 7 of these 8 eyes, the continuity of the IS/OS junction line was restored by the time of the final visit after surgery. The mean central retinal thickness recovered from 111.1 ±56.2 µm before surgery to 172.9 ±69.0 µm at the final visit after surgery (paired *t*-test; $P < .001$). No severe complications, such as retinal detachment or macular hole, occurred.

DISCUSSION

For a little while after surgery, SD-OCT shows the presence of epiretinal tissue inside the LMH. Subsequently, epiretinal tissue disappears, followed by the recovery of the foveal contour. Our SD-OCT evaluation of the postoperative foveal contour showed that 77% of patients had a regular contour, while 18% had an irregular contour and 5% had a lamellar defect. This foveal contour recovery may be due to the embedding of the epiretinal tissue containing glial cells.

In addition, in 7 of the 8 eyes in which SD-OCT showed photoreceptor defects prior to surgery, the IS/OS junction lines recovered. Inverted epiretinal tissues may play a key role in the recovery of foveal contour, and may be important for visual recovery. Indeed, all 7 eyes in which the continuity of the IS/OS junction line was restored had visual improvement of logMAR 0.3 or greater (mean final logMAR visual acuity, 0.047; range, -0.079 to 0.301). The regeneration of the IS/OS junction that is seen on SD-OCT may be caused by gliosis. The swelling of the glial cells may move the photoreceptors and may facilitate recovery of visual function.

With this new surgical technique, gas tamponade may be unnecessary. However, intraocular gas tamponade and face-down positioning for 24 hours after surgery may be required to ensure the placement of inverted epiretinal tissues inside the LMH.

Macular pigment may be able to protect against age-related macular degeneration due to its capacity to absorb blue light and scavenge free radicals. For this reason, surgeries in which the macular pigment is placed back inside the LMH may be suitable for relatively older patients. Based on our experience, this surgical procedure may be recommended as a standard surgery for LMH. ■

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