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Review Article

Vitreous Surgery for Idiopathic Macular Hole

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Idiopathic macular hole is a full-thickness defect of the neurosensory retina which can be treated by three-port vitrectomy. The pathogenesis is not understood completely, but tangential traction of the vitreous and retinal surface may cause opening of a macular hole. Vitrectomy with or without internal limiting membrane peeling may relieve the tangential traction. The anatomical success rate is 58-85% without internal limiting membrane peeling and 88-95% with internal limiting membrane peeling. A high success rate was achieved in patients who had surgery within 6 months of the onset of symptoms. Among the complications were retinal breaks, retinal detachment, retinal pigment epitheliopathy, and visual field defect, the most serious complication. Temporal visual field defect can be eliminated by reducing the fluid-gas exchange time and stabilizing intraocular pressure during surgery. Nasal visual field defect can be eliminated by the use of a lower concentration of indocyanine green or of triamcinolone acetonide instead of indocyanine green.

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Introduction

Idiopathic macular hole is characterized by a neurosensory retinal hole at the central fovea with localized subretinal fluid around the hole. Until recently, it has been considered to be untreatable. Visual acuity is decreased to less than 0.1, and patients complain of metamorphopsia and decreased central vision. Idiopathic macular holes occur most commonly in women but also in older men. In most cases macular hole is present in only one eye, but in about 10%, the other eye is also involved.

In 1991, Kelly and Wendel[1] proposed a new treatment for macular holes. They reported that macular holes can be closed by vitrectomy and gas tamponade in the face-down position. In their preliminary report, their success rate was 58%. Since their report, there have been many papers[2-11] describing closure of mcular holes. In this review, the pathogenesis, basic surgical techniques, rate of closure, visual acuity, improved technique, and complications of macular hole surgery are reported.

Pathogenesis of macular hole

The pathogenesis of macular hole is not yet understood completely, but tangential traction of the vitreous gel to the fovea (central point of the macula) may be the main cause of the opening of a macular hole.[12] So complete separation of the vitreous from the macular surface would be expected to facilitate closure of a macular hole. Gass classified macular holes from stage 1 to stage 4[13] and later he reappraised the classification.[14] Stage 1 is foveal detachment or small dehiscence at the fovea due to contraction of the vitreous gel as a change due to aging. Stage 2 is a small can-opener-like tear caused by a contracted prefoveal vitreous cortex. In stage 3 the hole is larger, and a stage 4 hole is posterior vitreous detachment, but some vitreous remnant around the hole prevents closure. So surgery is indicated to make an artificial posterior vitreous detachment (stages 1, 2 and 3), and to remove as much of the remnant of vitreous as possible (stage 4). If the vitreous remnant cannot be removed completely, gas tamponade or an adjunctive procedure facilitates attachment of the retina to the retinal pigment epithelium to oppose residual tangential traction.

Surgical techniques

Three-port vitrectomy is employed for macular hole surgery (Figure 1). Three-port vitrectomy means cutting the vitreous with three scleral incisions. After a conjunctival incision, full-thickness incisions of the eye wall (scleral incision) were made at 2:00

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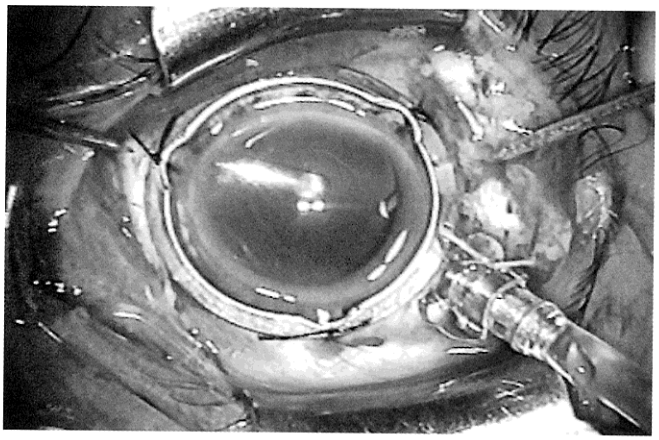

Figure 1. Three port vitrectomy. Three scleral incisions are made near the corneal limbus. An infusion cannula is fixed at 4:00 o'clock, and a vitreous cutter and illuminating light are inserted into the vitreous cavity to begin vitrectomy.

o'clock, 10:00 o'clock and 4:00 o'clock (left eye) or at 8:00 o'clock (right eye) near the corneal limbus. Through one scleral incision artificial aqueous humor is injected into the vitreous cavity to prevent ocular collapse. Through the other two incisions, a vitreous cutter and an illuminating light are inserted. After core vitrectomy, the attachment of the vitreous to the retina is severed by suction or mechanical lifting. Then almost all the vitreous is cut and removed, and the fluid in the vitreous cavity is replaced with 20% SF6 gas. The patient must remain in the face-down position for about a week.

Rate of closure of macular holes and of improvement of visual acuity

In their preliminary report, Kelly and Wendel[1] recorded a macular hole closure rate of 58%, and later Wendel et al[2] reported a higher rate of 80% when surgery was performed within 6 months of the onset of symptoms. Visual acuity improved by two or more lines in 68% and by four or more lines in 55% in the same group. The earlier macular hole surgery is performed after the onset of symptoms, the better is visual function. Many surgeons have begun to do macular hole surgery and are reporting better results. The anatomic success after one operation is reported to be 70-80% and improvement of visual acuity by two or more lines is recorded in 50-70%.[3-9] In chronic macular hole cases, the anatomic success rate is similar to that of acute holes, but poorer visual prognosis.[10-11] Reopening of macular holes occur in about 5% of cases.[15-16]

Improvement of technique

Macular holes are now treatable, but there are still many unsuccessful results. Several adjunctive variations have been tried to improve the rate of closure. Glaser et al[17] used transforming growth factor-ß2 (TGF-ß2), and Ligget et al[18] prepared autologous serum. The amounts used after fluid-gas exchange were small and they reported a higher closure rate. After that there have been many reports about TGF-ß2,[19-21] autologous serum or platelert,[22-26] glue,[27] plug,[28] retinal pigment epithelium debridement,[29] and endolaser.[30] However, their adjunctive methods have not become popular for macular hole surgery.

Brooks[31] reported that intentional peeling of the internal limiting membrane (ILM) facilitates the closure of macular holes. ILM, the innermost layer of the retina, is composed of the basement membranes of Mueller cells. The thickness of the ILM is 50 nm in the peripheral retina, 300nm in the equatorial retina, and 1900 nm in the posterior retina.[32] Biomicroscopically, ILM is transparent and curls easily (Figure 2). Electron microscopically, ILM is an amorphous structure in contact with the foot processes of Mueller cells (Figure 3, upper panel). Surgically removed ILM is also amorphous, and debris of Mueller cell foot processes adheres to its outer surface (Figure 3, lower panel). The reason that ILM peeling is effective for macular hole closure is as follows. Although ILM is a normal structure, it may cause tangential traction if it breaks, and ILM peeling can remove the vitreous remnant completely. The rate of closure was improved to 90-95% in many reports[33,34] with ILM peeling compared to an 80-85% success rate without ILM peeling,[2-8] visual function was comparable with or without ILM peeling. As the ILM is a transparent thin membrane (Figure 2), it is difficult even for expert surgeons to peel it off, and retinal damage such as retinal hemorrhage has been reported. In 2000, Kadonosono et al[35] developed ILM staining with indocyanine green (ICG) and reported that it is easier to peel off the ILM with ICG staining. They applied a mixture of ICG and hyaluronic acid gel to the macula after removal of the vitreous, because gel material was easier to treat than fluid material. Later, it became popular to use ICG without hyaluronic acid, since fluid ICG is also easy to use.[36,37] With the use of ICG staining, ILM peeling has become a safe and effective procedure. After removal of the vitreous, 0.25% of ICG is spread directly over the retina, a small incision of the ILM is made with a lancet, and the ILM is peeled off with a forceps (Figure 4).

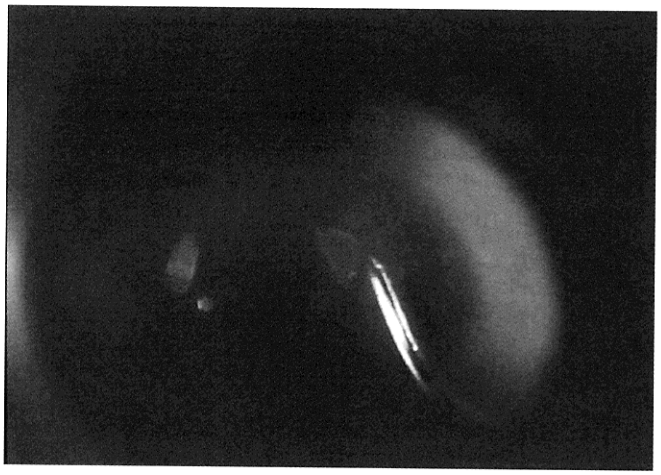

Figure 2. The transparent and curled internal limiting membrane is peeled off during the operation.

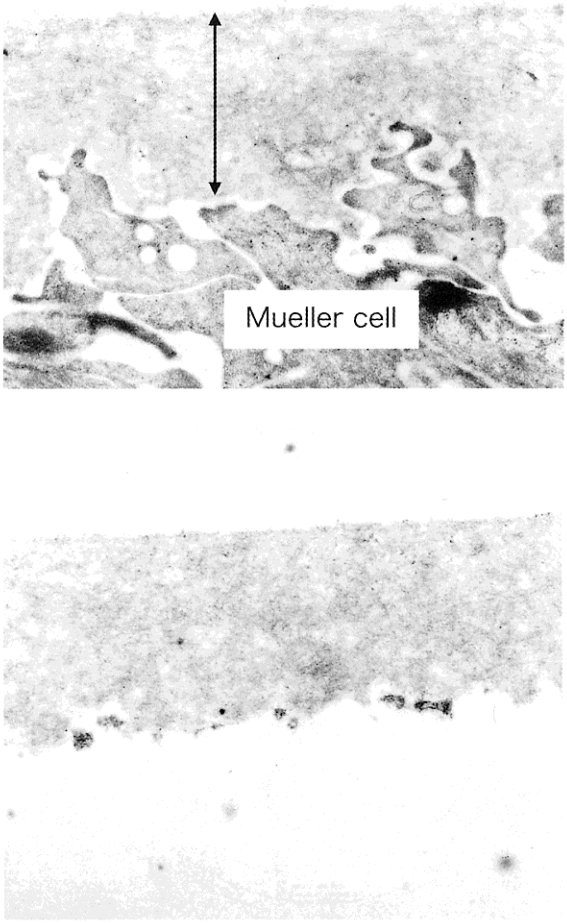

Figure 3. Electron microscopic images of inner retina (upper panel) and surgically removed internal limiting membrane (lower panel). The internal limiting membrane is an amorphous membrane connected to the Mueller cells. The double-headed arrow indicates the internal limiting membrane.

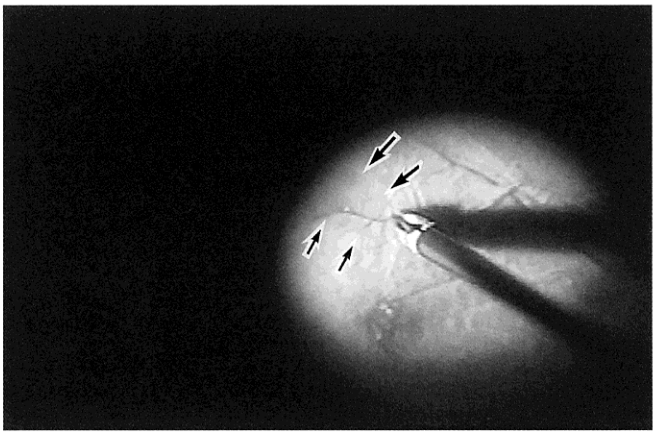

Figure 4. Indocyanine green staining of internal limiting membrane. With indocyanine green staining, the peeled off area of the internal limiting membrane is easily seen. The arrows indicate the edge of internal limiting membrane peeling.

Complications

As macular hole surgery became a popular procedure and many surgeons have employed it, several complications have been reported, such as retinal breaks and detachment,[38] retinal pigment epitheliopathy,[39] increased intraocular pressure,[40] and glaucoma.[41] These complications are not frequent, and some of them, such as retinal detachment and retinal breaks, are treatable.

In 1995, temporal visual field defect after macular hole surgery was reported as one of the serious complications[42] (Figure 5). After that initial report, there have been many publications[43-50] about temporal visual field defect. The incidence of temporal visual field defect is 10-30%. The possible mechanisms of its etiology are: gas flow during fluid-gas exchange can damage the retina directly,[45] and gas tamponade can produce mechanical compression of the optic nerve fibers;[46] artificial posterior vitreous detachment can cause mechanical trauma to the optic nerve;[47] circulatory disturbances such as retinal vein occlusion or ciliary artery occlusion may occur during surgery;[48] dryness of the retina caused by fluid-gas exchange may damage the retinal nerve fiber layer.[50,51] One report suggested that no temporal visual field defect occurred when a bent infusion cannula was used.[52] The etiology of temporal visual field defect is not clear, but it can be prevented by a shorter duration of fluid-gas exchange and by stabilizing the intraocular pressure during operation. Kuroki et al[53] reported that visual field defect could be treated by hyperoxigenation therapy.

Although with ICG staining ILM can be easily peeled off and a high rate of macular hole closure can be achieved, a new complication, nasal visual field defect has been observed (Figure 6). Gass et al[54] reported nasal visual field defects in 50% of patients with ICG staining. The incidence of nasal visual field defect varies from 5 to 50%.[54-56] The etiology of visual field defect with ICG staining is unknown, but two possibilities have been proposed. One is toxicity of ICG itself and the other is ICG-enhanced phototoxicity.[54] Low intensity of illuminating light and lower concentration (0.25% or less) of ICG may eliminate nasal visual field defect.[55] Recently, Kimura et al[57] reported ILM peeling was performed with the use of triamcinolone acetonide (TA). TA seems to be less toxic than ICG. TA-assisted ILM-peeling has less visibility of ILM, but macular hole surgeons may have to use TA instead of ICG.

Conclusion

Surgical removal of vitreous gel and tangential traction can close macular holes and improve visual acuity. Internal limiting membrane peeling results in a high success rate for macular hole closure and a good visual outcome. Serious complications, such as temporal and nasal visual field defects may occur, but can be eliminated by careful fluid-gas exchange and use of indocyanine green.

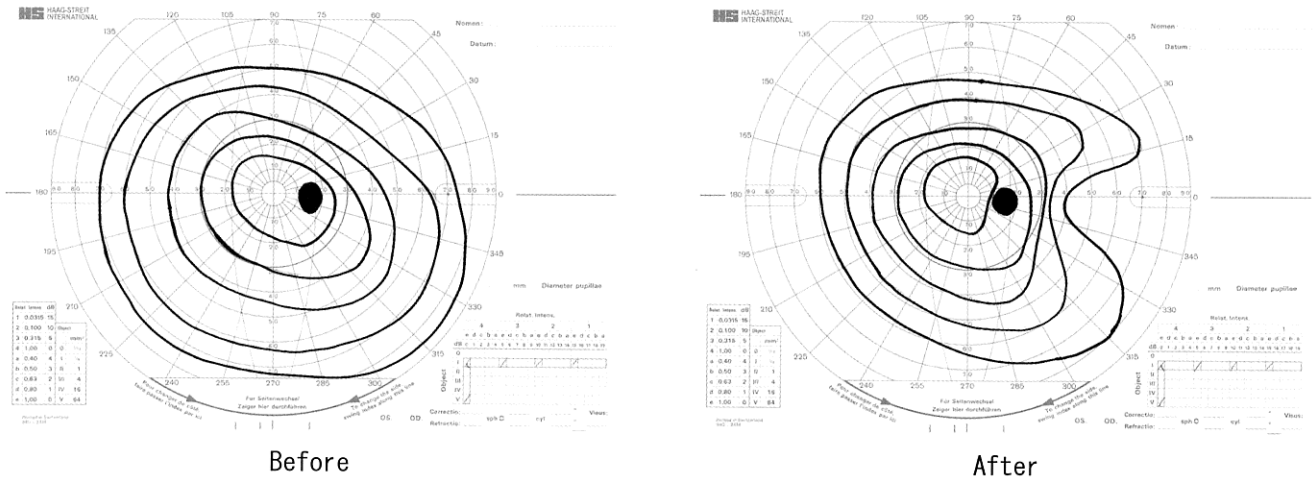

Figure 5. Visual field before (left panel) and after (right panel) macular surgery. A temporal visual field defect appeared after surgery.

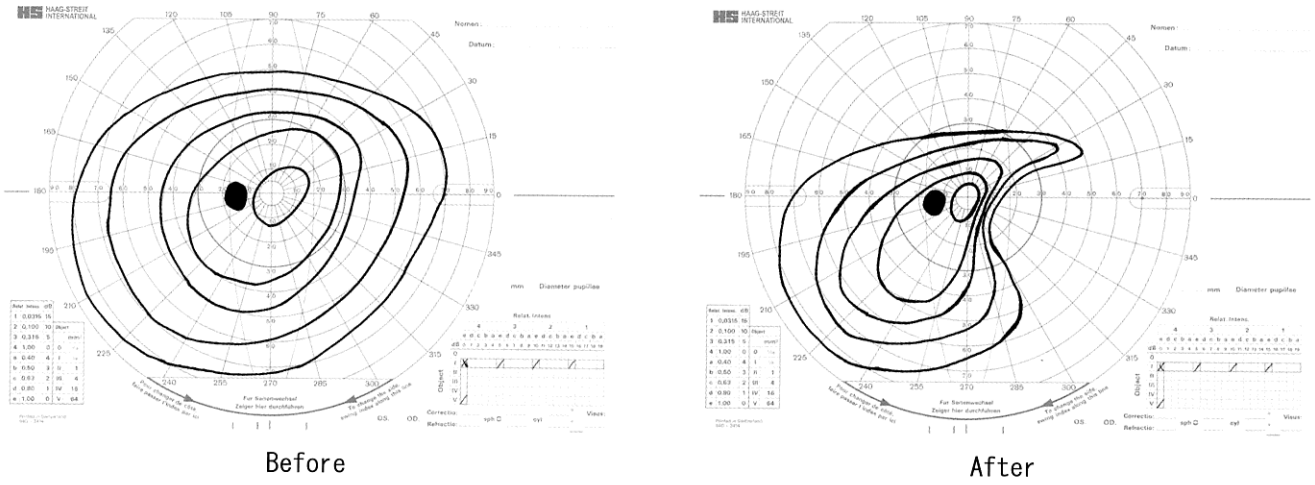

Figure 6. Visual field before (left panel) and after (right panel) macular surgery with indocyanine green staining. A nasal visual field defect appeared after surgery.

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