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Prevalence and characteristics of hyporeflective preretinal tissue in vitreomacular interface disorders

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Abstract

Aims—To assess prevalence and characteristics of hyporeflective preretinal tissue on spectral domain optical coherence tomography (SDOCT) in eyes with vitreomacular interface disorders.

Methods—4037 eyes (3195 patients) with diagnosis of lamellar macular hole (LMH), full-thickness macular hole (FTMH), epiretinal membrane (ERM) and vitreomacular traction (VMT) were included. Quantitative analysis was performed including volume and area of the epiretinal proliferation, as well as the brightness of the hyporeflective band. Clinical characteristics were also collected and analyzed.

Results—A hyporeflective preretinal tissue layer was identified in 204 of 4037 eyes (5.1%); 162 eyes in LMH (79.4 %), 23 eyes in FTMH (11.3%) and 19 eyes in ERM (9.3 %). In LMH, the visual acuity was significantly different between the cases with and without epiretinal proliferation at the initial and the final visit, ($P = 0.012$, 0.046 , respectively). The maximum thickness, area, volume of hyporeflective preretinal tissue became significantly larger during observation period ($P < 0.001$). Brightness of the preretinal tissue (109.3 ± 21.1 a.u.) was close to retinal ganglion cell layer (112.0 ± 19.5) and retinal outer plexiform layer (117.7 ± 19.5).

Conclusions—Hyporeflective preretinal tissue was found with significant frequency of eyes with LMH, FTMH and ERM, with a particularly high incidence in LMH. The increased presence of this tissue in cases of LMH may signify a particular subtype of LMH. More research is needed to better understand the implications of the presence of this tissue for visual and surgical outcomes.

Keywords

preretinal tissue; spectral-domain optical coherence tomography; vitreomacular interface disorders; lamellar hole

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Introduction

The revolution of optical coherence tomography (OCT) has allowed clinicians to evaluate retinal microstructures with much greater detail than previously possible. The accuracy of diagnosing vitreomacular interface disease and elucidation of its pathophysiology has been greatly improved with the increased resolution of OCT technology. Lamellar macular holes (LMH) is a distinct clinical entity, and several diagnostic criteria has been reported with widespread use of OCT. The features and pathophysiology that define LMH however remain controversial.¹⁻⁵ Haouchine et al indicated that LMH usually have a thin irregular center with loss of foveal tissue surrounded by a retina of almost normal thickness with the edge of the LMH split away from the underlying retina.³ Later, Witkin et al suggested 4 criteria for diagnosis of LMH made by ultra-high resolution OCT appearance: (1) an irregular foveal contour; (2) a break in the inner fovea; (3) separation of the inner from the outer foveal retinal layers, leading to an intraretinal split; and (4) absence of a full thickness foveal defect with intact photoreceptors posterior to the area of foveal dehiscence.⁴

In association with LMH, Pang et al reported an entity that described as “lamellar hole-associated epiretinal proliferation” (LHEP).⁶ This entity was first noted as a thickened ERM⁴ and was described as a feature specific to LMH. This paper suggested LHEP is primarily originated in a proliferation of Müller cells onto the inner retina and seen by SDOCT as a material of medium reflectivity that molds to the inner retinal anatomy.

The purpose of this study was to assess the prevalence of this hyporeflective preretinal tissue in vitreoretinal interface disorders such as LMH, full-thickness macular hole (FTMH), epiretinal membrane (ERM), and vitreomacular traction syndrome (VMT). In addition, the goal was to characterize the SDOCT features and the effect on visual acuity of this preretinal tissue seen in the setting of LMH.

Patients and Methods

This is a Cleveland Clinic IRB-approved, retrospective consecutive case series of eyes with LMH, FTMH, ERM, or VMT based on SDOCT imaging. A retrospective review was performed for patients from April 2009 to October 2014 who underwent OCT examination for associated ICD-9 billing codes for FTMH, ERM, VMT, macular pseudohole (MPH), or LMH. All SDOCT scans were reviewed for confirmation of diagnosis. OCT-based definitions for LMH and macular pseudohole were utilized as follows. LMH was defined based on OCT imaging to have the following findings: irregular foveal floor, tissue cleavage between inner and outer retina, a potentially thin foveal center and often normal parafoveal retinal thickness. A MPH was defined as a foveal-sparing ERM that resulted in alterations to the foveal architecture with steepened foveal pit with thickened smooth foveal edge and a small foveal pit diameter without associated complete inner and outer retinal cleavage, and/or loss of the retinal tissue.^{3,4,7} Given this definition, all MPH cases were grouped with the ERM cases. All OCT images had been obtained using the Cirrus HD-OCT (Cirrus V.6.1 software, Carl Zeiss Meditec, Dublin, CA). Both SDOCT macular cube and 5-line raster scans were obtained. SDOCT images were reviewed by two observers. Exclusion criteria included inability to grade SDOCT due to poor quality (e.g., scans with a signal strength of

less than 8 out of 10), lack of SDOCT scans, and eyes with history of previous vitrectomy or intravitreal drug injection. Clinical variables collected included age, gender, lens status and Snellen visual acuity (VA).

All eyes with hyporeflective preretinal tissues were further characterized. The mean gray scale, minimum and maximum gray scale was calculated to evaluate brightness of the epiretinal proliferation and each retinal layer [i.e., retinal nerve fiber layer (NFL), ganglion cell layer (GCL), inner plexiform layer (IPL), inner nuclear layer (INL), outer plexiform layer (OPL) and outer nuclear layer (ONL)] was evaluated with the Image J software version 1.45 (National Institutes of Health, Bethesda, MD). SDOCT images were exported and imported into ImageJ for analysis. The area of interest was specified by utilizing polygon selections to calculate the brightness utilizing the measurement tool in ImageJ. The Cirrus review software caliper tool was utilized to manually measure the maximum preretinal thickness. All measurements were performed in a masked fashion on each SDOCT scan.

For area and volumetric analysis, a custom software analysis program was utilized to segment the areas of interest. The frame-by-frame B-scans were processed by a masked grader for optimal segmentation of any hyporeflective preretinal tissue, resulting in the delineation of any epiretinal proliferation in every OCT frame.⁸ A three-dimensional surface representation of the preretinal tissue was generated and the volume and area were calculated.

The Snellen VA was converted to the logarithm of minimal angle of resolution (logMAR) units for the statistical analysis. To compare two groups, Student t-test was used with parametric distribution data. Wilcoxon signed rank test and Mann-Whitney U tests were used with nonparametric distribution data. The data were analyzed with Statcel software (3rd edition OMS, Tokyo, Japan). A p-value less than 0.05 was considered statistically significant.

Results

Clinical Overview of Eyes with Preretinal Tissue

A total of 5825 eyes with the diagnosis of a vitreoretinal interface disorder and SDOCTs were reviewed. Of those eyes, 1710 eyes were excluded because of insufficient SDOCT images, lack of vitreomacular interface pathology on OCT, or insufficient clinical data. An additional 73 eyes were excluded because of previous vitrectomy and 5 eyes because of previous intravitreal drug injection. In the remaining 4037 eyes of 3195 patients, 272 were classified as LMH, 3291 eyes had ERM, 102 eyes of 101 as MPH, 240 eyes of 233 cases as MH and 132 eyes of 118 cases as VMT (Table 1).

Hyporeflective preretinal tissue was identified in 204 eyes of 192 patients (5.1 %) out of the total 4037 eyes in 3195 patients (Figure 1). The mean age of patients with hyporeflective preretinal tissue was 72.0 ± 10.9 years (range; 23–96 years). Of those 204 eyes, 162 eyes had LMH (79.4 %), 23 eyes had MH (11.3%) and 19 eyes had ERM (9.3 %). Hyporeflective tissue was not identified in any of the VMT cases. When examining baseline VMI diagnosis,

preretinal tissue was present in 162 of 272 eyes (60.0 %) with LMH, 23 of 240 eyes (9.6 %) with FTMH and 19 eyes of 3291 eyes (0.6 %) with ERM ($P < 0.001$, Kruskal-Wallis test).

Characterization of LMH Eyes

Of those eyes with LMH, mean age of eyes with preretinal tissue (71.9 ± 10.9 ; mean $\pm$ SD) was not significantly different ($P = 0.61$, Student t-test) from those patients without the proliferation (71.3 ± 11.5). The mean visual acuity at initial visit of the patients with LMH with preretinal tissue was 20/48 (logMAR units; 0.38 ± 0.27) which was significantly less than the mean visual acuity of 20/42 (logMAR units; 0.32 ± 0.44) for eyes without preretinal tissue ($p = 0.012$). The visual acuity at the final visit was significantly lower in eyes with preretinal tissue [20/45 (logMAR units; 0.35 ± 0.31)] compared to those without [20/40 (logMAR units; 0.30 ± 0.40); $p = 0.046$]. The visual acuity did not change significantly during the observation period in either group (mean observation period = 16.8 months, 23.7 months, respectively).

Quantitative Assessment of OCT Features of Preretinal Proliferations in LMH

The mean maximum thickness of preretinal tissue in cases with LMH at initial visit was $35.9 \pm 21.9 \mu\text{m}$ and significantly increased to $43.0 \pm 23.7 \mu\text{m}$ at the final visit ($P < 0.001$, Wilcoxon signed rank test). The maximum preretinal tissue area at initial visit was $0.043 \pm 0.042 \text{ mm}^2$ and at final visit was $0.053 \pm 0.049 \text{ mm}^2$. This was a statistically significant difference ($P < 0.001$, Wilcoxon signed rank test). The volume of preretinal tissue at initial visit was $0.044 \pm 0.069 \text{ mm}^3$ and was $0.061 \pm 0.087 \text{ mm}^3$ at the final visit, which again was a statistically significant difference ($P < 0.001$, Wilcoxon signed rank test).

Two variants of preretinal tissue bands were identified. One variant (107/162, 66%) was encapsulated by ERM (i.e, a surrounding hyperreflective membrane) that created a discontinuous appearance Group A). The second variant appeared continuous across the retinal surface without any evidence of encapsulation (Group B). At the initial visit, the preretinal tissue in LMH was encapsulated (Figure 3) in 107 of 162 (66%) eyes (Group A) and was not encapsulated in 55 of 162 eyes (Group B). A multivariate analysis was performed to assess the correlation between the configuration of preretinal tissue with age, gender, lens status, laterality of eye, best-corrected visual acuity and frequency of retinal surgery during this observation period. None of these factors correlated with subtype of preretinal tissue. Maximum thickness of preretinal tissue (31.6 ± 18.4 , $55.1 \pm 25.6 \mu\text{m}$; Group A, Group B, respectively), maximum area (0.041 ± 0.040 , $0.085 \pm 0.059 \text{ mm}^2$) and volume (0.041 ± 0.079 , $0.13 \pm 0.13 \text{ mm}^3$) were however significantly greater in Group B compared to Group A ($P < 0.001$, for all, Mann-Whitney U test).

The brightness of preretinal tissue and each retinal layer were summarized in Figure 2. The mean brightness of the preretinal tissue with LMH was 109.5 ± 21.0 (mean $\pm$ SD, a.u.). Brightness of each retinal layer was also evaluated. The mean brightness of NFL, GCL, IPL, INL, OPL and ONL were 164.4 ± 24.6 , 112.3 ± 19.6 , 125.8 ± 20.5 , 91.3 ± 18.4 , 118.0 ± 19.7 and 69.1 ± 13.7 , respectively.

Discussion

Similar to other studies, hyporeflective preretinal tissue was identified in a significant number of patients with VMI disorders, particularly in those patients with LMH.^{2, 4, 6, 9} Pang et al suggested that the proliferation is seen primarily in association with defects extending to the middle retinal layers as is seen with inner lamellar holes. In their case series, 'LHEP' was seen in LMH (30.5 %), FTMH (8.0 %) and was not observed in ERM cases in which there were no lamellar defects on OCT.⁶ In our study, however, preretinal tissue was found in 162 of 272 eyes (60.0%) with LMH cases, 19 of 3393 cases (0.6%) with ERM and in a similar percentage of FTMH cases (9.6%). Of those 19 eyes with ERM, 15 cases were in the presence of other retinal diseases such as proliferative diabetic retinopathy, retinal vein occlusion, retinal artery occlusion, age-related macular degeneration and the remaining 4 cases were without any other ocular diseases. This result suggests that preretinal tissue formation in ERM may more frequently arise in the setting of other concurrent retinal disease and in idiopathic ERM the rates of preretinal tissue is considerably lower.

The definition of LMH remains controversial and the factors involved in LMH formation are also not entirely clear. Gaudric et al defined LMH by OCT architecture that irregular foveal floor, cleft between inner and outer retina and large aperture of the hole compared with MPH.⁷ Haouchine et al also suggested LMH has a thin foveal center surrounded by a retina of almost normal thickness. On the other hand, MPH was defined that resulted in a verticalized foveal pit, straight and smooth edge of fovea by centripetal ERM contractile force.³ MPH often have a center of almost normal retinal thickness, surrounded by a thickened retina.⁷

In a small case series, Bottoni et al suggested that LMH is a stable condition, and visual acuity and foveal thickness in cases (n = 10) with 'thicker ERMs' did not change during observation period (mean follow-up: 18 months), and did not differ significantly from eyes with 'normal ERMs', (n = 24).⁹ However, in a larger study (n = 145), Pang et al recently reported that visual acuity of LMH cases with "LHEP" was significantly poorer than eyes without LHEP similar to our findings. The authors' hypothesis for the variation in visual acuity was due to larger LMH diameter at the middle retinal level, thinner retinal thickness at the base of the LMH, and increased ellipsoid zone disruption (mean follow-up period; 26.7 ± 23.6 months).¹⁰

Both our study and the study by Pang et al reveal that the presence of this tissue is highly associated and characteristic of LMH, though these studies reveal a moderate prevalence in FTMH and rare prevalence in ERM.⁶ Given the high prevalence in LMH, it is certainly possible that the presence of this feature may be a by-product of LMH pathophysiology and formation. Conversely, the presence of this tissue may be a causative factor in the formation of LMH that may, in fact, play a significant role in a unique tangential tractional pattern that results in LMH formation.

The impact of the presence of the hyporeflective preretinal tissue on prognosis remains unclear. In this study, the maximum thickness, maximum area and volume of preretinal tissue increased with time although the visual acuity did not change during the observation

period. However, visual acuity in the cases with preretinal tissue was statistically significantly lower compared to the vision in those cases without the preretinal tissue both at initial visit and final visit.

Assessment of the OCT features identified two configurations: encapsulation (Group A) and lack of encapsulation (Group B). Interestingly, although the amount of tissue was significantly different between the groups, there was no difference in visual acuity or other clinical parameters. In addition, no case showed transformation from Group A to Group B or vice versa during observation. The importance of these two configuration of these two groups remains unclear and further study with larger sample size and with longer follow-up period by using eye-tracked OCT scans or further development of retinal imaging are needed to clarify this issue. Additionally, it is unclear whether the configuration of this tissue may impact surgical outcomes or optimal surgical technique. The brightness of epiretinal proliferation has been described as 'moderately reflective' or 'medium reflectivity'.^{2, 4, 6} In this study, we quantitatively analyzed the brightness on OCT. The mean brightness of the preretinal tissue was generally hyporeflective and was close to that of ganglion cell layer. However, the make-up and origin of this tissue is not well-established. Parolini et al evaluated the 'dense ERM' by using immunohistochemical procedure after vitrectomy and reported that its components were variable, i.e. hyalocytes, glial cells and retinal pigment epithelial cells, intracellular actin filaments and extracellular matrix.² Compera et al demonstrated the difference between the hyporeflective preretinal tissue in LMH and conventional epiretinal membrane of MPH in cell type and cell distribution by utilizing immunocytochemistry and transmission electron microscopy.¹¹ Fibroblast and hyalocytes were predominant in the hyporeflective preretinal tissue and were found in cellular agglomerations without contractive components. On the other hand, myofibroblasts were the predominant cell type in conventional epiretinal membranes found in MPH. Based on these findings, the hyporeflective tissue appears more likely to originate from vitreous and possess less traditional contractile properties than cells of conventional epiretinal membrane.¹¹

Surgical repair of LMH remains controversial. Evidence is limited regarding whether surgical repair of lamellar hole is beneficial. Concerns regarding attempt at surgical repair include the potential for FTMH formation and limited visual recovery. Consistent with the Compera study, our surgical experience is that this tissue behaves like compressed cortical vitreous and may be challenging to remove. Internal limiting membrane peeling, in our experience, has been the optimal approach to ensure removal with intraoperative OCT confirmation of removal. In a comparative surgical outcome, study of LMH (11 eyes) and MPH (14 eyes), both LMH and MPH improved in visual acuity following surgical repair. All of the surgeries were performed with vitrectomy, initial ERM peeling and internal limiting membrane peeling.¹¹

As with any retrospective analysis, there are important limitations of this study. The follow-up period in this study is relatively short. Although visual acuity in eyes with preretinal tissue in LMH cases was unchanged during observation period, the long-term implications are unclear. Additionally, surgical removal was not evaluated and the implications of these findings on surgical outcomes and surgical technique remain unclear.

To our knowledge, this represents the first quantitative evaluation of the preretinal tissue commonly seen in LMH as well as the first longitudinal quantitative characterization of this hyporeflective preretinal tissue. Further assessment and research is needed to better understand the underlying pathophysiology of this proliferation, the potential natural course of the proliferation during follow-up, and to determine if this proliferation impacts surgical outcomes in patients with LMH.

In conclusion, hyporeflective preretinal tissue is a common finding in LMH. It occurs with less frequency in FTMH and rarely occurs in ERM. The preretinal tissue exhibited growth in thickness, area, and volume over time and the presence of this tissue was associated with reduced visual acuity compared to those patients without the additional hyporeflective material. The pathogenesis of this tissue including whether it is a result of the LMH process or the plays a role in the pathogenesis of LMH formation remain unclear, but it may have impact on the outcomes of patients with LMH. Further investigation with longer prospective follow-up and larger comparative surgical studies are needed to better delineate its pathophysiology and indications for surgical intervention to LMH.

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Figure 1. Spectral domain optical coherence tomography images of 71-year-old woman with lamellar macular hole (A), 86-year-old woman with full thickness macular hole (B) and 93-year-old woman with epiretinal membrane (C). All cases include examples of hyporeflective preretinal tissue (white arrows).

Figure 2.

Quantitative comparison of brightness of preretinal tissue and each retinal layers at initial visit in lamellar hole macular hole cases.

Error bars indicates the standard deviation. (a.u.; arbitrary unit)

Figure 3.

Spectral domain optical coherence tomography image of case examples showing two types of hyporeflective preretinal tissue in LMH cases. (A, C, E) Characterization of encapsulated preretinal hyporeflective tissue, Group A, in a 67-year-old woman with lamellar macular hole. (A) B-scan revealing lamellar macular hole and encapsulated preretinal proliferation (white arrows). (C) Cross-sectional 3D construction fundus OCT images with segmentation of epiretinal proliferation (green). (E) 3D reconstruction of epiretinal proliferation with superior view (upper) and lateral view (bottom). (B, D, F), Characterization of non-encapsulated epiretinal hyporeflective tissue, Group B, in a 81-year-old man with lamellar macular hole. (B) B-scan revealing lamellar macular hole and preretinal tissue that is not encapsulated by epiretinal membrane and retinal nerve fiber layer (asterisk). (D) Cross-sectional 3D construction fundus OCT images with segmentation of epiretinal proliferation

(green). (F) 3D reconstruction of epiretinal proliferation with superior view (upper) and lateral view (bottom).

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Table 1

Clinical Characteristics

	LMH	FTMH	ERM	MPH	VMT
No. of eyes (patients) Total; 4037 (3195)	272 (259)	240 (233)	3291 (2484)	102 (101)	132 (118)
No. of cases with preretinal tissue (%)	162 (60)	23 (9.6)	19 (0.6)	0 (0)	0 (0)
Age at initial visit (mean $\pm$ SD) *	72.0 $\pm$ 10.9	69.9 $\pm$ 12.1	75.7 $\pm$ 10.8	na	na
Gender, no. (%) *					
Men	68 (42)	17 (7.4)	6 (33)	na	na
Women	83 (58)	6 (2.6)	12 (67)	na	na
Laterality of eye, no. (%) *					
Right	93 (57)	12 (5.2)	10 (53)	na	na
Left	69 (43)	11 (4.8)	9 (47)	na	na
Lens states, no. (%)#					
Phakic	75 (47)	14 (6.1)	5 (26)	na	na
Pseudophakic	85 (53)	9 (3.9)	14 (74)	na	na
Snellen Visual acuity (logMAR $\pm$ SD) *					
Initial visit	20/48 (0.38 $\pm$ 0.27)	20/122 (0.78 $\pm$ 0.49)	20/239 (1.08 $\pm$ 1.52)	na	na
Final visit	20/45 (0.35 $\pm$ 0.31)	20/86 (0.64 $\pm$ 0.58)	20/205 (1.01 $\pm$ 1.46)	na	na
Terms between initial and final visit (Mo) *	16.8	25	17.9	na	na
Maximum Thickness (μ m $\pm$ SD)					
Initial visit	35.9 $\pm$ 21.9	36 $\pm$ 14.2	30.0 $\pm$ 25.5	na	na
Final visit	43.0 $\pm$ 23.7 **	42 $\pm$ 4.9	32.8 $\pm$ 29.6	na	na
Maximum Area (mm ² $\pm$ SD)					
Initial visit	0.043 $\pm$ 0.042	0.037 $\pm$ 0.10	0.056 $\pm$ 0.058	na	na
Final visit	0.053 $\pm$ 0.049 **	0.053 $\pm$ 0.014	0.067 $\pm$ 0.068	na	na
Volume (mm ³ $\pm$ SD)					
Initial visit	0.044 $\pm$ 0.069	0.018 $\pm$ 0.011	0.058 $\pm$ 0.076	na	na

Final visit	LMH	FTMH	ERM	MPH	VMH
	0.061 ± 0.087**	0.028 ± 0.006	0.083 ± 0.11	na	na

No; number, SD; standard deviation, Mo; months,

* cases with preretinal proliferation,

** P < 0.001

lens status are unknown about 2 cases