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Macular Hole Surgery-Associated Peripheral Visual Field Loss

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Abstract: To examine in more detail the peripheral visual field loss after macular hole surgery, we reviewed a series of 38 consecutive patients (44 eyes) with idiopathic macular hole who underwent vitrectomy and fluid–gas exchange. Ten (22.7%) eyes of 9 patients developed peripheral visual field loss shortly after successful surgery. This complication was characterized by mild to moderate wedge-shaped visual field loss that predominantly affected the inferotemporal periphery. Of these 9 patients, 2 complained of peripheral visual field loss, and the 7 others remained asymptomatic. The peripheral visual field loss remained unchanged for a mean follow-up of 18.5 months, except in one case of complete recovery. The thickness of the retinal nerve fiber layer was measured postoperatively to determine whether any damage to the optic nerve head had occurred during surgery. The information obtained in this study did not provide conclusive evidence for the understanding of the pathomechanism of the macular hole surgery-associated visual field loss. Peripheral visual field defect after otherwise uneventful surgery for idiopathic macular hole is probably not uncommon. This complication is variable in its severity and is usually permanent. Whether it is caused by any surgical trauma to the optic nerve head remains to be elucidated. **Jpn J Ophthalmol 1998;42:476–483** © 1998 Japanese Ophthalmological Society

Key Words: Idiopathic macular hole, outcome of surgery, peripheral visual field loss, retinal nerve fiber layer, vitrectomy.

Introduction

Vitrectomy with fluid–gas exchange is now widely recognized as the recommended management for idiopathic macular holes.[1–2] In recent years, peripheral visual field loss has been reported to occur after an otherwise uncomplicated surgery. The first description by Melberg and Thomas[3] of this complication has been confirmed by subsequent reports. Nearly all previous studies were performed on patients who had complaints of postoperative peripheral visual field defect. The incidence and long-term functional outcome of this complication remain to be elucidated. The pathomechanism of this complication is also inconclusive, although several candidate factors, including intraoperative interference with the nerve fibers in the optic nerve head, have been suggested.[4–7]

We report herein 10 eyes of 9 patients with idiopathic macular holes who had peripheral visual field loss after uneventful macular hole surgery. Five of the 9 patients had had preoperative perimetric evaluations. In addition, we assessed the thickness of the retinal nerve fiber layer (RNFL) to determine whether optic nerve fibers were damaged during surgery.

Subjects and Methods

We reviewed a series of 38 consecutive patients who underwent vitrectomy with fluid–gas exchange for idiopathic macular hole between November 1993 and May 1997. Six had bilateral disease and underwent bilateral surgeries, hence a total of 44 eyes

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were reviewed. All patients were hospitalized 3 days before and at least 2 weeks after surgery. Routine pre- and postoperative examinations included ocular and medical history, visual acuity test, applanation tonometry, slit-lamp biomicroscopy, indirect binocular ophthalmoscopy, and fundus photography. Ocular ultrasonography, electroretinography, and fluorescein angiography were performed in some cases. Preoperative visual field test using a Goldmann perimeter was performed in 24 (54.5%) cases. The clinical information of these cases is summarized in Table 1. The stage of the macular hole was evaluated by the Gass criteria,[8] and 11 eyes were classified as stage 2, 22 as stage 3, and 11 as stage 4 macular holes. The interval from the presumed disease onset to the time of surgical management averaged 6.3 months (range: 18 days–48 months).

Vitrectomy with fluid–gas exchange was performed by the second author (AU) using standardized techniques and procedures. In brief, the lens was extracted by phacoemulsification. The central core of the cortical vitreous was removed through the pars plana, the posterior cortical vitreous adherent to the optic nerve head was peeled with a soft-tipped silicone cannula to create a posterior vitreous detachment, and the vitreous gel was excised as much as possible. Removal of the retinal pigment epithelium from the base of the macular hole was done in cases with a relatively large macular hole and those with repeat surgery. Epimacular membranes were peeled when indicated. After the insertion of an intraocular lens in the capsular bag in selected cases, fluid–gas exchange and closure of the sclerotomies, the vitreous cavity was flushed with 30 mL of 10–30% sulfur hexafluoride (SF_6). Postoperatively, patients were instructed to maintain a strict face-down positioning for at least 10 days. Goldmann perimetry was first performed when the intraocular gas bubbles were absorbed and repeated at each outpatient visit. The mean follow-up period was 18.5 months (range: 7–41 months).

Fifteen eyes were randomly selected for the measurement of the thickness of the RNFL postoperatively, using a commercially available instrument (Nerve Fiber Analyzer, Laser Diagnostic Technology, San Diego, CA, USA).[9] In brief, on the basis of confocal scanning laser polarimetry, the instrument yielded 20 images for different polarization states of the test beam to quantify the RNFL thickness. To minimize bias, all measurements were carried out by one of the authors (SO), an experienced operator, who was masked as to the affected eye. Measure-

Table 1. Clinical Information of Patients Undergoing Macular Hole Surgery (Comparison Between Eyes With and Without Postoperative Visual Field Loss)

	All Eyes (n = 44)[a]	Field Loss (n = 10)[b]	No Field Loss (n = 34)[c]	P[d]
Age (years)[e,f]	64.1 ± 6.4	68 ± 6.0	63.3 ± 6.0	0.221
Sex (male/female)[e]	12/32	2/8	10/24	0.121
Side affected				
Right eye	18	2	16	0.194
Left eye	26	8	18	
Stage of macular hole				
Stage 2	11	1	10	0.003
Stage 3	22	9	13	
Stage 4	11	0	11	
Size of macular hole (disc diameter)[f]	0.25 ± 0.12	0.21 ± 0.06	0.27 ± 0.13	0.300
Interval from onset to surgery (months)[f]	6.3 ± 8.9	3.5 ± 3.2	7.2 ± 9.9	0.088
Removal of retinal pigment epithelium	15	4	11	0.324
Peeling of epiretinal membrane	5	1	4	0.372
Inadvertent retinal break	6	3	3	0.199
Simultaneous intraocular lens implantation	27	6	21	0.599
Postoperative intraocular pressure elevation	4	0	4	0.351

[a]44 eyes of 38 patients.
[b]10 eyes of 9 patients.
[c]34 eyes of 29 patients.
[d]Probabilities were calculated by Fisher's exact test, except for age, size of macular hole, and interval from disease onset to surgery where Mann-Whitney U-test was used.
[e]Data calculated twice for 6 patients undergoing surgery for bilateral disease.
[f]Mean ± SD.

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ments were obtained in the four peripapillary quadrants (superior, temporal, inferior and nasal), each measurement 1.5 disc diameter away from the optic disc margin. Control data were obtained from a series of 35 normal volunteers aged 50 years or older (mean age, 64.9 years).

Results

Intraoperative and postoperative complications are shown in Table 1, including inadvertent retinal breaks in 6 eyes (13.6%), which were treated with photocoagulation and did not cause postoperative retinal detachment or visual field defects. Four eyes (9.1%) developed an elevation of the intraocular pressure, which lasted for a few postoperative days and resolved with acetazolamide.

Favorable surgical results with closure of the macular hole were obtained in 36 eyes after the initial surgery and in 5 eyes after repeat surgery, hence the overall anatomical success was 41 (93.2%) of 44 eyes.

Peripheral Visual Field Loss

Of the 44 eyes of 38 patients, 10 eyes (22.7%) of 9 patients developed peripheral visual field loss after surgery (Figure 1). Table 2 summarizes their clinical information; 1 patient had complications in both eyes (Case 1 and Case 6). Two of the 9 patients (Case 6, Case 7) complained of peripheral visual field loss in the operated eye soon after the intraocular gas bubbles were absorbed. The remaining 7 patients were asymptomatic for peripheral vision despite a distinct peripheral visual field loss revealed by Goldmann perimetry performed 2 to 3 weeks after surgery. Preoperative perimetries performed on 5 of these 10 eyes were unequivocally within normal limits in the peripheral visual field. This indicated that the postoperative peripheral visual field loss was directly associated with the surgical damage (Figure 2).

The macular hole surgery-associated peripheral visual field loss was characterized by a wedge-shaped defect predominantly affecting the inferotemporal field; one case (Case 10 in Table 2) had a loss in the superotemporal far periphery. The extent of the visual field loss was variable in the affected eyes and ranged from a moderate loss consisting of an absolute scotoma extending to within 20 to 40° of fixation, to a relative scotoma extending to the physiologic blind spot (Case 3, Case 7, and others), and to a mild loss in the far periphery (Case 10). These visual field losses remained unchanged in the majority of cases on repeat tests during a mean follow-up of 18.5 months (range, 7–41 months). Figure 2 illustrates a

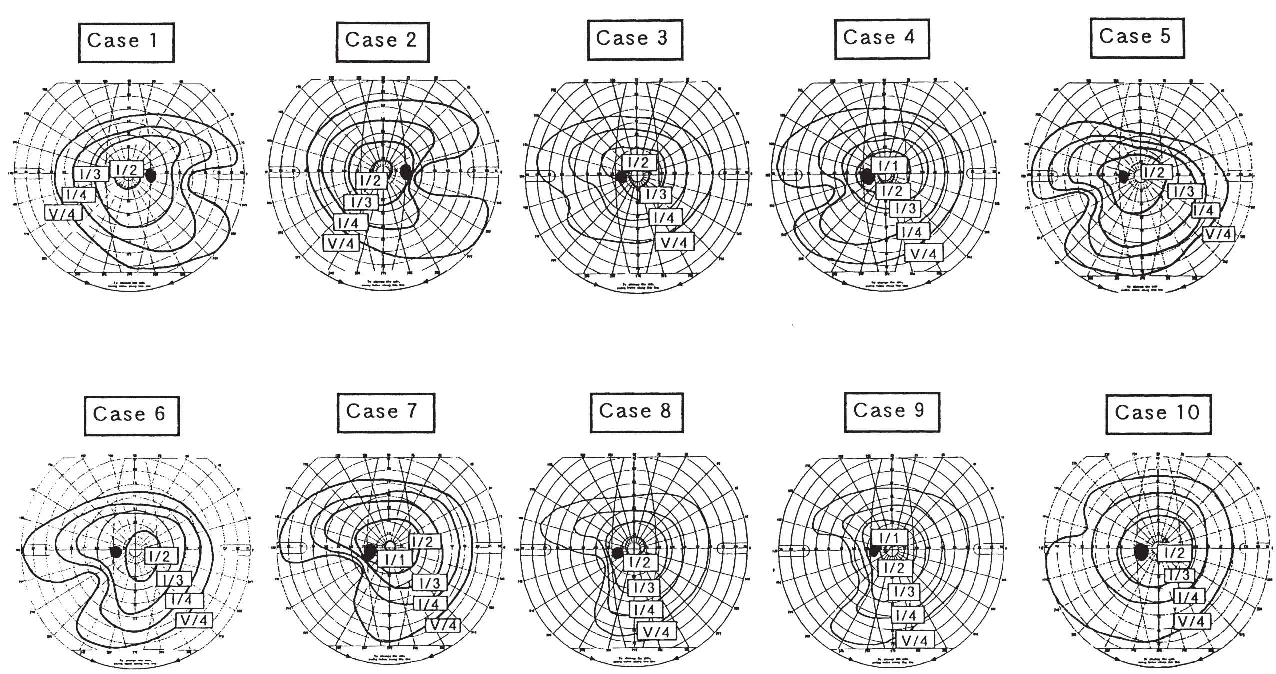

Figure 1. Goldmann perimetric visual fields obtained from 10 eyes of 9 patients who underwent vitrectomy with fluid–gas exchange for idiopathic macular hole. These fields illustrate postoperative peripheral visual field loss. Clinical information of cases is described in Table 2.

Table 2. Clinical Data of Patients With Peripheral Visual Field Loss Following Surgery for Idiopathic Macular Hole

	Case 1[a]	Case 2	Case 3	Case 4	Case 5	Case 6[a]	Case 7	Case 8	Case 9	Case 10
Age (years)/sex	66/Female	60/Female	65/Female	66/Male	70/Female	66/Female	53/Female	59/Female	64/Female	65/Male
Medical history	No	Hyper-tension	No	No	No	No	No	Hyper-tension	Hyper-tension	No
Macular hole										
Interval after visual symptom	18 days	2 months	3 months	4 months	2 months	3 months	2 months	3 months	1 month	12 months
Side	Right	Right	Left	Left	Left	Left	Left	Left	Left	Left
Stage	Stage 3	Stage 3	Stage 3	Stage 3	Stage 2	Stage 3	Stage 3	Stage 3	Stage 3	Stage 3
Size (disc diameter [μm])	0.25	0.18	0.10	0.25	0.27	0.25	0.17	0.17	0.14	0.25
Posterior vitreous detachment	No	No	No	No	No	No	No	No	No	No
Epiretinal membrane	No	No	Yes	No	No	No	No	No	No	No
Gas tamponade, SF_6 (%)	30%	10%	30%	30%	20%	25%	25%	30%	30%	30%
Anatomic closure of macular hole	Repeat surgery	Repeat surgery	Initial surgery	Initial surgery	Initial surgery	Initial surgery	Initial surgery	Initial surgery	Initial surgery	Initial surgery
Visual acuity (corrected)										
Preoperative	0.3	0.2	0.1	0.15	0.4	0.07	0.2	0.15	0.06	0.2
Postoperative	0.6	0.2	0.2	0.2	0.8	0.4	0.8	0.2	0.2	0.8
Peripheral visual field										
Preoperative visual field	Examined normal	Not exam-ined	Not exam-ined	Examined normal	Not exam-ined	Not exam-ined	Not exam-ined	Examined normal	Examined normal	Examined normal
Postoperative visual complaint	No	No	No	No	No	Yes	Yes	No	No	No
Postoperative visual field defect	Temporal	Temporal	Infero-temporal	Infero-temporal	Infero-temporal	Infero-temporal	Infero-temporal	Infero-temporal	Infero-temporal	Supero-temporal
Postoperative optic disc pallor	No	No	No	No	No	No	Mild	No	No	No
Postoperative IOP elevation	No	No	No	No	No	No	No	No	No	No
Follow-up period (months)	18	41	15	7	17	24	30	7	9	17

IOP: Intraocular pressure, SF6: sulfer hexafluoride.
[a]Case 1 and Case 6 are 2 eyes of same patient.

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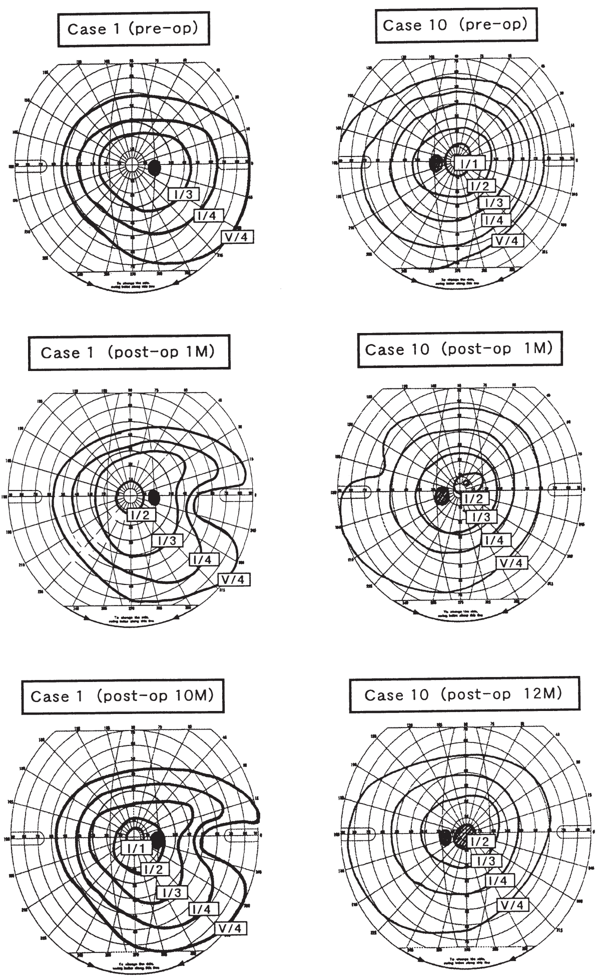

Figure 2. Left three visual fields illustrate preoperative and postoperative visual fields of Case 1 in Table 2. Patient had normal peripheral visual fields preoperatively. Although patient was asymptomatic for peripheral vision after successful macular hole surgery, field tests at 1 month postoperative revealed inferotemporal wedge-shaped defect. Defect remained unchanged 10 months after surgery. Right three visual fields show preoperative and postoperative visual fields of Case 10 in Table 2, illustrating preoperative normal peripheral visual field and postoperative mild defect in superotemporal periphery 1 month after surgery. Patient remained asymptomatic. Follow-up examination revealed that defect was reversible with only subtle loss at 6 months, and return to normal 12 months after surgery.

representative case of an irreversible visual field defect. However, one eye, the mildest among the affected cases (Case 10 in Table 2), showed complete recovery 12 months after surgery (Figure 2).

With regard to postoperative ophthalmoscopy and fluorescein angiography, only one case (Case 7 in Table 2) showed a subtle optic disc pallor in the superotemporal area, and the optic disc and the peripheral retina were unremarkable in the remaining 9 cases.

To determine whether any clinical or surgical factors were responsible for the macular hole surgery-associated peripheral visual field loss, a comparison was performed between 10 eyes with visual field loss and the 34 eyes without visual field loss. Table 1 summarizes the data with statistical analyses. There was no significant difference in age or sex between the two groups. With regard to the laterality of involvement, 2 (11.1%) of 18 right eyes and 8 (30.8%) of 26 left eyes were affected; thus, the left eye tended to be more vulnerable, although the difference was not statistically significant. The stage of the macular hole was significantly related to the incidence of postoperative visual field loss; namely, 10 (30.3%) of 33 eyes with stage 2 or 3 macular hole developed visual field loss, while none of 11 eyes with stage 4 showed any loss. The size of the macular hole was unrelated to the incidence of visual field loss. The mean interval from presumed disease onset to surgery was estimated to be 3.5 months in the visual field loss group and 7.2 months in the no visual field loss group; this difference was not significant. Surgical procedures, such as the removal of the retinal pigment epithelium from the base of the macular hole, inadvertent retinal break, and simultaneous intraocular lens implantation did not influence the development of postoperative visual field loss. Postoperative transient rise of the intraocular pressure occurred in none of the visual field loss group and in 4 of the no visual field loss group, but this unexpected findings was not statistically significant.

Thickness of Retinal Nerve Fiber Layer

The peripapillary thickness of the RNFL in 35 normal subjects showed marked individual variation. It also varied in the four quadrants, measuring 75.7 ± 14.9 μm (mean ± SD) in the superior, 42.4 ± 10.3 in the temporal, 78.5 ± 15.4 in the inferior, and 51.9 ± 9.6 in the nasal quadrants. Thus, the vertical quadrants were significantly thicker (analysis of variance, $P < 0.0001$).

The RNFL thickness was evaluated postoperatively in 15 eyes with idiopathic macular hole, including the 5 eyes that developed peripheral visual field loss. The measurements of the RNFL thickness in the four peripapillary quadrants of the operated eyes are compared with those from normal subjects of comparable ages (Figure 3). The RNFL thickness measured 69.7 ± 12.6 μm (mean ± SD) in the supe-

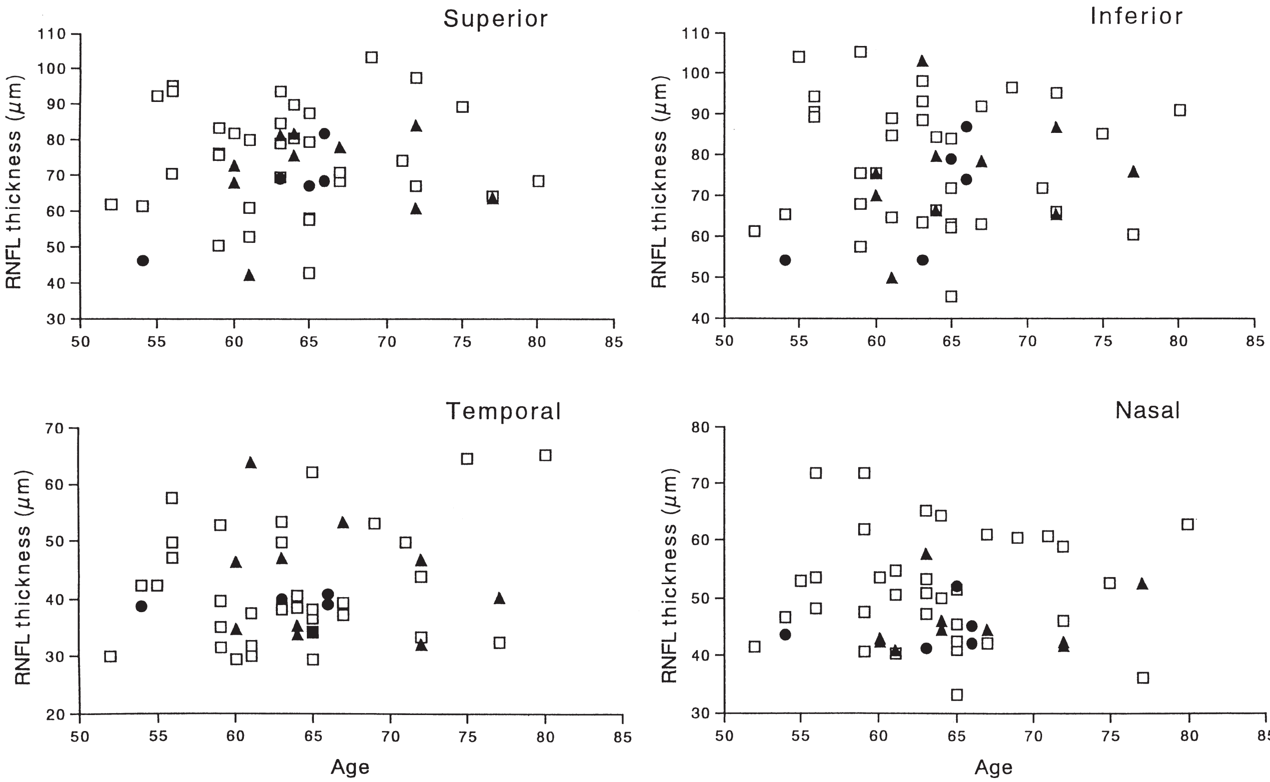

Figure 3. Thickness (μm) of retinal nerve fiber layer (RNFL) in four peripapillary quadrants, plotted against age. Five eyes with field loss after macular hole surgery ●; 10 eyes with no field loss after macular hole surgery ▲; 35 normal control subjects □. It is obvious that there is marked scatter of data points in each group, making it difficult to distinguish normal from operated eyes. Note data from field loss group overlap those from no field loss group. These features also apply for nasal quadrant where RNFL was statistically thinner in operated eyes.

rior, 42.0 ± 8.5 in the temporal, 73.4 ± 14.1 in the inferior, and 45.5 ± 4.9 in the nasal quadrants of the operated eyes. A comparison with the control eyes revealed no significant difference in the superior, temporal, and inferior quadrants, but in the nasal quadrant, the RNFL thickness was significantly thinner in the 15 operated eyes ($P = 0.0036$). It is also noticeable in Figure 3 that the majority of data points are scattered and overlap those from the controls and the 5 eyes with postoperative peripheral visual field loss are not distinct from the other 10 cases with no visual field loss.

An additional comparison of the RNFL thickness was made between the operated and nonoperated eyes of the 10 patients who underwent unilateral macular hole surgery (Table 3). Although the operated eyes had lower values, a few of the operated eyes had thicker RNFL than the nonoperated eyes. There was no significant difference in any quadrants between the operated and nonoperated eyes.

Discussion

These results provide additional information on the peripheral visual field loss after successful surgical management of idiopathic macular holes. First, previous reports[3–7] described perimetric results that were obtained only postoperatively. The present prospective perimetric studies demonstrated that the preoperatively normal peripheral visual fields were altered soon after surgery, hence giving unequivocal evidence for the association between surgical intervention and postoperative visual field loss. Second, previous reports dealt with those patients who complained of postoperative peripheral visual field loss due to moderate to severe visual field defect up to a general peripheral contraction. In two large series, this complication was reported in 9 (7%) of 125 cases[4] and 8 (16%) of 47 cases.[7] Thus, the incidence in the present series, 10 (22.7%) of 44 cases, is slightly higher than that in the previous reports. It is of interest in this connection that 8 of the present 10

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Table 3. Peripapillary RNFL Thickness in Operated and Nonoperated Eyes of 10 Patients With Unilateral Idiopathic Macular Hole

	Superior Quadrant		Inferior Quadrant		Temporal Quadrant		Nasal Quadrant	
Case	Operated	Nonoperated	Operated	Nonoperated	Operated	Nonoperated	Operated	Nonoperated
1[a]	69.1	69.9	54.3	63.3	40.1	53.6	41.3	47.3
2[a]	46.4	61.8	54.1	65.4	39.0	42.6	43.9	46.7
3[a]	67.2	88.0	78.9	84.2	34.3	38.3	52.3	41.0
4	73.2	72.0	70.2	82.6	46.5	43.2	42.7	49.6
5	82.2	90.2	79.9	84.5	35.5	38.5	44.7	64.5
6	75.9	80.9	66.2	66.2	34.2	40.7	46.1	50.2
7	63.9	64.6	76.2	60.4	40.5	32.7	52.8	36.2
8	84.7	97.8	86.9	95.3	46.9	44.1	42.0	58.9
9	68.3	82.4	75.8	75.8	34.9	29.7	43.2	53.7
10	78.3	69.0	78.4	62.9	53.6	39.6	44.6	42.4
Mean	70.9	77.7	72.1	74.1	40.6	40.3	45.4	49.1
SD	10.9	12.0	10.9	12.0	6.5	6.5	4.0	8.4
t[b]	−1.314		−0.383		0.086		−1.247	
P[b]	0.205		0.706		0.993		0.229	

[a]Cases of peripheral visual field loss after surgery. 1 = Case 2; 2 = Case 7; 3 = Case 10 in Table 2.
[b]Two-sample paired t-test between the operated and nonoperated eyes.

affected cases showed mild to moderate visual field defect on perimetry, but remained asymptomatic so that the loss of visual field testing was performed. Further careful preoperative as well as routine postoperative perimetric studies are justified to determine whether the complication is more common than recognized to date.

A review of the literature together with the present cases, indicates that macular hole surgery-associated peripheral visual field loss is characteristic in its location and pattern. The temporal or inferotemporal visual field is predominantly affected and the defect is usually wedge-shaped. The severity of the defect is variable among cases with some developing mild, focal involvement of the far periphery and others suffering wide and severe impairment up to general contraction of the peripheral visual fields. With regard to the functional outcome, previous reports emphasized the irreversible feature of this complication.[4–7] Although the present long-term follow-up results confirm that the defect is usually permanent, it is noteworthy that one of our patients showed complete recovery. This unusual case was distinct in that mild visual field loss developed in the superotemporal far periphery, and may indicate that visual field loss is not irreversible.

Determination of risk factors for macular hole surgery-associated peripheral visual field loss is of practical importance. Our analyses indicate that, while there is no significant association with age, sex, interval from the presumed disease onset to surgery, or surgical procedures, this complication occurred in patients with stage 2 or 3 macular hole, but not with stage 4. This finding is consistent with previous analyses.[7] The left eye appeared more vulnerable than the right in the present series of cases, but the difference was not statistically significant. This is compatible with the results in the literature,[3–7] which report a comparable number of right and left eyes affected.

The pathogenesis of macular hole surgery-associated visual field loss remains to be defined. It has been suggested that intentional peeling of the posterior vitreous cortex could lead to damage of the optic nerve fibers. The occurrence of the complication in stage 2 or 3 macular hole and predominant involvement of the temporal visual field favor the mechanical traction hypothesis. The traction hypothesis is supported by the stronger attachment of the posterior vitreous cortex to the nasal margin of the optic nerve head.[9] Hutton et al[10] reported a significant decrease in RNFL thickness in 6 of the 8 patients who developed peripheral visual field loss after successful macular surgery. In the present study, the RNFL thickness was significantly thinner on the nasal quadrant of the operated eyes. However, a conclusion must be reserved because the difference from the normal control was only statistical with the majority of measurements overlapping the normal values. Furthermore, measurements were obtained only postoperatively, and there was no difference between the operated and nonoperated eyes of the same patient. A possible explanation for our failure to detect a distinct decrease in RNFL thickness is that the damage to the optic nerve head was not severe enough to be detected. It is of interest in this connection that only 1 of our 10 affected cases developed optic disc pallor. Alternatively, the current technique for RNFL thickness measurement lacks sensitivity and precision to detect subtle abnormalities. In any event, further studies are justified to ascertain whether damage to the optic nerve fibers is relevant to the pathomechanism of peripheral visual field loss after macular hole surgery.

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