Foveal Cysts

A Premacular Hole Condition Associated With Vitreous Traction

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Objective: To define the clinical findings, cause, and outcome of patients with foveal cysts due to vitreous traction.

Methods: Follow-up of 18 patients with foveal cysts and no posterior vitreous detachment (PVD). Changes were documented in visual acuity, the appearance of the fovea, or the development of a macular hole or PVD. We studied 8 eyes using the retinal thickness analyzer.

Results: On follow-up, 9 of 23 eyes did not develop a PVD and still had a foveal cyst; 8 of 23 developed a full-thickness macular hole; 4 of 23 developed a PVD with resolution of the cyst; and 2 eyes underwent vitrectomy

for the cyst before a full-thickness hole developed. Analysis with the retinal thickness analyzer showed splitting within the middle retinal layers and in some cases unroofing or absent inner retinal layers in the center of the cyst.

Conclusions: Foveal cysts are caused by vitreous traction. These eyes may remain stable, develop full-thickness holes, or develop a PVD with resolution of the cystic changes. A foveal cyst seems to be a common finding in patients with foveal traction from a variety of mechanisms.

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hole. He believes that vitreous traction causes a localized detachment of the fovea that is seen as a yellow spot. A dehiscence of the fovea then occurs that is usually located in the center of the umbo. The dehiscence causes centrifugal displacement of the foveolar retina that includes the xanthophyll which is seen as an enlarging yellow ring. The dehiscence in the fovea may be hidden initially, however, because of contraction and opacity of the prefoveolar vitreous cortex. Eventually the condensed cortical vitreous separates slightly from the retina or thin areas develop in the cortical vitreous allowing for visualization of the underlying macular hole. Usually within months of the initial symptoms and appearance of the yellow ring, the vitreous either detaches from the fovea and the yellow ring resolves with good vision or a readily visible full-thickness macular hole with surrounding subretinal fluid develops.

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classification of the development of a macular

A growing number of reports indicate that not all patients with foveal changes caused by vitreous traction follow the Gass classification. Most of the authors have described such foveas as appearing "cystic." Bronstein et al² described a group of patients with a macular hole in one eye and a macular cyst in the fellow eye. McDonnell et al³ documented 30 eyes with macular cysts within a larger report on macular holes. Both of these reports, however, were published prior to the Gass classification on the stages of a macular hole. Therefore, some of the eyes these authors described as having cysts may actually have had stage 1 or 2 macular holes according to Gass. Guyer et al4 "observed premacular hole lesions without yellow dots or halos" that were purely cystic and did not seem to fit into the Gass classification and thus were termed stage 1c lesions. Guyer et al⁵ also described the histopathologic features of 7 eyes with macular cysts. Acosta et al6 used the scanning laser ophthalmoscope to test fixation and central fields in patients with macular holes and also in patients who they believed had macular cysts. They found dense scotomas in eyes with macular holes but not in most eyes with macular cysts. Hee et al⁷ demonstrated cysts in the fovea using optical coherence tomography. Kishi et al,⁸ after imaging patients with various stages of macular holes using the helium-neon laser, concluded that

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From the Department of

PATIENTS AND METHODS

PATIENTS

Patients were included if they were symptomatic; on biomicroscopic examination were found to have striae that were usually radial and appeared to be located in the vitreous cortex overlying the fovea or perhaps in the inner retina along with outer retinal thinning or schisis; had no posterior vitreous detachment (PVD); and were followed up by one of the senior authors (J.C.F. or H.C.B.). The absence of a vitreous detachment was confirmed by echography or by peeling of the posterior vitreous during vitrectomy. Foveal cysts can result from a variety of mechanisms of traction on the retina that include epiretinal membranes and vitreomacular traction syndrome. In an attempt to report on a homogeneous group of patients, we excluded those patients with any areas of vitreous detachment on examination or echography, or visible epiretinal membranes or traction lines located outside the fovea that could be due to transparent epiretinal membranes.

Our criteria for inclusion into the study were developed as we were seeing these patients. About half of the data were analyzed retrospectively and half collected and analyzed prospectively. Eighteen patients (23 eyes) met our criteria. Evaluation of these patients included a complete eye examination including slitlamp biomicroscopy, the Watzke-Allen test and 50-µm aiming beam test for a central scotoma, fundus photography and fluorescein angiography, and echography. Eight eyes were also evaluated using the retinal thickness analyzer (Talia Technology Ltd, Zion, Israel).

Patients were observed at frequent intervals because of the traction on the fovea. At the beginning of this study, the

"tractional elevation of Henle's fiber layer with intraretinal foveolar cyst formation is the initial feature of macular hole development."

Following is our study of 18 patients who had cysts in the fovea that appeared to be due to vitreous traction and often represented a premacular hole condition. We use the term cysts because it has been prevalent in the literature and is descriptive of the outer thinning seen in the fovea on biomicroscopic examination of these patients. We realize that the areas of thinning are probably not lined by epithelium and the term "cyst" may therefore be a misnomer.

REPORT OF CASES

CASE 1

A 65-year-old woman (patient 13 in the **Table**) was first seen with a 2-month history of decreased visual acuity and metamorphopsia in the left eye. The visual acuity was 20/40 OS and radial striae were seen that were thought to be either in the cortical vitreous or inner layers of the foveal retina (**Figure 1**, A). The patient denied a central scotoma. One month later the visual acuity was still 20/40 and the clinical findings were unchanged. Seven months later the visual acuity was 20/200 and a fullhickness hole with surrounding fluid had developed (Figure 1, C). eye examinations and ancillary tests were performed as part of the routine management of the patient. When we decided to analyze and then publish the accumulated data, we obtained informed consent to do so from the patients.

DESCRIPTION OF FOVEAL CYST

Unfortunately we do not have clinical pathologic correlation for any of these patients. Patients with foveal cysts had combinations of two findings although one could be more prominent than the other. First there were striae present either in the vitreous cortex overlying the fovea or within the inner retina. Usually the striae radiated outward from the fovea in a spokelike pattern. The overall thickness of the fovea is normal, although in some cases the inner layer of the fovea appears to be pulled forward slightly. However, it is seldom pulled forward to the level of the retina surrounding the fovea or as forward as the inner retina in eyes with holes that follow the Gass classification.

The second main finding of eyes with cysts is a reddish appearance to the outer retina beneath the inner striae. This reddish appearance appears as tissue loss within the layers of the fovea, perhaps like a very localized area of schisis. The area of redness (or perhaps schisis) is usually circular or oval although it can have scalloped edges. It usually involves an area about equal to the foveal avascular zone. Ocassionally, however, the reddish change is confined to just a few small round or oval facets in the fovea. In patients who developed full-thickness holes, the hole started as a small round defect in the center of the outer wall of thinning or schisis within the fovea. Patients could have decreased vision but no detectable central scotoma until a full-thickness hole formed.

When the patient was initially examined, the right eye was asymptomatic. The visual acuity was 20/15, the macula looked normal, and there was no PVD. Fortyfive months later the patient noticed decreased acuity in the right eye. The acuity was now 20/25 and radial striae with outer retinal redness were seen in the fovea of the right eye (Figure 1, D). The appearance of the fovea and visual acuity remained fairly stable on multiple visits until 40 months after the striae had first appeared. The patient then returned with complaints of further decrease in vision. The acuity was now 20/40 and a small fullthickness macular hole was noted in the center of the floor of the outer retinal thinning (Figure 1, F). A vitrectomy was performed and the vitreous was peeled from the posterior pole using suction followed by a fluid-gas exchange. The hole sealed and the patient regained 20/20 acuity. Twenty-six months after surgery the acuity is 20/40, probably due to nuclear sclerosis of the lens.

CASE 2

A 66-year-old woman (patient 9 in theTable) was referred for "macular holes in both eyes." The acuity was 20/200 OS and a full-thickness macular hole with a cuff of fluid was present.

The acuity was 20/20 OD and a small round area of outer retinal thinning was seen in the fovea. Over mul-

Patients With Foveal Cysts*

Patient No./ Age, y/Sex	Visual Acuity and Clinical Findings on Examination	Follow-up Comments
1/63/F	OD: 20/40 with striae and thinning-S	Vx with peeling hyaloid 1 wk after initially seen; 20/20 56 mo after Vx
	OS: 20/100, stage 2 macular hole	20/400, open hole
2/63/F	OD: 20/25 with striae and thinning-S	11 mo later 20/80, stage 2 macular hole with yellow ring, Vx with peeling and gas; 20/25 41 mo after Vx, hole sealed; RTA: irregular foveal depression
	OS: 20/100, stage 2+ macular hole	Vx 1 wk later; 20/30 50 mo after Vx
3/64/M	OD: 20/20 inner striae, minimal outer thinning-S	71 mo later 20/25, no PVD, still inner striae; RTA: splitting in middle retina and loss of inner retinal layer in center
	OS: 20/200, stage 2 macular hole	1 mo later Vx; 20/300, hole sealed but adverse reactions to light
4/65/F	OD: 20/30, outer thinning-S	40 mo later 20/30, still thinning; now partial PVD but still attached to fovea; RTA: splitting in middle layers and loss of inner retina in center
	OS: 20/30–, thin area but not sure if PVD present on initial examination	2 mo later PVD noted; 40 mo later 20/50
5/63/F	OD: 20/25+3, inner striae and large area of outer thinning-S	4 mo later PVD and area of thinning much less prominent; 20/20 23 mo after PVD; RTA: essentially normal fovea
	OS: 20/100, amblyopia; no foveal changes	No change
6/62/M	OD: 20/30, outer thinning-S	19 mo later 20/50, thinning thought to be worse; had Vx; during Vx fovea took on yellow appearance as vitreous peeled; 20/60 with adverse reactions to phototoxicity signs 59 mo after Vx
	OS: 20/30, outer thinning but less prominent than OD-S	31 mo later had PVD; 47 mo after PVD 20/20 with residual thinning; RTA: loss of inn retinal layer
7/48/F	OD: 20/25, mostly striae, minimal thinning-S	34 mo later 20/30 with minimal striae; no PVD; RTA: splitting in middle retinal laye but intact inner layer
	OS: 20/200, stage 2 macular hole	Vx; 34 mo later 20/30, hole sealed
8/66/F	OD: 20/200, with macular hole	No change
	OS: 20/30, inner striae and outer thinning-S	18 mo later PVD; striae and outer thinning much less prominent; 11 mo after PVD 20/20
9/66/F	OD: 20/20, outer thinning-S	Acuity slowly decreased to 20/40 with outer thinning still present; 44 mo later 20/70 with small central hole in base of thinning; Vx 20/40 7 mo after Vx
10/01/5	OS: 20/200, macular hole	No change
10/61/F	OD: 20/25, inner striae-S	7 mo later full-thickness macular hole; 20/200 5 mo after full-thickness macular hol first noted; no treatment
	OS: 20/25, inner striae-S	5 mo later 20/50 with small central hole and yellow spot; 20/200 7 mo after full-thickness macular hole first noted; no treatment
11/78/F	OD: 20/25, outer thinning-S	70 mo later 20/40 with outer thinning still present; initially no PVD; starting 43 mo after first seen shows subtotal PVD with insertion into disc and macula
	OS: 20/60, outer thinning-S	11 mo later 20/200 with small central hole; had Vx; 20/60 with sealed hole 59 mo after Vx
12/64/F	OD: 20/20, striae and outer thinning-S	14 y 2 mo later 20/20 with small areas of outer thinning; findings on echography: no PVD and small area of adhesion temporally to fovea
13/65/F	OS: 20/70, macular hole	Counting fingers OS
13/05/F	OD: 20/15, normal-S	45 mo later 20/25, striae; 40 mo after striae 20/40 and small central hole; had vitrectomy, 20/20 with sealed hole; now 20/40 due to cataract 20 mo after Vx
14/63/F	OS: 20/40, inner striae-S	7 mo later 20/200 with macular hole
	OD: 20/15, normal macula-S	10 mo later 20/15, small areas of outer thinning; 3 mo later 20/15, still thinning, no PVD; RTA: imperfectly shaped fovea
	OS: 20/30, small area of outer thinning-S	3 wk later 20/25, PVD, cystic appearance almost gone
15/70/F	OD: 20/20, macula shows a little thinning, PVD	No change
16/63/M	OS: 20/50, small area of outer thinning-S OD: 20/20, small area of outer thinning-S	26 mo later 20/50, still small area of outer thinning, no PVD
	OS: 20/150, stage 2 macular hole	Thinning worsened; 4 mo later 20/50, small central hole; Vx: 20/25+2 7 y after Vx No treatment; 87 mo later 20/20. hole sealed
17/59/F	OS: 20/150, stage 2 machina noie OD: 20/50, vitreomacular traction syndrome, small central hole	Vx 3 mo later, hole closed, 20/20, nois sealed Vx 3 mo later, hole closed, 20/20; red cystic appearance became more prominent with epiretinal membrane; 13 mo after Vx had cataract extraction; 6 mo after cataract sm central hole at base of cyst, 20/80; second Vx, epiretinal membrane peeled; 20/30 w hole sealed 39 mo after second Vx
	OS: 20/20, inner striae, outer thinning-S	56 mo later 20/25; still prominent striae and outer thinning; no PVD and thin membrane on macula seen on echography
18/75/M	OD: 20/80, macular hole	20/500, no treatment
	OS: 20/20, small area of outer thinning-S	9½ y later 20/30, outer thinning no PVD; RTA: minimal splitting in middle layers

*-S and boldfaced words indicate eye included in the study; PVD, posterior vitreous detachment; Vx, vitrectomy with peeling of the hyaloid off the retina; RTA, retinal thickness analyzer; and striae and thinning, foveal changes seen on biomicroscopic examination.

tiple visits during the next 3 years, the visual acuity decreased to 20/40 and the outer thinning became more prominent, but the patient denied a central scotoma. Forty-four months after the initial visit the patient returned com-

plaining of further loss of visual acuity. The acuity was now 20/70 and a small full-thickness macular hole was noted in the center of the base of the outer retinal thinning. A vitrectomy was performed and the vitreous was peeled from

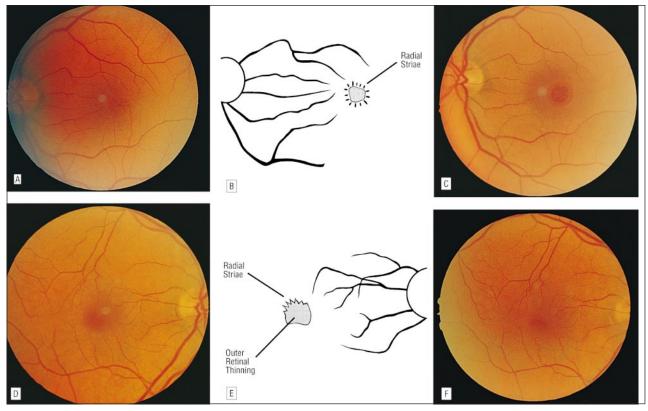


Figure 1. *A*, The left eye of case 1 (patient 13 described in the Table) when she first was seen. The main findings at this time were radial folds in the overlying vitreous cortex and possibly in the inner retina of the fovea. B, Drawing of Figure 1, A, left showing radial folds. C, This patient was followed up and developed a full-thickness retinal hole with a cuff of fluid 7 months later. No pictures were taken at that time, but a follow-up photograph 15 months after our initial examination shows a well-developed macular hole with surrounding fluid. D, Initially the right eye was normal on multiple follow-up visits. Forty-five months after the patient had first been seen, she developed symptoms and now had developed radial striae and outer retinal thinning in the fovea as shown in the fundus photograph. E, Drawing showing radial striae that are most prominent in the superotemporal aspect of the fovea as well as an oval area of outer retinal thinning. F, The vision and cystic change in the fovea stayed relatively stable for an additional 40 months at which time the patient returned with complaints of further decreased acuity. A small full-thickness macular hole was noted in the center of the floor of the outer retinal thinning as shown in the photograph.

the posterior retina using suction followed by a fluid-gas exchange. The hole sealed and the acuity was 20/40 7 months after the vitrectomy at which time the patient returned to her referring physician.

CASE 3

A 66-year-old woman (patient 8 in the Table) was first seen with decreased acuity in both eyes that was worse in the right. The acuity was 20/200 OD and a fullthickness macular hole with a cuff of fluid was seen.

The acuity was 20/30 OS. There was a cloverleaf pattern of radial striae along with outer retinal thinning seen in the fovea. No PVD was seen clinically or on echography. The patient was followed at 4- to 6-month intervals and there was no change in acuity or the appearance of the fovea. Eighteen months after our initial examination, the patient returned and the acuity was 20/ 20. The striae and thinning were now barely evident. Findings from repeated echography now revealed a total PVD. Eleven months after this last visit the acuity remains 20/20 and the fovea appears normal.

CASE 4

A 64-year-old man (patient 3 in the Table) was seen with decreased acuity in the left eye and metamorphopsia in

the right. The acuity was 20/200 OS and a stage 2 fullthickness macular hole was seen. A vitrectomy was performed and the hyaloid peeled from the retinal surface. The hole sealed, but the patient developed extensive pigmentary changes in the posterior pole that were thought to be due to phototoxicity. The acuity is 20/300 6 years after the vitrectomy.

The acuity was 20/20 OD. Radial striae and minimal outer retinal thinning were seen in the fovea. Seventyone months later, the visual acuity was 20/25 and minimal radial striae with outer thinning was still seen in the fovea. The patient denies a central scotoma. Findings on echography show an attached vitreous. The retinal thickness analyzer showed an optically empty region in the middle retinal layers of the fovea. This optically empty region extended throughout most of the 2-mm section, or for perhaps 1.5 mm. In the center of the optically empty region, the inner retina appeared to be absent.

CASE 5

A 48-year-old woman (patient 7 in the Table) was seen with decreased acuity in the left eye. The acuity was 20/200 and a stage 2 full-thickness macular hole was present. A vitrectomy was performed with peeling of vitreous from the retina followed by fluid-gas exchange. The hole sealed and the acuity was 20/30 34 months after surgery.

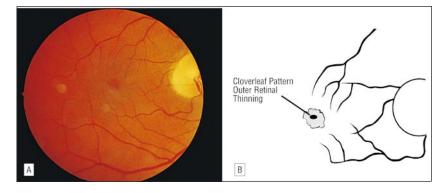


Figure 2. A, The right eye of case 5 (patient 7 described in the Table). Striae and mild cloverleaf outer retinal thinning were seen in the fovea of the right eye on examination. This eye has remained stable over 34 months of follow-up. B, Drawing showing a small cloverleaf area of outer retinal thinning.

On examination, the acuity was 20/25 OD and there were prominent striae and mild outer retinal thinning present in the fovea (**Figure 2**). The acuity and foveal appearance remained stable over multiple follow-up visits. Thirty-four months after the initial presentation the acuity remained 20/30 and minimal stria and thinning were still present although minimal. Findings from echography show no vitreous detachment. The retinal thickness analyzer shows an optically empty region within the middle retinal layers but no unroofing of the cyst or defect in the inner wall.

RESULTS

Eighteen patients had foveal cysts and no vitreous detachment. Fourteen of the patients were women. Fifteen patients were between the ages of 59 and 70 at the time of our examination. The remaining 3 were seen at ages 48, 75, and 78. Five patients had cysts in both eyes making 23 total eyes in the study. Eighteen eyes in 13 patients underwent fundus fluorescein angiography. The capillaries surrounding the foveal avascular zone did not appear to be distorted on fluorescein angiography even in eyes with prominent striae. There was also no evidence of late leakage or cystoid macular edema in these eyes. There was either normal background or slight hyperfluorescence in the center of eyes with prominent thinning. The hyperfluorescence was minimal, however, and not as prominent as seen with a full-thickness macular hole. Stereoscopic fluorescein angiography was the best method to detect when a hole did develop in the outer retinal layer because it resulted in a small round transmission defect in the center of the fovea.

On final follow-up, 9 of the 23 eyes did not develop a PVD and still had a cystic appearance in the fovea. The times of follow-up after the cystic fovea was first seen and the acuities in these 9 patients are as follows: 3 months (20/15); 26 months (20/50); 34 months (20/30); 40 months (20/30); 56 months (20/25); 70 months (20/40); 71 months (20/25); 114 months (20/30); and 168 months (20/20).

Eight of the 23 eyes developed full-thickness macular holes on follow-up. The time between when the foveal cyst was first seen and the macular hole detected on examination, the acuity at the time the hole was first diagnosed, and the follow-up acuities are as follows: 4 months (20/50; now 20/25 after vitrectomy); 5 months (20/50; now 20/200 with no treatment); 7

months (20/200; no treatment); 7 months (20/200; no treatment); 11 months (20/80; now 20/25 after vitrectomy); 11 months (20/200; now 20/60 after vitrectomy); 40 months (20/40; was 20/20 after vitrectomy, now 20/40 due to cataract); 44 months (20/70; now 20/40 after vitrectomy).

Four of the 23 eyes developed PVDs on follow-up. The time the PVD was noted after the cysts were first seen and the final acuity of these eyes are as follows: 3 weeks (20/25); 4 months (20/20); 18 months (20/20); and 31 months (20/20) (**Figure 3**).

Two of the 23 eyes had undergone vitrectomies for the cysts before either a macular hole or PVD had developed (**Figure 4**). One of these eyes had a vitrectomy only 1 week after the cyst was first noted and acuity improved from 20/40 to 20/20. The other eye had a vitrectomy 19 months after first being seen with a cyst. The acuity decreased slightly to 20/60 after vitrectomy from the 20/50 preoperative value. There was also pigmentation in the macula thought to be due to phototoxicity.

Eight patients and eyes were investigated using the retinal thickness analyzer. Two patients (Nos. 3 and 4 in the Table) showed optically empty regions within the middle layers of the retina that extended well beyond the area of the cyst visible on clinical examination and also unroofing of the cyst or loss of the inner layer centrally (Figure 5). Both of these patients denied a central scotoma and had 20/25 and 20/30 acuity. Two patients (Nos. 7 and 18 in the Table) had optically empty regions in the middle layers of the retina but no unroofing of the cyst or tissue loss in the inner layer. Both patients denied a central scotoma and had 20/30 visual acuity. One patient (No. 14 in the Table) had only 3 months of follow-up after minimal cystic changes were seen in the fovea. The retinal thickness analyzer showed no splitting, but the fovea was not shaped perfectly because one side was steep rather than gently sloping posteriorly as seen in normal patients. The visual acuity was 20/15 in this eye. One patient (No. 5 in the Table) was examined 23 months after a PVD and had an essentially normal fovea. Another patient (No. 6 in the Table) had what appeared to be a lamellar hole with inner retinal tissue loss in the fovea. The patient had an acuity of 20/20, denied symptoms, and was examined with the retinal thickness analyzer 47 months after a PVD in this eye. The final patient (No. 2 in the Table) had an irregular foveal depression 41 months after a vitrectomy to seal a stage 2 macular hole.

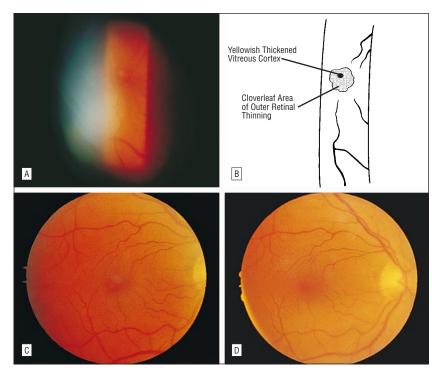


Figure 3. A, Slitlamp photograph of the fovea of patient 5 in the Table. On examination, this patient had a cloverleaf area of outer retinal thinning with mild inner striae in the right eye. B. Drawing of the area illuminated by slitlamp showing highlight over the superior fovea that probably represents thickened cortical vitreous as well as a cloverleaf area of outer retinal thinning. C, Fundus photograph of the right eye taken at the same time as seen in Figure 3, A. D, The patient developed a posterior vitreous detachment 4 months after the initial examination and the area of thinning and striae became much less prominent as shown in the follow-up fundus photograph. Visual acuity has remained 20/20 23 months after the posterior vitreous detachment.



Figure 4. Fundus photograph of the right eye of patient 6 described in the Table showing prominent outer retinal thinning at the time of initial examination. The visual acuity was 20/30 and the patient denied a central scotoma. No full-thickness macular hole was seen in the base of the cyst.

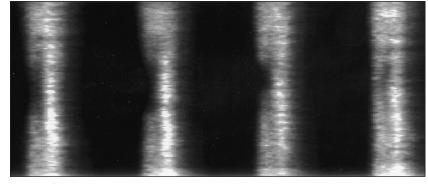


Figure 5. Slitlamp images taken with the retinal thickness analyzer centered on the fovea of the right eye of case 4 (patient 3 in the Table). Images at the far left and right show an optically empty region in the middle retinal layers. The central 2 images show an absent or extremely attenuated inner retinal layer in the center of the fovea.

COMMENT

On biomicroscopic examination, radial striae were seen in the vitreous cortex immediately overlying the fovea in most patients. In some cases it appeared that the striae also involved the superficial retina; although on fluorescein angiography, we did not detect distortion of the foveal capillaries that were surrounding the perimeter of the striae. Kishi et al⁸ believe that the radial striae are due to traction and distortion of Henle's layers.

These patients also have round or oval red cysts often with sharp margins deep to the striae. The round cysts usually comprise an area about equal to the foveal avascular zone or slightly smaller. Sometimes the round areas appear to composed of smaller lobules or facets although this segmented appearance may be due to blocking by the overlying striae. We believe these lesions are the ones described in the previous reports as macular cysts.²⁻⁷ Within this report we have described this outer retinal thinning as "redness," "thinning," "tissue loss," a "cyst," or "schisis." It seems clinically that the redness represents tissue loss or splitting within the fovea. However, histopathologic examinations of patients with well documented foveal cysts are needed to provide the conclusive answer.

We excluded patients with the vitreomacular traction syndrome or visible epiretinal membranes. Both of these conditions, however, can cause cystic changes in the fovea indistinguishable clinically from the patients described. The foveal changes in all of these conditions are probably the same or very similar because the common cause appears to be traction from the vitreous or preretinal membranes. An advantage of this study is that these patients were followed up sequentially by 1 of 2 of us (J.C.F. or H.C.B.) and the status of a PVD was documented by

multiple echographic examinations or by peeling of the posterior hyaloid during a vitrectomy. When the traction was relieved by a spontaneous PVD or a vitrectomy, the cystic changes became much less prominent or disappeared and the acuity stablilized or improved.

Nine of the patients so far still have no PVD despite a prolonged follow-up in some cases and these patients still have foveal cysts. Eight patients developed fullthickness holes on follow-up. These patients noticed an abrupt decrease in visual acuity and there was a sharp decrease in acuity on examination. The holes first appeared on bimomicroscopic examination as small round defects in the center of the outer layer of the cyst often with a thin surrounding border of yellowish subretinal fluid or perhaps luteal pigment. A defect in the outer layer centrally exposing the underlying retinal pigment epithelium was also readily visible on fluorescein angiography.

Examination with the retinal thickness analyzer revealed splitting or schisis in the middle layers of the retina in 4 patients and unroofing of the central area of splitting in 2 of these 4. These findings are not conclusive, however, because the retinal thickness analyzer shows mainly reflections from the inner retina and the retinal pigment epithelium. Therefore, the "splitting" of the middle layers could be due to fluid at the margins of a full-thickness macular hole. The area of splitting was often large which would mean that there would have to be an extensive, albeit shallow, detachment of the perifoveal retina that was not observed clinically. In addition, the visual function in these patients was good which does not support the presence of an extensive detachment of the perifoveal retina. Therefore, we believe the splitting is within the retinal layers. In addition, there may still have been an extremely attenuated layer of inner retina present that was not detected by the "gain" of the machine in the cases that showed unroofing of the cyst or absent inner retinal layers. If the inner retinal layers were indeed absent, these patients could be said to now have lamellar holes in addition to splitting within the retina. The vitreous remained attached in these patients, however, so they are probably still at risk for the future development of a full-thickness macular hole.

The findings of schisis and unroofing of the inner layer found by the retinal thickness analyzer do correlate nicely with the clinical findings and course. We believe that foveal cysts are caused by traction on or at the edges of the fovea for whatever reason. The traction is evident by radial folds in the vitreous cortex immediately overlying the foveal retina and perhaps folds in Henle's layer within the retina. The traction causes damage to the fovea that is seen as reddish cystlike areas clinically and as splitting in the middle retinal layers on examination with the retinal thickness analyzer. The vision remains good even with unroofing of the inner retinal layers. The outer fovea appears to be markedly resistant to traction. The vision and cyst can remain stable for years; but once a small defect develops in the center of the outer retinal thinning, there is an abrupt loss of acuity and a larger stage 3 full-thickness macular hole with surrounding fluid follows quickly.

The correct management appears to be to follow up on these patients and warn them to return promptly if their visual acuity decreases. A defect in the outer wall of the cyst is an early full-thickness macular hole. Three of such eyes were followed up without treatment and progressed to large macular holes with surrounding fluid and poor vision. Five had vitrectomies with peeling of the posterior hyaloid and intraocular gas. Four regained 20/20 to 20/25 acuities and the fifth has an acuity of 20/60 with signs of phototoxicity. Therefore, a prompt vitrectomy with gas should be considered once a full-thickness macular hole occurs.

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100 Years Ago in the ARCHIVES

A look at the past . . .

s group of four cases of optic atrophy due to sexual excess, by Dr. J. A. SPAULDING, of Portland, Me. Four young men became affected with optic-nerve atrophy. No cause could be found but sexual excess. There were no signs of tabes.

Reference: Arch Ophthalmol. 1897;26:439.