

Fellow Eye Findings of Highly Myopic Subjects Operated for Retinal Detachment Associated with a Macular Hole

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Purpose: To identify anatomic risk factors involved in the onset of retinal complications causing decrease of visual acuity (VA) in the fellow eyes of highly myopic patients operated for retinal detachment with macular hole (RDMH).

Design: Cohort study.

Participants: Ninety-eight patients (mean age, 51.5 ± 8.0 years) with bilateral high myopia (mean myopia of the fellow eye, 20.4 ± 5.5 diopters) affected by RDMH in the other eye at baseline.

Methods: Evaluation of the anatomic features at baseline and during 84 ± 2.7 months of follow-up by biomicroscopic examination, indirect binocular ophthalmoscopy, B-scan ultrasonography, and optical coherence tomography.

Main Outcome Measures: Detection of anatomic features associated with onset of retinal complications causing decrease of VA during the follow-up period.

Results: The fellow eyes were divided into 2 groups according to the clinical features of the RDMH eyes: Group 1, presence of posterior vitreous detachment (PVD); and Group 2, presence of posterior vitreous schisis (PVS). At baseline, the incidence of PVD in group 1 was 31 of 47 eyes (65.9%) and the incidence of PVS in Group 2 was 42 of 51 eyes (82.3%). At the end of follow-up, group 1 eyes had a lower incidence of retinal complications causing visual decrease than group 2 eyes (group 1, 2/47 eyes; group 2, 9/51 eyes).

Conclusions: Fellow eyes of RDMH cases with higher degree of myopia and peculiar vitreoretinal features including PVS, posterior epiretinal membrane, severe posterior staphyloma, and chorioretinal atrophy are more likely to develop retinal complications causing decrease of VA.

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Eyes with high myopia (>6 diopters [D] and with a corresponding axial length usually >26 mm)¹ are reported to have a higher incidence of retinal detachment (RD) associated with macular hole (RDMH) compared to emmetropic eyes.^{2,3} Chorioretinal defects, posterior staphyloma, myopic foveoschisis, and vitreous changes are some anatomic factors that seem to have a role in the genesis of RDMH in high myopic eyes.^{4–17}

Some authors have already reported the risk and the incidence of bilateral RDMH in myopic fellow eyes^{2,3}; however, as far as we are aware, there is a lack of information regarding the anatomic factors that may be involved in the onset of the same or different retinal pathologies in fellow eyes of highly myopic patients affected by RDMH and with a similar degree of myopia.

In a previous study, we reported the surgical techniques used for the repair of RDMH in highly myopic eyes. The studied eyes were divided into 2 groups on the basis of their vitreoretinal features. The choice of the surgical technique employed depended on the vitreous features of the eyes, the extent of the chorioretinal atrophic areas, and the presence of a posterior staphyloma; all these characteristics were related to the degree of myopia.¹⁸

The fellow eyes of operated cases of RDMH were in-

cluded in this work, and 18 are the object of the present study, with the aim of identifying anatomic risk factors involved in the onset of retinal complications causing a decrease of visual acuity (VA).

Materials and Methods

One hundred twenty fellow eyes of 120 highly myopic patients who underwent surgery for RDMH repair, already reported in a previous study, were examined between February 1997 and September 1999.¹⁸ Patients with bilateral high myopia and an asymptomatic fellow eye were included in the study. Informed consent was obtained from each patient.

Exclusion criteria were presence or history of glaucoma (1 eye), uveitis (1 eye), eyes with dense opacity of the lens at baseline (2 eyes), eyes subjected to retinal surgery or extensive prophylactic laser treatments before the baseline examination (7 eyes), or patients who missed the scheduled follow-up controls (6 eyes).

Because surgical treatment of cataract in myopic eyes may contribute to the development of posterior vitreous detachment (PVD) and RD,¹⁹ by including these eyes we would have been unable to discriminate whether the onset of vitreoretinal modifications during follow-up was related to cataract surgery or whether they developed independently from surgery. We therefore decided

to exclude 5 eyes in which cataract surgery was performed before baseline or during the follow-up period.

The application of these criteria led to the overall exclusion of 22 fellow eyes of 22 patients (13 in group 1 and 9 in group 2); therefore, 98 eyes of 98 patients (45 male and 53 female) were considered for the study. The age ranged from 42–68 years (mean, 51.5 ± 8.0), and myopia ranged from 14–30 negative D (mean, 20.4 ± 5.5). The enrolled eyes were studied at baseline and every 6 ± 3.5 months for a total follow-up period of 84 ± 2.7 months.

At each timepoint the anatomic features of the eyes were evaluated by means of the following examinations.

- (1) Indirect ophthalmoscopy, slit-lamp biomicroscopy using a +90 D no-contact lens (Volk Optical, Mentor OH) after pupillary dilation using tropicamide 1%.
- (2) A-scan and B-scan ultrasonography using a 10-MHz probe (Humphrey Instruments, San Leandro, CA).
- (3) Optical coherence tomography (OCT) examination performed by a commercially available instrumentation (OCT1; OCT3 Stratus; Carl Zeiss Ophthalmic Systems, Inc. Humphrey Division, Dublin, CA). A scanning superluminescent diode (820 nm) was used to scan and analyze the macular area. After pupillary dilation, OCT images of the macular region were obtained with a series of horizontal and vertical scans, with a length of 6.0 mm. Axial resolution in tissue was 10 microns for OCT1 and OCT3; the transverse resolution was 20 microns for both OCT1 and OCT3.
- (4) Best-corrected VA was assessed by modified Early Treatment Diabetic Retinopathy Study table (Lighthouse Low Vision Products, Long Island City, NY); VA was expressed in logarithm of minimum angle resolution values obtained at the distances of 4, 2, 1, and 0.5 m.

In the previous study, the choice between different surgical techniques for RDMH repair in highly myopic eyes depended on vitreous features, extent of chorioretinal atrophic areas, and presence of posterior staphyloma.¹⁸ Preoperative vitreous modifications in RDMH eyes were essentially represented by a PVD and a posterior vitreous schisis (PVS). A PVD is defined as a separation of the posterior vitreous cortex from the internal limiting lamina of the retina, whereas PVS is defined as an anomalous form of vitreoretinal separation, characterized by a forward displacement of the anterior portion of the posterior vitreous cortex, leaving part of the posterior portion of the vitreous cortex still attached to the retina.^{20,21} The presence of PVD or PVS was correlated to the degree of myopia and represented the precipitating factor for the onset of RD with a macular hole, influencing its anatomic characteristics. Considering these observations, and assuming that both eyes of the same patient with an interocular difference of myopia < 6 -D (see below) could share common vitreoretinal characteristics, we divided fellow eyes into 2 groups on the basis of vitreous characteristics (presence of PVD or PVS) of the operated RDMH eye.¹⁸ Therefore, the fellow eyes of patients with a PVD in the RDMH eye were included in group 1 and the fellow eyes of patients with a PVS in the RDMH eye were included in group 2.

The diagnosis of PVD or PVS resulted from a combination of the different depictions of the fundus obtained by indirect ophthalmoscopy, indirect biomicroscopic fundus examination with a +90-D lens, B-scan ultrasonography, and OCT findings, as described previously.¹⁸

Group 1 included 47 eyes belonging to 27 female and 20 male patients ranging in age from 47 to 68 years (mean, 52.6 ± 8.4) and with a degree of myopia (range, 14–27 -D; mean, 16.20 ± 3.54 ; axial length, 29.11 ± 1.06 mm). Mean VA was 0.16 ± 0.12 .

Group 2 included 51 eyes belonging to 26 female and 25 male subjects ranging in age from 42 and 61 years (mean, 50.4 ± 7.6) and with

Table 1. Clinical and Vitreoretinal Features of the Eyes of Groups 1 and 2 at Baseline

	Group 1	Group 2
N	47	51
Age (yrs)	52.64 ± 8.45	50.43 ± 7.58
Gender (M/F)	20/27	25/26
Myopia (diopters)	-16.19 ± 3.54	-24.33 ± 3.77
Axial length (mm)	29.11 ± 1.06	31.43 ± 1.13
Visual acuity (LogMAR)	0.16 ± 0.12	0.21 ± 0.09
VL	16 (34.0%)	3 (5.8%)
PVD	31 (65.9%)	6 (11.8%)
PVS	—	42 (82.3%)
Mild chorioretinal atrophy	33 (70.2%)	—
Posterior staphyloma with severe chorioretinal atrophy	13 (27.6%)	51 (100%)

F = female; LogMAR = logarithm of minimum angle resolution; M = male; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; VL = vitreous liquefaction.
Values are expressed as means \pm 1 standard deviation.

a degree of myopia (range, 18–30 -D; mean, -24.33 ± 3.77 ; axial length, 31.43 ± 1.13 mm). Mean VA was 0.21 ± 0.09 .

In each group 1 and group 2 patient, myopic interocular difference did not exceed 6 D (see mean myopic values in Table 1 of our previous manuscript¹⁸).

Demographic data of enrolled eyes are presented in Table 1.

Statistical Analysis

Values are expressed as means \pm 1 standard deviation (SD). The differences observed between groups were evaluated by 2-by-2 tables and the chi-square test. Differences in VA between groups and between values observed at the end of follow-up with respect to baseline were analyzed by 1-way analysis of variance.

The incidence of pathologic events causing visual decrease was evaluated using the method of Kaplan-Meier; the Kaplan-Meier method with log-rank test was also used to estimate if the probability of developing pathologic events causing visual decrease during the follow-up is higher in group 1 or 2. $P < 0.05$ was considered as statistically significant.

Results

Baseline

Vitreoretinal conditions of group 1 eyes at baseline were the following: presence of a vitreous liquefaction (VL) was found in 16 of the 47 (34.0%) eyes. A PVD was found in 31 of the (65.9%) 47 eyes. A mild chorioretinal atrophy was present in 33 out of 47 (70.2%) eyes, whereas a posterior staphyloma with severe chorioretinal atrophy was detected in 13 out of 47 (27.6%) eyes.

Chorioretinal atrophy was considered as “mild” when the atrophy was located in the peripapillary area, with or without a moderate loss of retinal pigmented epithelium in the macular area. Chorioretinal atrophy was considered as “severe” in the presence of extensive peripapillary chorioretinal atrophy with, eventually, a localized or more extensive loss of the choroid in the area included between the temporal vascular arcades.

Vitreoretinal conditions of group 2 eyes at baseline were the following: a VL, not associated with PVD or PVS, was found in 3 of the 51 (5.8%) eyes. A PVD was present in 6 of the 51 (11.8%)

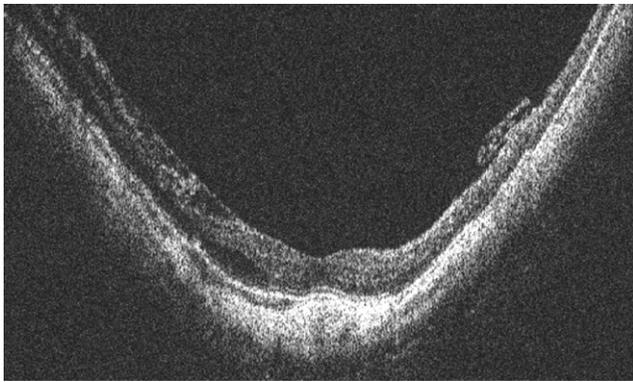


Figure 1. Case # 47, group 2, fellow eye with posterior vitreous schisis and epiretinal membrane; horizontal optical coherence tomography (OCT) scan of the macular region passing through the fixation point. Right = nasal, left = temporal. A thin, posterior epiretinal membrane attached to the inner layer of the macula is evident. A moderate disruption of the neuroretinal layers in the temporal side of the macula can be observed.

eyes. In 2 of the 6 eyes with PVD, baseline OCT examination detected an asymptomatic macular hole (AMH). On the basis of the OCT findings, an AMH was defined as a full-thickness loss of tissue in the region corresponding to the fovea without increase of the retinal thickness, no intraretinal fluid accumulation on the surrounding neuroretinal tissue, and no associated visual symptoms. A PVS was found in 42 of the 51 (82.3%) eyes. A posterior staphyloma with severe chorioretinal atrophy was present on 51 (100%) eyes (Table 1).

A thin, posterior epiretinal membrane associated with PVS was detected by means of OCT scans in 30 of the 51 (58.8%) eyes (Fig 1). In 18 of these 30 eyes, OCT scans revealed a moderate disruption of the neuroretinal layers, associated in some cases with focal areas of more severe disruption.

In 6 of the 51 (11.8%) eyes, there was a PVS with a thin posterior epiretinal membrane associated with a shallow, asymptomatic RD (defined as morphologic retinal changes without visual symptoms) involving the paramacular area, corresponding to the superior (4 eyes) or the inferior (2 eyes) temporal vascular arcades. Visual acuity in these 6 eyes was >0.2 logarithm of minimum angle resolution. A PVS associated with an AMH with or without evidence of epiretinal membrane (EM) was present respectively in 3 (5.9%) and in 2 (3.9%) of the 51 eyes (Table 2).

Follow-up

During follow-up, 2 eyes in group 1 and 9 eyes in group 2 had a significant VA decrease compared with baseline (Tables 3, 4, and 5). In group 1 eyes, 12 of the 15 (80%) eyes with a VL at baseline

Table 2. Further Baseline Vitreoretinal Features of Group 2 Eyes with Posterior Vitreous Schisis

Feature	n (%)
PVS+EM	30 (58.8)
PVS+EM+ARD	6 (11.8)
PVS+EM+AMH	3 (5.9)
PVS+AMH	2 (3.9)

AMH = asymptomatic macular hole; ARD = asymptomatic retinal detachment; EM = epiretinal membrane; PVS = posterior vitreous schisis.

Table 3. Demographic and Visual Acuity Observed in Group 1 and 2 Eyes with a Decrease in Visual Acuity (at 84 Months Follow-Up)

	Group 1	Group 2
Age	43.33±5.13	46.12±4.49*
Visual acuity	0.97±0.23	0.73±0.06†
Myopia	-18.00±5.13	-24.25±3.45†

*P>0.05 group 1 versus group 2 (chi-square test).

†P<0.05 group 1 versus group 2 (chi-square test).

Values are expressed as means ± 1 standard deviation (SD).

developed a PVD during the follow-up in a period of time ranging between 4 and 64 months. Among these 12 eyes with PVD, 3 eyes developed a retinal pathology after the onset of PVD: 1 eye had a RDMH, 1 eye had a superior RD owing to peripheral retinal breaks on lattice degenerative areas, and 1 eye had a peripheral retinal break on the superior quadrants on lattice degenerative areas without RD. Two of the 31 (6.4%) eyes in group 1 with a PVD at baseline developed peripheral retinal breaks at 24 and 30 months of the follow-up; 1 eye had an RD on the superior quadrants originating from the retinal breaks on lattice areas.

In group 2 eyes, 1 of the 3 (33.3%) eyes with VL at baseline developed a PVD at 38 months of the follow-up. Six of the 30 (20%) eyes that at baseline examination had a PVS associated with EM developed an RDMH (from 18 to 80 months of the follow-up). In 1 of the 6 (16.6%) eyes that at baseline had a PVS associated with EM and asymptomatic RD, the RD became symptomatic owing to its extension to the macula at 12 months of follow-up. In 2 of the 3 (66.6%) eyes that at baseline had PVS associated with EM and AMH, the macular hole became symptomatic without evidence of RD at 48 months and at 54 months of follow-up.

The features of patients who developed vitreoretinal changes at the end of the follow-up are summarized in Table 6.

In Figure 2, a Kaplan-Meier plot showing the cumulative probability of VA decrease during the follow-up period for groups 1 and 2 is presented. The risk of pathologic events causing VA decrease in group 2 was significantly higher than in group 1 (P = 0.041; log-rank test = 4.18; df = 1).

Discussion

The present study aims to evaluate anatomic risk factors involved in the onset of retinal complications causing a decrease of VA in the fellow eyes of highly myopic patients operated for RDMH. At baseline, PVD was present in 31 out of 47 (65.9%) fellow eyes of patients with PVD in the

Table 4. Number of Eyes with a Decrease in Visual Acuity of Groups 1 and 2 Reported According to Vitreoretinal Features (at 84 Months Follow-Up)

Feature	Group 1	Group 2
Vitreous liquefaction	1	—
Posterior vitreous detachment	1	—
Posterior vitreous schisis	—	9
Total (n)	2	9*

*P<0.05 group 1 versus group 2 (chi-square test).

Table 5. Vitreoretinal Changes in Relation to Baseline Vitreoretinal Features (at 84 Months Follow-Up)

Case	Baseline		84 Months Follow-up
	Vitreoretinal Features	VA (LogMAR)	VA (LogMAR)
55	VL (group 1)	0.2	0.7
73	PVD (group 1)	0.1	0.8
8	PVS+EM (group 2)	0.1	0.7
87	PVS+EM (group 2)	0.1	0.7
49	PVS+EM (group 2)	0.2	0.8
71	PVS+EM (group 2)	0.2	1
22	PVS+EM (group 2)	0.1	1.1
34	PVS+EM (group 2)	0.1	1.1
62	PVS+EM+ARD (group 2)	0.3	0.8
79	PVS+EM+AMH (group 2)	0.2	1
93	PVS+EM+AMH (group 2)	0.2	1.1
Mean VA ± 1 SD		0.16±0.06	0.89±0.17*

AMH = asymptomatic macular hole; ARD = asymptomatic retinal detachment; EM = epiretinal membrane; LogMAR = logarithm of minimum angle resolution; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; VA = visual acuity; VL = vitreous liquefaction. Values are expressed as means ± 1 standard deviation (SD).

eye operated for RDMH, which were previously included in another study¹⁸ (group 1). Conversely, PVS was present in 42 out of 51 (82.3%) fellow eyes of patients with PVS in the eye operated for RDMH (group 2).

Because group 2 eyes had higher mean values of myopia with respect to group 1 eyes (−24.33±3.77 and −16.19±3.54 −D respectively), the data could suggest that patients with

Table 6. Baseline and 84 Months Follow-up Visual Acuity in Patients with a Decrease in Visual Acuity

Baseline Vitreoretinal Features	Group	
	Group 1	Group 2
VL	12/15 PVD (80%) (1 RD with MH 1 superior RD from PRB 1 PRB on lattice areas)	1/3 PVD (33.3%)
PVD	2 PRB (6.4%) (1 RD from lattice areas)	—
PVS+EM	—	6/30 (20.0%) RD with MH
PVS+EM+ARD	—	1/6 (16.6%) extension of RD to the macula
PVS+EM+AMH	—	2/3 (66.6%) SMH

AMH = asymptomatic macular hole; ARD = asymptomatic retinal detachment; EM = epiretinal membrane; MH=macular hole; PRB = peripheral retinal breaks; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; RD = retinal detachment; SMH = symptomatic macular hole; VL = vitreous liquefaction.

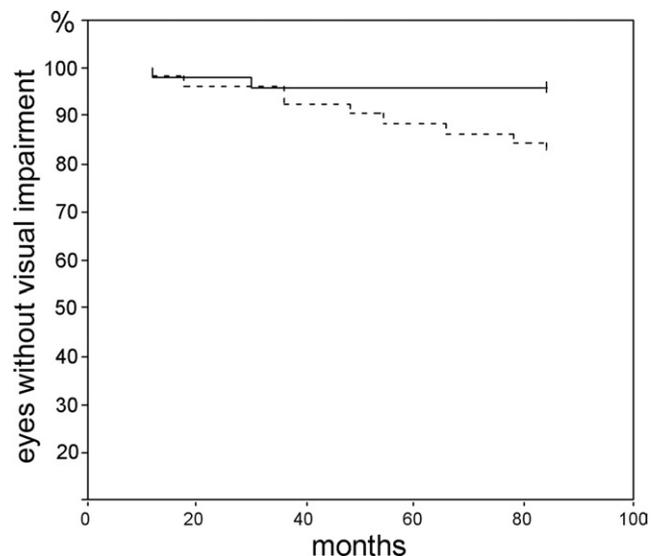


Figure 2. Kaplan-Meier plot shows the cumulative probability of visual impairment during the follow-up period for group 1 (dotted line) and group 2 (dashed line). The risk of pathologic events causing visual decrease is different if the eyes are divided according to the vitreous features of the fellow eye; the risk of visual decrease is significantly higher in group 2 than in group 1 (P = 0.041; log-rank test = 4.18; df = 1).

higher degree of myopia may show similar vitreous characteristics (PVS) in both eyes.

At the end of follow-up, the incidence of pathologic events causing a decrease in VA between the 2 groups was significantly different: group 1, 2 of 47 (4.2%) eyes; group 2, 9 of 51 (17.6%) eyes. These data seem to be related to the different anatomic features among the 2 groups of studied eyes. The presence of a posterior EM, almost a constant finding in PVS eyes (39 of 42 group 2 eyes), had an important role in these studied eyes for the development of retinal complications (group 2, 9 of 9 eyes with visual decrease). It is supposed that an EM can cause tractional forces on the underlying macular tissue that determine structural changes and the development of complications with a decrease in VA. Spaide¹⁴ reported that the presence of a posterior precortical vitreous pocket (PPVP) might be a risk factor for the development of a macular hole in the emmetropic fellow eye of those patients who had a macular hole in the contralateral eye. We believe that the PPVP described by Spaide could represent an interesting model to explain why in this study the presence of a PVS seems to be a risk factor for the development of posterior retinal complications in high myopic eyes. The PVS could share similar anatomic features with the PPVP (i.e., in both cases a consistent region of premacular liquefied vitreous with a residual layer of posterior vitreous attached to the macula is documented); therefore, we might hypothesize that a PVS could be considered as an enlarged PPVP, in relationship with the higher axial elongation of the posterior pole in high myopic eyes compared with emmetropic eyes. However, in high myopic eyes, additional factors, represented by the chorioretinal dysfunction and the presence of a severe posterior staphyloma, may also be relevant for the onset of posterior retinal complications.

In this study, RDMH developed in 7 of the 98 total fellow eyes (1 of 47 group 1 eyes, 6 of 51 group 2 eyes) with a total incidence of RDMH cases of 7.1% at the end of the 7 years of follow-up. Oie and Emi³ reported the incidence of developing RDMH in high myopic fellow eyes to be 12.8% at the end of 5 years of follow-up, pointing out that the higher the degree of myopia, the higher the incidence of RDMH. Similarly, in our studied eyes the incidence of RDMH was higher in group 2 eyes than in group 1 eyes; group 2 eyes had a higher degree of myopia and more anatomic predisposing factors (PVS, EM, posterior staphyloma, and chorioretinal atrophy) compared with group 1 eyes. Even if the incidence of RDMH in the fellow eyes of our study compared to the incidence reported in other studies seems to be lower, it should be noted that in our series additional pathologic changes affecting the posterior retina developed during the follow-up (4 out of the 98 studied eyes), reaching an overall 11.2% incidence of retinal complications causing a decrease of the VA. We also observed that some eyes at baseline already had an asymptomatic retinal pathology such as asymptomatic RD or AMH. In these eyes, the subsequent decrease of the VA was related to further modifications that occurred at the vitreoretinal interface of the macular region as shown by OCT examination. These observations are consistent with those previously reported in another study, in which the evolution from an AMH to a symptomatic macular hole was observed in eyes with PVS, EM, and higher degree of myopia.²²

In conclusion, our data support the hypothesis that if the degree of myopia in the 2 eyes is similar, both eyes of RDMH patients have a similar vitreoretinal structure, particularly those eyes with higher degree of myopia. Anatomic features, including PVS, EM, severe posterior staphyloma, and severe chorioretinal atrophy, seem to represent risk factors not only for the development of RDMH in the fellow eye, but also for the development of other retinal pathologies that in some cases can be already present at first examination, just asymptomatic. A careful evaluation of the fellow eyes of RDMH patient, including OCT assessment of the macular region, allows an early detection of these anatomic features.

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