

Prevalence of Asymptomatic Macular Holes in Highly Myopic Eyes

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Objective: To evaluate the presence of macular holes (MHs) in very highly myopic eyes in the absence of visual symptoms.

Design: Retrospective case series.

Participants: Three hundred eighty-three eyes from 383 patients (mean age, 51.70 ± 12.73 years) affected by very high myopia (between -14 and -32 diopters [D]), with no visual disturbance such as metamorphopsia and with visual acuity (VA) of >0.2 logarithm of the minimum angle of resolution, were considered in the study. They were evaluated at baseline and every 6 months during a mean total period of 30.2 ± 0.5 months (range, 28.3–32.1).

Methods: Evaluation of macular morphology by optical coherence tomography (OCT).

Main Outcome Measures: Changes in OCT macular morphology ascribed to the presence of an MH.

Results: Macular holes were detected by OCT in 24 of 383 (6.26%) myopic eyes. These MHs were defined as asymptomatic (AMHs). The presence of AMHs was more prevalent (although not significantly when compared with the overall study population) in myopic patients younger than 50 years and with concomitant myopia of >-20 D. Posterior vitreous schisis was the most frequent vitreous modification associated with AMH (18/24 eyes [75.0%]). During follow-up, a decrease in VA was observed in 5 of 24 (20.8%) AMH eyes due to the enlargement of the lesion or to posterior retinal detachment, leading to the development of a symptomatic MH. Greater degree of myopia and younger age were associated with the evolution of the macular disease, consisting of an enlargement of the MH and/or the onset of a macular detachment.

Conclusions: A small percentage (6.26%) of highly myopic eyes may develop an MH in the absence of visual symptoms. The absence of symptoms could be related to the localization of the hole in a juxtafoveal area. Its evolution in a symptomatic MH seems to be related to the presence of epiretinal tissue, younger age, and the degree of myopia. *Ophthalmology* 2005;112:2103–2109 © 2005 by the American Academy of Ophthalmology.

The presence of a macular hole (MH), associated or not associated with retinal detachment (RD), is a not uncommon complication that can be observed in highly myopic eyes.¹ The MH may induce loss of central vision and reduction in visual acuity (VA) that can be in part recovered after surgical treatment performed by pars plana vitrectomy (PPV) or by the posterior episcleral buckling procedure. In a previous study by our group, we reported that the posterior episcleral buckling procedure may give better postoperative anatomic and functional results than PPV.²

Sometimes, in the absence of RD, the biomicroscopic diagnosis of MH in highly myopic eyes is difficult. This could be due to lack of contrast between the MH and the

retinal pigment epithelium (RPE), especially in the presence of a deep posterior staphyloma and severe chorioretinal atrophy.³ The presence of an MH may be inferred in the presence of clinical symptoms such as metamorphopsia or reduced central VA.⁴

Optical coherence tomography (OCT), which allows in vivo examination of macular morphology, is an objective method that provides useful information regarding macular characteristics and relative morphological changes in the presence of MHs.^{5–7}

In our preliminary study, performed in 48 highly myopic eyes without visual impairment, OCT examination revealed, in a small percentage of these eyes, the presence of changes in macular morphology. These changes had characteristics similar to those observed in MHs of myopic eyes with reduced VA. We considered these findings interesting, and because visual functional impairments were not detected in the myopic eyes studied, they were defined as asymptomatic MHs (AMHs) (*Invest Ophthalmol Vis Sci* 39[4]:S107, 1998).

Thus, the aim of our work is to evaluate the presence of AMHs by OCT assessment in a large number of patients affected by different degrees of high myopia, but in the absence of visual symptoms.

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Table 1. Demographic and Clinical Characteristics of Enrolled Myopic Eyes

	Degree of Myopia			Total
	Group A (-14 to -19 D)	Group B (-20 to -25 D)	Group C (-26 to -32 D)	
Men (n)	46	53	53	152
Women (n)	71	74	86	231
Total (n)	117	127	139	383
Age (yrs) (mean \pm 1 SD)	52.79 \pm 12.95	52.01 \pm 12.38	50.50 \pm 12.84	51.70 \pm 12.73
RE (D) (mean \pm 1 SD)	-16.5 \pm 1.67	-22.59 \pm 1.62	-29.01 \pm 1.94	-23.07 \pm 5.40
AL (mm) (mean \pm 1 SD)	29.91 \pm 0.55	31.91 \pm 0.54	34.03 \pm 0.64	32.07 \pm 1.78
logMAR VA (mean \pm 1 SD)	0.11 \pm 0.08	0.09 \pm 0.08	0.12 \pm 0.08	0.11 \pm 0.08
Vitreoretinal findings				
SL	14	9	1	24
PVD	99	68	79	246
PVS	4	50	59	113

AL = axial length; D = diopters; logMAR = logarithm of the minimum angle of resolution; n = no. of eyes; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; RE = refractive error; SD = standard deviation; SL = slight liquefaction; VA = visual acuity.

Materials and Methods

Patients

From January 1997 to September 2002, we examined a consecutive series of 647 patients in which at least one eye had a myopia between 14 and 32 negative diopters (D).

Inclusion criteria were presence of myopia of more than -14 D (axial length >29.08 mm, evaluated by B-scan ultrasonography [see below]), absence of moderate to dense lens opacities, implanted intraocular lens, glaucoma or ocular hypertension, history of intraocular inflammation such as anterior or posterior uveitis, multifocal choroiditis, history of RD or laser treatment for peripheral retinal diseases, diabetes, connective tissue diseases, history of ocular trauma or optic neuropathy, and other systemic or neurological diseases. We also excluded patients with myopic macular juxtafoveal or subfoveal choroidal neovascularization.

A very important inclusion criterion was the presence of VA of >0.2 logarithm of the minimum angle of resolution (logMAR) (see method below). In addition, the absence of other visual symptoms (i.e., metamorphopsia) was required.

On the basis of the above-mentioned systemic and ocular criteria, we selected 383 patients (mean age, 51.70 \pm 12.73 years). Because our study aimed to evaluate the prevalence of changes in macular morphology in a large cohort of patients (in which age could play a role [see below]) and not in a cohort of eyes, when both eyes from the same patient were eligible for inclusion in the study we randomly considered only one eye (this condition was present in 79 patients only). Therefore, 383 eyes (from 383 patients [mean refractive error, -23.07 \pm 5.40 D]) were considered for the study. These eyes were defined as asymptomatic myopic eyes (AMEs). The remaining eyes were excluded.

On the basis of the degree of myopia, AMEs were divided into

3 groups: group A (117 eyes), degree of myopia between -14 and -19 D (axial length between 29.08 and 30.73 mm); group B (127 eyes), degree of myopia between -20 and -25 D (axial length between 30.81 and 32.95 mm); and group C (139 eyes), degree of myopia between -26 and -32 D (axial length between 33.04 and 35.02 mm). Demographic and clinical characteristics of the enrolled AMEs are presented in Tables 1 and 2.

Examinations

All enrolled AMEs underwent the following examinations:

- Indirect ophthalmoscopy and slit-lamp biomicroscopy using a +90-D no-contact lens (Volk Optical, Mentor, OH) after pupillary dilation using tropicamide 1%. Biomicroscopic signs of an MH were defined as the presence of a well-defined round foveal area surrounded by a ring-shaped lighter area, slightly elevated with respect to the perifoveal retina.
- A-scan and B-scan ultrasonography using a 10-megahertz probe (Humphrey Instruments, San Leandro, CA).
- Optical coherence tomography examination was performed with commercially available instrumentation (OCT1 and OCT3 Stratus, Carl Zeiss Ophthalmic Systems, Inc., Humphrey Division, Dublin, CA). Until May 2002, OCT1 was used for the evaluations performed at baseline conditions and during the follow-up period (see below). After May 2002, OCT3 was used for the evaluations performed at baseline conditions and during the follow-up period (see below). OCT1 was always utilized in those myopic eyes in which the baseline evaluation was performed before May 2002, and successive evaluations were performed after May 2002. A superluminescent diode (820 nm) was used to scan and

Table 2. Age Distribution of Patients with Enrolled Myopic Eyes

	30-40 yrs/ Total	41-50 yrs/ Total	51-60 yrs/ Total	>61 yrs/Total	Total
n (%)	102/383 (26.6)	96/383 (25.1)*	87/383 (22.7)*	98/383 (25.6)*	383/383 (100)

n = no. of eyes.

* $P > 0.05$, χ^2 with respect to the 30-40 years group.

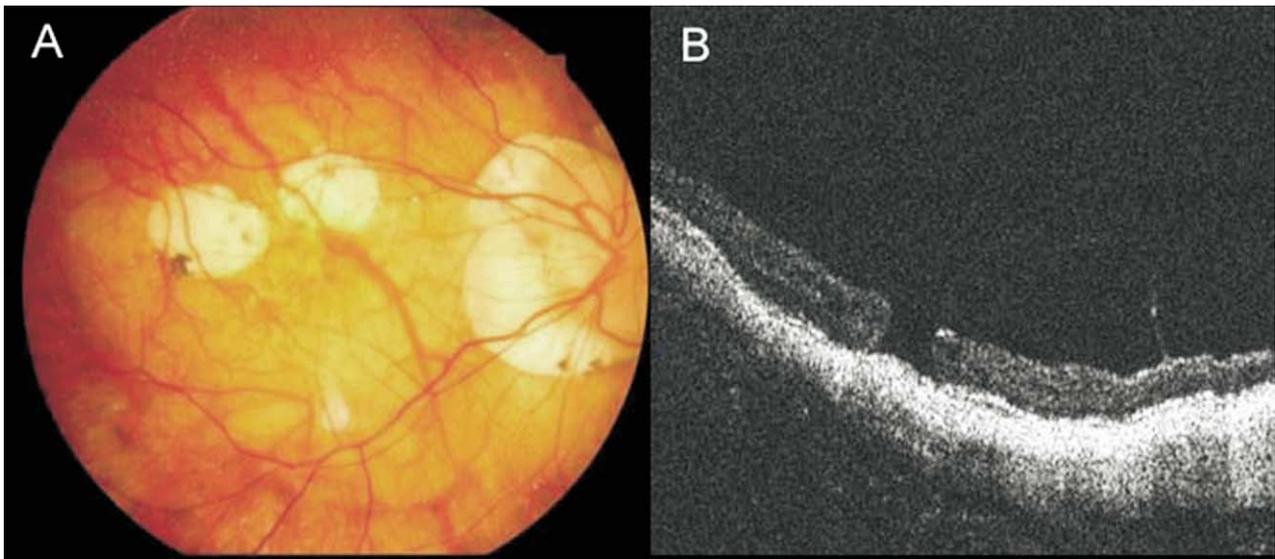


Figure 1. A, Example of fundus photography of one asymptomatic macular hole. The lesion is not visible on the background of the hypopigmented posterior pole. B, Optical coherence tomography scan performed in the same eye. The diameter of the hole is relatively small ($<350\ \mu\text{m}$), and the edges of the lesion are flat, with a very slight accumulation of intraretinal fluid in the perilesional area and no subretinal fluid cuff.

analyze the macular area. After pupillary dilation, OCT images of the macular region were obtained with horizontal and vertical scans, with a length of 6.5 mm. The axial resolution was $<10\ \mu\text{m}$, and the transverse resolution was $20\ \mu\text{m}$. OCT1 and OCT3 instruments are provided with the same software, which allows us to measure the specific characteristics of the lesion such as mean diameter and thickness of the perilesional retina, which were measured using a pair of cursors directly on the scanned image. The values of hole width and retinal thickness at the edge of the lesion were measured on the horizontal and vertical meridians and averaged.

- Best-corrected VA was assessed by the Modified Early Treatment Diabetic Retinopathy Study Table (Lighthouse, Low Vision Products, Long Island City, NY); VA was expressed in logMAR values obtained at a distance of 4, 2, 1, and 0.5 m. Visual acuity was consistent with the degree of myopia of each single patient and the associated presence of posterior staphyloma and chorioretinal atrophy.

All enrolled AMEs were further examined every 6 months (mean, 6.1 ± 0.25 ; range, 5.3–6.7) for a total period of approximately 30 months (mean, 30.2 ± 0.5 ; range, 28.3–32.1).

Statistical Analysis

Values are expressed as mean \pm 1 standard deviation. The differences observed between the different groups divided according to age or degree of myopia were evaluated by 2×2 tables and chi-square test, assuming that each eye of each patient may independently contribute to the observed incidence results. A P value of <0.05 was adopted as statistically significant.

In eyes with AMHs that developed visual symptoms during the follow-up period, changes in VA and in lesion diameter observed at the end of follow-up with respect to baseline values were analyzed by 1-way analysis of variance. In those eyes, the Pearson correlation was used to correlate the changes in diameter of the lesion with the changes in logMAR VA. Morphometric data from the lesion (greatest linear dimension) underwent logarithmic transformation to approximate a normal distribution better. In

both analyses, a P value of <0.01 was considered statistically significant.

Results

On the basis of the OCT findings, an AMH was defined as a full-thickness apparent absence of tissue in the foveal region, surrounded by a region of slightly reduced optical reflectivity and slightly increased retinal thickness due to intraretinal fluid accumulation. These morphological retinal changes were present in the concomitant absence of visual symptoms. An example of an AMH observed by OCT examination with the relative biomicroscopic evaluation is reported in Figure 1.

The OCT examination revealed the presence of an AMH in 24 of 383 (6.26%) eyes, belonging to eyes with myopia between -19 and -29 D. Biomicroscopic findings of an AMH were present in only 6 of these 24 eyes.

The morphology of the lesion differed slightly from that of classic idiopathic MHs: the diameter of the hole was relatively small ($<350\ \mu\text{m}$; Table 3), and the edges of the lesion were flat, with a very slight accumulation of intraretinal fluid in the perilesional area and no subretinal fluid cuff.

The characteristics of AMH eyes are reported in Table 3.

Table 4 shows the presence of AMHs in relation to the degree of myopia. The presence of AMHs seems to be more frequent in AMEs with a higher degree of myopia. The 16.47% prevalence of AMEs observed in eyes with myopia between -20 and -25 D and the 16.40% prevalence of AMEs observed in eyes with myopia between -26 and -32 D differed significantly ($P < 0.05$) with respect to eyes with myopia between -14 and -19 D (3.47%).

Table 5 shows the distribution of AMHs in relation to patient age and degree of myopia. Analyzing this distribution, we observed a greater presence of AMHs in a range between 30 and 40 years (41.66% of AMHs with respect to the total number of AMHs) and between 41 and 50 years (25.00% of AMHs with respect to the total number of AMHs), although this difference did not reach statistical significance. In the younger patients (30–40 years), the greater prevalence of AMH eyes was detected for a range of myopia between -26 and -32 D, whereas in AMH eyes

Table 3. Characteristics of Eyes with an Asymptomatic Macular Hole

	Degree of Myopia			Total
	Group A (-14 to -19 D)	Group B (-20 to -25 D)	Group C (-26 to -32 D)	
n	2	11	11	24
RE (D) (mean \pm 1 SD)	-19.00 \pm 0.00	-23.54 \pm 1.75	-27.27 \pm 11.19	-28.88 \pm 2.92
AL (mm) (mean \pm 1 SD)	30.73 \pm 0.00	32.23 \pm 0.58	33.46 \pm 0.39	32.66 \pm 0.96
logMAR (VA) (mean \pm 1 SD)	0.20 \pm 0.00	0.04 \pm 0.08	0.12 \pm 0.09	0.09 \pm 0.09
DL (μ m) (mean \pm 1 SD)	309.50 \pm 20.50	313.18 \pm 56.00	323.64 \pm 50.65	317.66 \pm 53.35
PRT (μ m) (mean \pm 1 SD)	217.00 \pm 1.41	213.54 \pm 25.01	224.55 \pm 27.94	218.88 \pm 25.31

AL = axial length; D = diopters; DL = diameter of the lesion; logMAR = logarithm of the minimum angle of resolution; n = no. of eyes; PRT = perilesional retinal thickness; RE = refractive error; SD = standard deviation; VA = visual acuity.

with age between 41 and 50 years, the greater prevalence of AMHs was found in those eyes with refractive error between -20 and -25 D. Therefore, the presence of AMHs was more prevalent in myopic patients younger than 50 and with concomitant myopia of $>$ -20 D. Nevertheless, this difference is not significant ($P = 0.11$) when compared with the overall study population.

The characteristics of vitreous changes observed in eyes with AMHs are reported in Table 6. Vitreous changes were found in all eyes with AMHs. The most frequent vitreous modifications consisted of posterior vitreous schisis (PVS), which was present in 18 of 24 (75.0%) AMH eyes, whereas a posterior vitreous detachment (PVD) was present in only 6 of 24 (25%) AMH eyes.

In all AMH eyes, there were a posterior staphyloma, of variable degree, and a rarefaction of the RPE. A thin layer of epiretinal tissue was observed by OCT in 15 of 18 (83.33%) AMH eyes with PVS and in only 1 of the 6 (16.66%) AMH eyes with PVD. The epiretinal tissue was detected in 7 AMH eyes of patients younger than 50 years and in 9 AMH eyes of patients older than 50.

Follow-up

During the entire follow-up period of 30.2 \pm 0.5 months, VA in 19 of 24 (79.17%) AMH eyes remained stable.

In 5 of 24 (20.83%) AMH eyes, we observed a significant decrease in VA (mean baseline VA values in those 5 AMH eyes: 0.1 \pm 0.1; mean VA values in those 5 AMH eyes after 30 months: 0.84 \pm 0.09; $P < 0.001$). Therefore, in these 5 eyes, the MH became symptomatic and was thus defined as a symptomatic MH (SMH).

In 3 of the 5 SMH eyes, the MH became symptomatic without any evidence of RD. During the follow-up period, in those AMH eyes (AMH#3, AMH#192, and AMH#243) the reduction in VA was detected after 6, 30, and 18 months, respectively.

An example of OCT findings observed in these SMH eyes is reported in Figure 2A, B. These eyes showed a significant increase in diameter of the lesion with respect to baseline values (baseline, 335.00 \pm 42.72 μ m; after follow-up, 610.00 \pm 43.59 μ m; $P < 0.001$). The morphology of the edge of the hole, however, was similar to baseline. No significant correlation ($P > 0.05$) was found between the diameter of the lesion and decreased VA.

The remaining 2 SMH eyes developed a shallow RD localized in the area of the staphyloma. During the follow-up period, in those 2 AMH eyes (AMH#34 and AMH#312) the reduction in VA was detected after 6 and 24 months, respectively. An example of OCT findings observed in these SMH eyes is shown in Figure 2C, D.

Table 7 reports the distribution of eyes with SMHs in relation to age and degree of myopia. We observed that the evolution from AMH to SMH was detectable only in those eyes with the highest degree of myopia (between -26 and -32 D) and concomitant lower age (between 30 and 50 years). The highest prevalence (60% of eyes with reduced VA) was found in the age range between 31 and 40 and with refractive error between -26 and -32 D. In all 5 SMH eyes, the vitreous modifications observed during the first examination consisted of PVS, and the presence of a thin layer of epiretinal tissue was detected during OCT examination.

Discussion

The aim of our work was to evaluate, by OCT examination, the presence of MHs in a population of highly myopic eyes without visual symptoms.

In our series of 383 myopic eyes, we found the presence of an AMH in 24 (6.26%) eyes, and all had good vision. The

Table 4. Presence of a Macular hole (MH) with Respect to All Enrolled Asymptomatic Myopic Eyes (AMEs) and Relative Vitreoretinal Findings

	Group A (-14 to -19 D) (n/Total AMEs)	Group B (-20 to -25 D) (n/Total AMEs)	Group C (-26 to -32 D) (n/Total AMEs)	Total (n/Total AMEs)
MH	2/117 (3.47%)	11/127 (16.47%)*	11/139 (16.40%)*	24/383 (6.26%)
SL	0/14 (0.00%)	0/9 (0.00%)	0/1 (0.00%)	0/24 (0.00%)
PVD	1/99 (1.01%)	3/68 (4.41%)	2/79 (2.52%)	6/246 (2.43%)
PVS	1/4 (25.00%)	8/50 (16.00%)	9/59 (15.25%)	18/113 (15.92%)

D = diopters; n = no. of eyes; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; SL = slight liquefaction.

* $P < 0.05$, χ^2 with respect to group A eyes.

Table 5. Distribution of Presence of an Asymptomatic Macular Hole (AMH) in Relation to Age and Degree of Myopia

	Group A (-14 to -19 D) (n/Total AMH Eyes)	Group B (-20 to -25 D) (n/Total AMH Eyes)	Group C (-26 to -32 D) (n/Total AMH Eyes)	Total (n/Total AMH Eyes)
30-40 yrs	1/24 (4.16%)	3/24 (12.50%)	6/24 (25.00%)	10/24 (41.66%)
41-50 yrs	0/24 (0.00%)*	4/24 (16.66%)*	2/24 (8.33%)*	6/24 (25.00%)*
51-60 yrs	0/24 (0.00%)*	2/24 (8.33%)*	2/24 (8.33%)*	4/24 (16.66%)*
≥61 yrs	1/24 (4.16%)*	2/24 (8.33%)*	1/24 (4.16%)*	4/24 (16.66%)*
Total	2/24 (8.33%)	11/24 (45.83%) [†]	11/24 (45.83%) [†]	24/24 (100%)

D = diopters; n = no. of eyes.
 * $P > 0.05$, χ^2 with respect to the group of AMH eyes between 30 and 40 yrs.
[†] $P < 0.05$, χ^2 with respect to group A.

presence of an AMH was more prevalent in eyes with the highest degree of myopia and with a pronounced posterior staphyloma. In our asymptomatic myopic eyes in which OCT revealed the presence of an MH, we observed peculiar characteristics of the lesion: a small MH diameter (<350 μm); in all cases, flat lesion edges, without the perilesional swelling that is generally associated with an MH; and no detectable fluid cuff. The absence of perilesional swelling associated with the small diameter of the lesion could be considered as typical in our AMHs; in idiopathic MHs, the absence of perilesional swelling may be observed only in a longstanding MH, but in these cases it is associated with a very large diameter of the lesion.⁸

The pathogenesis of MHs in eyes with high myopia has been widely investigated, but it is still not completely understood; axial elongation of the myopic eye, posterior staphyloma, chorioretinal atrophy, and vitreous modifications such as PVD and PVS causing anteroposterior or tangential vitreous tractions have been supposed to be causative factors.⁹⁻¹³

In our asymptomatic myopic eyes, vitreous modifications were present in all patients with AMHs, with a large prevalence of PVS (18/24 eyes), and epiretinal tissue adhered to the macula in 16 of 24 eyes, as documented by OCT. All this leads us to suppose a possible role of vitreous modifications in the pathogenesis of AMH. However, the finding that in 8 eyes with AMHs there was absence of epiretinal tissue leads us to suppose that other retinal defects, probably

related to the presence of a posterior staphyloma, may also play an important role in the development of AMHs.

To explain the formation of an AMH, a sequence of events is hypothesized. First, the retina thins near the fovea¹⁴; second, the inner retinal surface weakens and degenerates, followed by a break in the inner limiting membrane; third, there is an inappropriate healing process leading to epiretinal cell proliferation, with an increase of the rigidity of the inner limiting membrane (migration of Müller cells, found in the epiretinal operculum)¹⁵; and fourth, the contact of liquefied vitreous with the inner and external retinal layers causes swelling of the perilesional retina and enlargement of the edges of the MH.¹⁶ In highly myopic eyes, the frequent presence of epiretinal tissue, liquefied vitreous, and reduced function of the RPE may facilitate the onset of these kinds of lesions.

Our most interesting finding may be the presence of an MH in the absence of visual symptoms. This finding is difficult to explain, and some hypotheses may be offered. A juxtafoveal origin of the lesion may be supposed, justifying the presence of nonimpaired VA. The presence of a posterior staphyloma associated with vitreous modifications (18 PVS eyes, 6 PVD eyes) in AMH eyes could be another factor inducing the development of MHs in a juxtafoveal area. All this can be only supposed, because OCT examination in AMH eyes does not allow us to obtain morphological information related to the foveal location.

During the 30 months of follow-up, we observed a wors-

Table 6. Distribution of Vitreoretinal Changes in Asymptomatic Macular Hole (AMH) Eyes with Respect to the Total Number of AMH Eyes

	Group A (-14 to -19 D) (n/Total AMH Eyes)	Group B (-20 to -25 D) (n/Total AMH Eyes)	Group C (-26 to -32 D) (n/Total AMH Eyes)	Total (n/Total AMH Eyes)
PVD	1/2 (50.00%)	3/11 (27.27%)	2/11 (18.18%)	6/24 (25.00%)
PVS	1/2 (50.00%)*	8/11 (72.72%)*	9/11 (81.82%)*	18/24 (75.00%) [†]
SL	0/2 (0.00%)*	0/11 (0.00%)*	0/11 (0.00%)*	0/24 (0.00%) [†]
Total	2/2 (100%)	11/11 (100%)	11/11 (100%)	24/24 (100%)

D = diopters; n = no. of eyes; PVD = posterior vitreous detachment; PVS = posterior vitreous schisis; SL = slight liquefaction.
 * $P > 0.05$, χ^2 with respect to PVD.
[†] $P < 0.05$, χ^2 with respect to PVD.

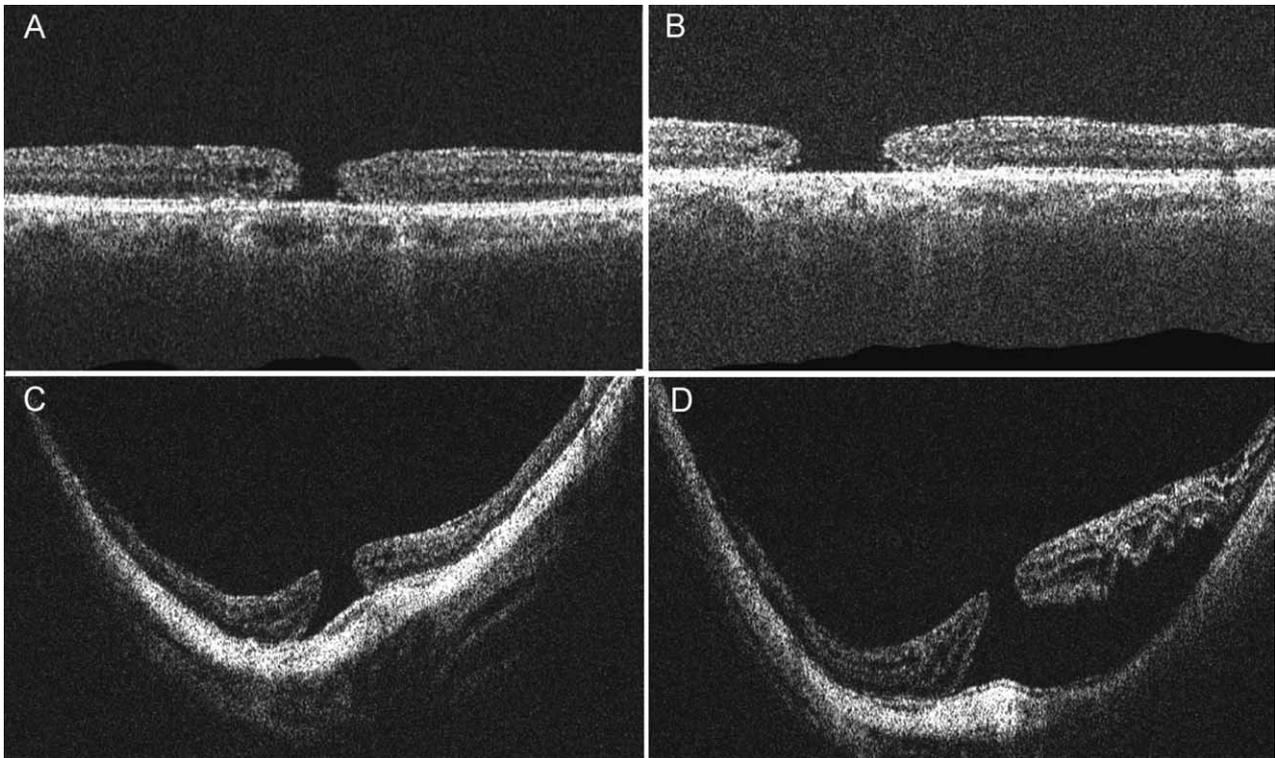


Figure 2. Example of an optical coherence tomography (OCT) scan in 2 evolving asymptomatic macular holes (AMHs). **A**, An OCT scan of AMH eye #243 at baseline examination. The diameter of the lesion is 280 μm , and visual acuity (VA) is 0.2 logarithm of the minimum angle of resolution (logMAR). **B**, An OCT scan of AMH#243 after 18 months. The hole has enlarged, reaching 660 μm , and has probably involved the fovea; VA has dropped to 1.0 logMAR. A thin layer of epiretinal tissue is detectable on the left and right sides of the lesion. In this eye, the development of a retinal detachment (RD) was not detected. **C**, An OCT scan of AMH#312 at baseline examination. The diameter of the lesion is 325 μm , and VA is 0.2 logMAR. **D**, An OCT scan of AMH#312 after 6 months. The hole has enlarged, reaching 540 μm , and has probably involved the fovea; VA has dropped to 1.0 logMAR. The perilesional area is thick and optically hypodense. A thin layer of epiretinal tissue is detectable on the right side of the lesion. In this eye, a posterior RD is present.

ening of VA in 5 of 24 AMH eyes. This visual impairment, associated with an enlargement of the MH diameter, was observed in the presence of the following concomitant conditions: presence of epiretinal tissue during the baseline OCT evaluation, highest degree of myopia (between -26 and -32 D), and younger age (<50 years).

Because epiretinal tissue was found in 11 other AMH

eyes (2 eyes from 2 patients younger than 50 years and 9 eyes from 9 patients older than 51) in which no changes in VA were observed during the entire follow-up period, it is possible that the presence of epiretinal tissue may play an important, but not exclusive, role in the development of the reduction in VA in the presence of an AMH.

We hypothesize that in younger patients with AMH eyes

Table 7. Distribution of AMH Eyes That Developed a Reduction in Visual Acuity during the 30 Months' Follow-up with Transformation to a Symptomatic MH (SMH), in Relation to Age and Degree of Myopia

SMH	Group A (-14 to -19 D) (n/Total SMH Eyes)	Group B (-20 to -25 D) (n/Total SMH Eyes)	Group C (-26 to -32 D) (n/Total SMH Eyes)	Total (n/Total SMH Eyes)
30–40 yrs	0/5 (0.00%)	0/5 (0.00%)	3/5 (60.00%)	3/5 (60.00%)
41–50 yrs	0/5 (0.00%)*	0/5 (0.00%)*	2/5 (40.00%)*	2/5 (40.00%)*
51–60 yrs	0/5 (0.00%)*	0/5 (0.00%)*	0/5 (0.00%)*	0/5 (0.00%)*
≥ 61 yrs	0/5 (0.00%)*	0/5 (0.00%)*	0/5 (0.00%)*	0/5 (0.00%)*
Total	0/5 (0.00%)	0/5 (0.00%) [†]	5/5 (100%)*	5/5 (100%)

D = diopters; n = no. of eyes.

* $P > 0.05$, χ^2 with respect to the group of 30–40-year AMH eyes.

[†] $P > 0.05$, χ^2 with respect to group A.

* $P < 0.05$, χ^2 with respect to group A.

the tractional action induced by the epiretinal tissue may induce an enlargement of the hole. A possible explanation is the fact that the vitreous body in highly myopic patients is affected by a progressive degenerative process at a younger age with respect to emmetropic eyes^{1,9}; the vitreoretinal adhesion between the posterior vitreous cortex and the inner retina is also stronger in younger patients.¹⁷ Therefore, in these subjects the traction of epiretinal vitreous cortex adherent to the foveal area may enlarge small MHs and cause the development of a shallow RD localized at the posterior pole.

In conclusion, our results suggest that a small percentage (about 6%) of highly myopic eyes may demonstrate an MH in the absence of visual symptoms. This condition is more prevalent in myopic eyes of patients younger than 50 years and with a concomitant degree of myopia of > -20 D. The absence of symptoms could be related to a localization of the hole in a juxtafoveal area. The evolution from AMH to SMH seems to be related to the presence of epiretinal tissue, younger age, and the highest degree of myopia.

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