Natural course and efficacy of treatment of lamellar macular holes

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Ламелярні макулярні розриви: нативний перебіг та ефективність хірургічного лікування

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Abstract

Purpose: *To investigate the natural course of lamellar macular holes (LMH), identify possible pathways for their formation, and assess the efficacy of surgical treatment for them.*

Methods. *LMH were generally classified into tractional and degenerative based on optical coherence tomography (OCT) findings. Of the 60 patients included in the study, 41 were managed conservatively (group 1), and 19, surgically (group 2). All 19 patients in group 2 had tractional LMH. Patients from the conservative treatment group were subdivided into two subgroups, subgroup 1a of 18 patients (18 eyes) with tractional LMH, and subgroup 1b of 23 patients (23 eyes) with degenerative LMH. The minimum retinal thickness (MRT) and inner and outer diameters and depth of LMH were measured. Surgically managed patients underwent a 25G three-port pars plana vitrectomy (PPV) with epiretinal membrane (ERM) and internal limiting membrane (ILM) peeling.*

Results. *In subgroup 1a, best-corrected visual acuity (BCVA) at baseline, 6 months and 12 months was 0.18 ± 0.16 logMAR (18 eyes), 0.19 ± 0.16 logMAR (18 eyes) (p = 0.5), and 0.22 ± 0.15 logMAR (16 eyes) (p = 0.07). Additionally, the outer and inner diameters of LMH increased significantly (p = 0.042 and p = 0.037, respectively), while MRT decreased significantly, from baseline to 12 months. In subgroup 1b, BCVA at baseline, 6 months and 12 months was 0.2 ± 0.18, 0.22 ± 0.18 logMAR (p = 0.08), and 0.28 ± 0.19 logMAR (p = 0.006), respectively. Additionally, the outer diameter of LMH increased from 769.53 ± 254.01 µm to 821.73 ± 253.78 µm (p=0.04) from baseline to 12 months. In group 2, BCVA at baseline, 1 month and 6 months was 0.34 ± 0.27, 0.36 ± 0.32 logMAR (p = 0.67), and 0.28 ± 0.24 logMAR (p = 0.75), respectively. Moreover, at 1 month, LMH closure was seen in 18 of the 19 eyes (94.7%). We established three mechanisms of tractional LMH formation, with these mechanisms being associated with intraretinal cystic cavities.*

Conclusion. *The natural course of degenerative LMH was generally stable, whereas tractional LMH apparently tended to worsen anatomical OCT parameters. We found three developmental pathways for the formation of tractional LMH. Tractional LMH surgery with PPV and ERM and ILM peeling resulted in hole closure in 94.7% of the eyes.*

Keywords: *lamellar macular hole, retina, vitrectomy, optical coherence tomography, epiretinal membrane.*

DOI: https://doi.org/10.31288/Ukr.j.ophthalmol.202636167

UDC: 617.735-007.251-089

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Received 2025-05-03

Accepted 2026-06-04

Cite this article as: Novytskyy IIa, Tomkiv UM. Natural course and efficacy of treatment of lamellar macular holes. Ukr J Ophthalmol. 2026;3:61-67.

Резюме

Мета. *Дослідити нативний перебіг ламелярних макулярних розривів, виявити можливі шляхи їх формування, а також оцінити ефективність хірургічного лікування тракційних ламелярних макулярних розривів.*

Методи. *Ламелярні макулярні розриви було класифіковано на підставі ОКТ дослідження на тракційні або дегенеративні. З 60 пацієнтів, включених у дослідження, 41 пацієнт був віднесений у групу консервативного ведення (перша група) і 19 пацієнтів, яким встановлено тракційний ламелярний розрив, віднесено у групу хірургічного лікування (друга група). Пацієнтів у групі консервативного ведення було поділено на підгрупу 1а (18 пацієнтів з тракційним ламелярним розривом) і підгрупу 1б (23 пацієнти з дегенеративним ламелярним розривом). Вимірювалась мінімальна товщина сітківки, зовнішній і внутрішній діаметр та глибина розриву. Хворим другої групи проводилася закрита вітректомія (ЗВЕ) з видаленням епіретинальної мембрани (ЕРМ) та внутрішньомежової мембрани.*

Результати. *У підгрупі 1а вихідна гострота зору становила 0,18±0,16, через 6 місяців – 0,19±0,16 (p=0,5), через 12 місяців – 0,22±0,15 logMAR (p=0,07). Зовнішній та внутрішній діаметр ламелярного розриву протягом 12 місяців спостереження статистично збільшився (p=0,037), а мінімальна товщина сітківки зменшилась (p=0,028). У підгрупі 1б вихідна гострота зору становила 0,2±0,18, через 6 місяців – 0,22±0,18 (p=0,08), через 12 місяців – 0,28±0,19 logMAR (p=0,006). У цій підгрупі пацієнтів зовнішній діаметр ламелярного макулярного розриву збільшився протягом 12 місяців спостереження від 769,53±254,01мкм до 821,73±253,78мкм (p=0,04). У 2-й групі до операції гострота зору становила – 0,34±0,27, через 1 місяць після операції – 0,36±0,32 (p=0,67), через 6 місяців – 0,28±0,24 LogMAR (p=0,75). Через 1 місяць після операції – закриття ламелярного макулярного розриву у 18-ти з 19-ти оперованих очей (94,7%).*

Встановлено три механізми формування ламелярного макулярного розриву, обумовлені тракцією та пов'язані з формуванням інтраретинальних кістозних порожнин.

Висновки. *Нативний перебіг дегенеративного ламелярного макулярного розриву характеризується відносною стабільністю, в той час як тракційний ламелярний розрив має виражену тенденцію до погіршення анатомічних параметрів. Виявлені три механізми формування тракційного ламелярного розриву. Хірургічне лікування тракційного ламелярного розриву шляхом проведення операції ЗВЕ з видаленням ЕРМ і внутрішньо-межової мембрани приводить до закриття розривів в 94,7 % випадків.*

Ключові слова*: ламелярний макулярний розрив, сітківка, вітректомія, оптична когерентна томографія, епіретинальна мембрана.*

Introduction

Lamellar macular hole (LMH) is a partial thickness defect of the inner retina in the macula, with the presence of irregular foveal contour and reduced foveal thickness. The term was coined in 1975 by Gass who observed a macular lesion in a pseudophakic eye with cystoid macular edema [1]. It was the advent of optical coherence tomography (OCT) that enabled more detailed studies on the morphology of LMH and the differentiation of the latter from full-thickness macular holes (FTMH) or pseudoholes.

LMHs are generally classified into tractional and degenerative based on the morphological characteristics seen in OCT [2]. Some eyes, however, can share common features with both degenerative and tractional LMHs [3]. A panel of experts [4] reached a consensus on the definitions of three clinical entities: LMH (with the morphological features conforming to degenerative LMH), epiretinal membrane (ERM) foveoschisis (with the morphological features conforming to tractional LMH), and macular pseudohole. Although various OCT-based definitions and classifications of LMH and associated vitreoretinal are available, the pathogenesis remains not completely understood.

Additionally, it is not yet understood whether there are different ways for LMH formation or there is a single pathogenetic pathway including several stages. The panel of experts [4] proposed two major pathophysiologic mechanisms for LMH formation: (1) ERM contraction that causes traction leading to the formation of tractional LMH, and (2) a loss of foveal tissue leading to the formation of degenerative LMH. Lee and colleagues (2022) [5] identified four distinct tractional development pathways of idiopathic LMH and noted that transformation into degenerative condition might be possible after LMH formation.

LMH is a slowly progressive condition that can impair visual acuity (VA) in a subset of patients. As of today, there is no clearly determined consensus as to the timing and indications for the surgical treatment of LMH [6].

The purpose of this study was to investigate the natural course of LMH, identify possible pathways for their formation, and assess the efficacy of surgical treatment for them.

Material and Methods

Study Design

The medical records of patients, who underwent surgical treatment for LMH between January 1, 2019, and December 31, 2025, at the Oculus center, were retrospectively reviewed. This study was approved by the local ethics committee (approval No. 01/01/26). A total of 154 patients were diagnosed with LMH during the period mentioned.

Inclusion criteria were LMH verified by OCT without ocular comorbidity.

Exclusion criteria were follow-up shorter than 12 months; the presence of ocular comorbidity that could affect VA (another vitreoretinal disease, cataract, glaucoma or degenerative myopia); a history of vitrectomy; senile cataract progression during follow-up; phacoemulsification during follow-up; OCT image of low quality; or the presence of macular pseudohole.

Of the 154 patients with LMH, 60 met inclusion and exclusion criteria and were included in the study. LMHs were broadly classified as tractional or degenerative on the basis of initial OCT image.

Tractional LMH was defined as the presence of separation of neurosensory retina between outer plexiform and outer nuclear layers with an intact ellipsoid zone and ERM. Degenerative LMH was defined as the presence of round-edged intraretinal cavitation, damage to the ellipsoidal zone, epiretinal proliferation, and, possibly, a foveal bump (a bulge of retinal tissue in the foveal center, surrounded by foveal cavities [3].

Of the 60 patients included in the study, 41 were managed conservatively (group 1), and 19, surgically (group 2).

Patients from the conservative treatment group were subdivided into two subgroups, subgroup 1a of 18 patients (18 eyes; 43.9%) with tractional LMH, and subgroup 1b of 23 patients (23 eyes; 56.1%) with degenerative LMH. All 19 patients in group 2 had tractional LMH with ERM. In these patients, indication for surgery was progression of morphological OCT changes (increased traction, an increase in the number of cystic cavities or defect size) and either a decrease in visual function over the period of dynamic observation or metamorphopsia at presentation.

Group 1 included 8 men and 33 women with a mean age of 68.37 ± 10.09 years, and group 2, 2 men and 17 women with a mean age of 72.45 ± 11.19 years.

Patients underwent best-corrected visual acuity (BCVA) assessment, Maklakoff tonometry, anterior eye and fundus examination using slit lamp and wide-angle lens (Volk Digital Wide Field lens; Volk Optical, Mentor, OH), and macular OCT imaging (3D OCT-1 Maestro, Topcon Inc., Tokyo, Japan). Patients in the conservative treatment group had follow-up visits at months 3, 6, 9 and 12 of treatment. Patients in the surgical treatment group had follow-up visits at days 1 and 7 and months 1, 3, 6, 9 and 12 after surgery.

OCT parameters assessed

Using the OCT caliper tool, two ophthalmologists measured the minimum retinal thickness (MRT) and inner and outer diameters and depth of LMH. They also evaluated the ellipsoid zone and the presence of ERM or epiretinal proliferation. Central foveal thickness (CFT) was calculated by the OCT software. The central 1-mm circle on the Early Treatment Diabetic Retinopathy Study (ETDRS) grid was used.

MRT was measured as the smallest distance between the retinal pigment epithelium (RPE) and the inner border of the retinal tissue on the fovea on a line perpendicular to the RPE. The outer diameter of LMH was defined as the largest distance measured across the foveal cavity (for degenerative LMH) or the foveoschisis (for tractional LMH) at the level of the outer retina. The inner diameter of LMH was defined as the smallest distance measured across the LMH at the level of the inner retina. The depth of LMH was determined as follows: (1) on the macular scan, a horizontal line was drawn across the LMH at the level of the inner limiting membrane (ILM), (2) a perpendicular was drawn from this line to the RPE, and (3) the depth was determined as the distance along the perpendicular from the horizontal line to the edge of the retina.

Anatomic progression was defined as increasing in the inner and/or outer diameter of the LMH by more than 50 mm.

Surgery technique

Surgically managed patients underwent a 25G three-port pars plana vitrectomy (PPV) (Alcon Constellation vision system; Alcon AG, Geneva, Switzerland) with ERM and ILM peeling. Triamcenolone acetonide was used to facilitate visualization of the vitreous and posterior hyaloid. The posterior hyaloid was peeled off circumferentially to expose the vitreous base. The ERM and ILM were exposed to the Twin dye (Alchimia, Ponte San Nicolò, Italy; composed of 0.18% Trypan Blue + 0.03% Blulife dye) for about 20 s. Eckardt microforceps were used to peel off the ERM as much as possible. The ILM was peeled off over an area of 1.5-2.0 optic disc diameters around the foveal center. Air endotamponade was performed in all cases excluding patients with a single eye operated.

Statistics

VA measurements were recorded as decimals. Results were then converted into logarithm of minimum angle of resolution (logMAR) VA for statistical analysis. MS Excel was used for statistical analysis. Data are presented as mean ± standard deviation (SD). A paired Student's t-test or Wilcoxon test was used for the comparison of two dependent samples. Shapiro-Wilk test was used to test the normality of the data. Independent-sample t-test was used for the comparison of mean values between independent samples. Significance was established at $p < 0.05$.

Results

In subgroup 1a (tractional LMH) of the conservative treatment group, BCVA at baseline, 6 months and 12 months was 0.18 ± 0.16 logMAR (18 eyes), 0.19 ± 0.16 logMAR (18 eyes) ($p = 0.5$), and 0.22 ± 0.15 logMAR (16 eyes) ($p = 0.07$), respectively, indicating the stability of visual function with no statistically significant change over the follow-up period. At baseline, of the 18 eyes with tractional LMH, 16 (88.9%) had OCT evidence of ERM only, 5 (27.8%) had OCT evidence of ERM plus epiretinal proliferation, and 4 (23.2%) had OCT evidence of damage to the ellipsoid zone.

Table 1 shows tractional LMH parameters measured by the OCT at baseline, 6 months and 12 months. In subgroup 1a, the outer and inner diameters of LMH increased significantly from baseline to 12 months ($p = 0.042$ and $p = 0.037$, respectively). There was also a significant change in MRT ($p = 0.028$). Anatomic progression was seen in 55.5% of eyes with tractional LMH.

In subgroup 1b (degenerative LMH), BCVA at baseline, 6 months and 12 months was 0.2 ± 0.18 logMAR (23 eyes), 0.22 ± 0.18 logMAR (23 eyes) ($p = 0.08$), and 0.28 ± 0.19 logMAR (23 eyes) ($p = 0.006$), respectively.

At baseline, of the 23 eyes with tractional LMH, 19 (82.61%) had OCT evidence of epiretinal proliferation

Table 1. Optical coherence tomography parameters (mean ± standard deviation) in patients with conservatively treated tractional lamellar macular holes (subgroup 1a) at baseline and 6 and 12 months

	Baseline (n =18)	6 months (n =18)	12 months (n =18)
Outer diameter, µm	961.33±433.82	1171.11±413.28 p1=0.05	1127.51±443.85 p2=0.042
Inner diameter, µm	437.56±240.27	476.78±240.44 p1=0.017	510.32±220.83 p2=0.037
Hole depth, µm	177.22±40.31	177.33±45.54 p1=0.58	180.61±46.18 p2=0.74
Minimal retinal thickness, µm	149.11±17.36	153.33±19.91 p1=0.026	154.42±19.24 p2=0.028
Central foveal thickness, µm	298.33±45.91	300.56±46.78 p1=0.68	299.5±42.38 p2=0.82

Notes: p1, p value for the difference between baseline and 6 months; p2, p value for the difference between baseline and 12 months; n, number of eyes

only, 3 (13.04%) had OCT evidence of ERM plus epiretinal proliferation, and 16 (69.57%) had OCT evidence of damage to the ellipsoid zone.

Table 2 shows degenerative LMH parameters measured by the OCT at presentation and, 6 and 12 months. In subgroup 1b, from baseline to 12 months, there was a significant increase in the outer diameter of LMH significantly (p = 0.04), but no statistically significant change in other OCT parameters (particularly, the inner diameter and depth of LMH, MRT, and central retinal thickness) was seen. Anatomic progression was seen in 65% of eyes with degenerative LMH.

In group 2 (19 eyes), BCVA at baseline, 1 month and 6 months was 0.34 ± 0.27 logMAR, 0.36 ± 0.32 logMAR (p = 0.67), and 0.28 ± 0.24 logMAR (p = 0.75), respectively.

Additionally, at 1 month, LMH closure was seen in 18 of the 19 eyes (94.7%). LMH, however, was complicated by the formation of an FTMH in one eye (5.3%); this patient received a vitreous cavity revision procedure with C3F8 15% tamponade, resulting in a closure of FTMH and BCVA recovery to baseline.

Moreover, in group 2, at 6 months after PPV, mean CFT decreased from 350.14 ± 75.29 µm to 289.16 ± 56.93 µm (p = 0.016). Fig. 1 shows successful closure of a tractional LMH at one month after surgery.

Table 3 compares tractional LMH in the conservative treatment group (subgroup 1a) and the surgical treatment group (group 2) with regard to baseline OCT parameters. There was no significant difference in the inner diameter of the LMH, outer diameter of the LMH, or MRT (p = 0.15, p = 0.56, and p = 0.51, respectively) between the conservative treatment group and the surgical treatment group. CFT and the depth of the LMH were, however, statistically significantly larger (p = 0.016 and p = 0.007, respectively) in the surgical treatment group than in the conservative treatment group.

There was a significant between-group difference in BCVA at baseline (p = 0.036), but not at 6 months (p = 0.191).

The mechanisms of LMH formation were followed on OCT in 3 of the 60 patients included in the study.

Table 2. Optical coherence tomography parameters (mean ± standard deviation) in patients with conservatively treated degenerative lamellar macular holes (subgroup 1b) at baseline and 6 and 12 months

	Baseline (n =23)	6 months (n =23)	12 months (n =23)
Outer diameter, µm	769.53 ± 254.01	818.87 ± 252.61 p1 = 0.03	821.73 ± 253.78 p2 = 0.04
Inner diameter, µm	601.13 ± 222.33	615.93 ± 235.05 p1 = 0.51	608.36 ± 232.04 p2 = 0.57
Hole depth, µm	157.8 ± 29.03	161.8 ± 34.56 p1 = 0.49	165.73 ± 33.26 p2 = 0.13
Minimal retinal thickness, µm	130.87 ± 32.89	129.4 ± 32.06 p1 = 0.61	130.73 ± 31.76 p2 = 0.76
Central foveal thickness, µm	244.2 ± 38.71	244.07 ± 40.01 p1= 0.32	245 ± 40.3 p2 = 0.27

Notes: p1, p value for the difference between baseline and 6 months; p2, p value for the difference between baseline and 12 months; n, number of eyes

Table 3. Baseline optical coherence tomography parameters (mean ± standard deviation) in patients with conservatively treated (subgroup 1a) versus surgically treated (group 2) tractional lamellar macular holes

	Outer diameter, μm	Inner diameter, μm	Hole depth, μm	Minimal retinal thickness, μm	Central foveal thickness, μm
Group 1a	961.33 ± 433.82	437.56 ± 240.27	177.22 ± 40.31	149.11 ± 17.36	244.2 ± 38.71
Group 2	1189 ± 500.75 p = 0.15	473.14 ± 112.88 p = 0.56	236.57 ± 79.72 p = 0.007	155.14 ± 34.29 p = 0.51	350.14 ± 75.29 p = 0.016

Note: p, p value for difference between group 1a and group 2 in baseline optical coherence tomography parameters

In patient 1, vitreoretinal traction in the central fovea resulted in intraretinal cyst formation with subsequent spontaneous resolution of the traction and the formation of tractional LMH (Fig. 2).

In patient 2, baseline OCT scan showed ERM; subsequently, OCT showed the formation of parofoveals cysts and schisis, and rupture of the medial wall of cystic cavities with the formation of tractional LMH (Fig. 3).

In patient 3, baseline OCT scan showed a dense ERM; subsequently, a round cyst developed in the central fovea, and the rupture of the upper wall of the cyst resulted in the formation of LMH (Fig. 4).

Discussion

The management of LMH is still a challenge for the ophthalmologist, given an insufficiently understood pathogenesis, the course and the absence of clear indications for surgery.

We found that visual function was stable throughout our period of observation of the natural course of trac-

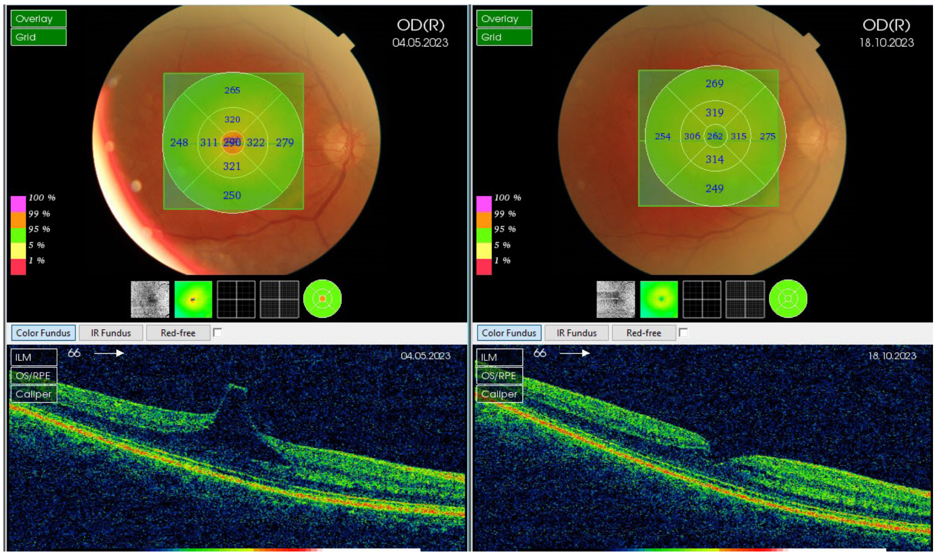

Fig. 1. Surgical treatment of tractional lamellar macular hole. Optical coherence tomography scans obtained prior to and 1 month after pars plana vitrectomy.

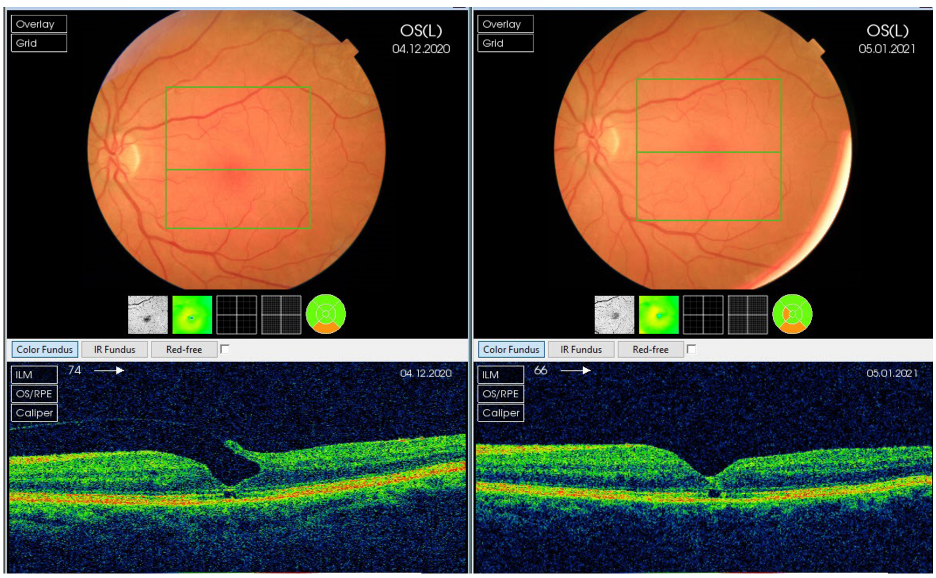

Fig. 2. Optical coherence tomography images illustrating the mechanisms of lamellar macular hole: Vitreoretinal traction in the central fovea resulted in intraretinal cyst formation with subsequent spontaneous resolution of the traction and the formation of tractional lamellar macular hole.

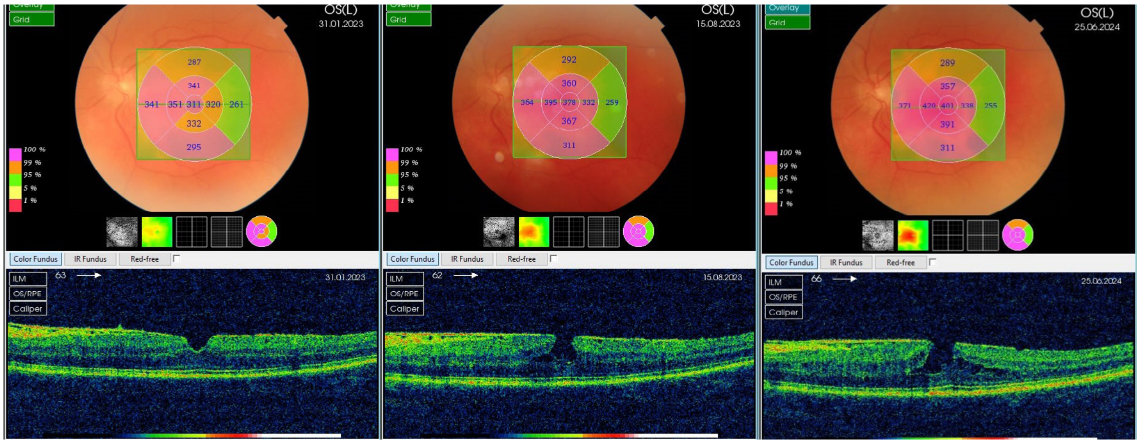

Fig. 3. Optical coherence tomography images illustrating the mechanisms of lamellar macular hole: Epiretinal membrane with the formation of parofoveals cysts and schisis, and rupture of the medial wall of cystic cavities.

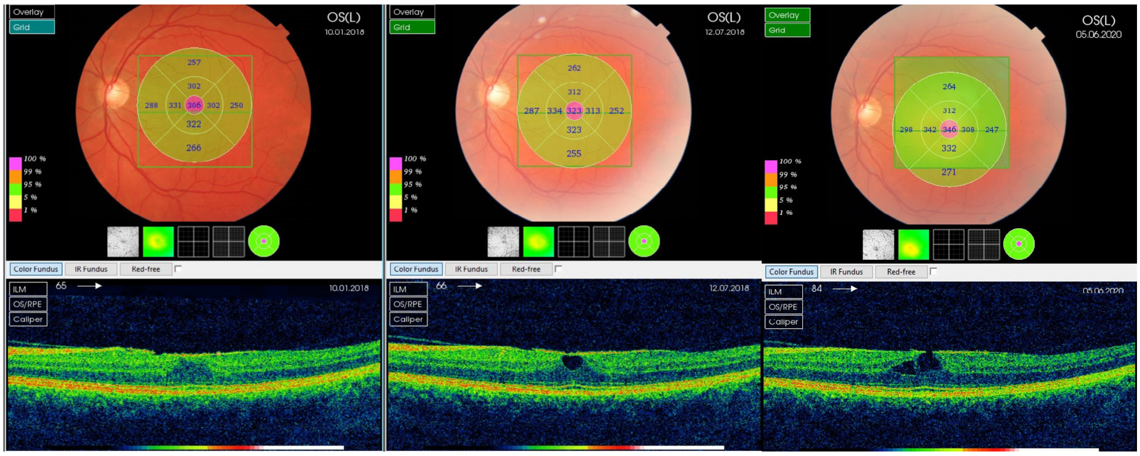

Fig. 4. Optical coherence tomography images illustrating the mechanisms of lamellar macular hole: A dense epiretinal membrane causes foveal flattening with subsequent formation of a cyst, and rupture of the top of the cyst.

tional LMH. Morphological OCT parameters of tractional LMH (e.g., the inner and outer diameters), however, had a statistically significant tendency to progression. This is in agreement with the findings of Kim and colleagues (2026) [7] who evaluated the long-term functional outcomes in patients with LMH without surgical intervention and found that BCVA was stable, but lamellar hole diameter increased significantly. Govetto and colleagues (2016) [3] reviewed the clinical charts and OCT images of patients diagnosed with LMH. They found that, in patients with tractional LMH, BCVA remained stable whereas the inner and outer diameter of LMH increased over the follow-up period.

It is noteworthy that surgery for tractional LMH has allowed us to achieve a closure rate of 94.7%, while the absence of a significant change in BCVA over 6 months of the follow-up may indicate the stabilization of visual function. Our LMH closure rate of 94.7% is similar to that (94.2%) in a study by Figueroa and colleagues (2019) [8].

In the current study, baseline BCVA was worse in the group of eyes that underwent surgery for tractional LMH than in the group of eyes that received conservative treatment for LMH (0.34 ± 0.27 logMAR versus 0.18 ± 0.16 logMAR, respectively). Although BCVA was worse at baseline for the surgical treatment group, there was no significant between-group difference in BCVA at month 6 (0.28 ±0.24 LogMAR and 0.19 ± 0.16 LogMAR, respectively; $p = 0.191$). Therefore, in the surgical treatment group, the BCVA improved compared to baseline. Additionally, at baseline, CFT was larger (350.14 ± 75.29 µm versus 298.33 ± 45.91 µm) and the depth of the LMH was larger (236.57 ± 79.72 µm versus 177.22 ± 40.31 µm) in the surgical treatment group than in subgroup 1a, which definitely had an effect on the final BCVA, and indicates that surgery was conducted in eyes with more apparent morphological alterations and, likely, more apparent tractional component.

With regard to the natural course of tractional LMH, we observed an increase in the outer diameter of LMH over 12 months, and a statistically significant decrease in VA at 12 months. Although Govetto and colleagues (2016) [3] also did find an increase in the outer diameter of LMH over the observation period, the found no significant difference between baseline and final BCVA in the degenerative LMH group.

Based on our observations, we found three possible mechanisms for the formation of LMH. The first mechanism is the formation of an intreretinal cyst, with a subsequent rupture of the internal wall of the cyst due to vitreomacular traction (VMT) resulting in LMH. It is possible that it is an aborted process of FTMH formation referred to by Chen and Lee (2008) [9]. The second mechanism is the rupture of the medial wall of a parafoveal cyst resulting from ERM traction. The third mechanism involves the presence of a dense ERM in the center of the fovea with subsequent foveal flattening, formation of a cyst, and rupture of the internal wall of the cyst. Actually, all the mechanisms of LMH formation observed in the present study were tractional mechanisms associated with either VMT or ERM traction. Lee and colleagues (2022) described four tractional development pathways of idiopathic LMH [5]. In the fourth mechanism, the centrifugal traction force provided by ERM initially caused the elevation of the foveal and parafoveal tissue from the nerve fiber layer to the outer plexiform layer (OPL). There were no intraretinal cysts typically seen in other developmental pathways. Instead, gradual undermining of HFL/OPL layer extended toward outer retina under the persistent ERM traction led to the LMH, confirmed by fundus autofluorescence [5].

A limitation of this study was its retrospective study design. The study was also limited by a small sample size, because LMH (particularly, tractional LMH) may be underdiagnosed in eyes with a sufficiently high VA. Another limitation was that no OCT was performed at the time of LMH detection, which makes it impossible to trace the formation of LMH. Overall, the natural course of degenerative LMH was generally stable, whereas tractional LMH apparently tended to worsen anatomical OCT parameters. We identified three mechanisms of the formation of tractional LMH: vitreoretinal traction with intraretinal cyst formation; ERM with the formation of parofoveals cysts and schisis, and rupture of the medial wall of cystic cavities; and ERM with cyst formation and rupture of the upper wall of the cyst. Surgical treatment (PPV with ERM and ILM removal) for tractional LMH resulted in hole closure with an insubstantial improvement in VA in 94.7% of eyes.

Author Contributions

IlaN: Conceptualization, Methodology, and Data Analysis; UMT: Conceptualization, Methodology, Data Curation, Investigation, Data Interpretation, Writing - original draft preparation, Writing – Review and Editing. All authors reviewed the results and approved the final version of the manuscript.

Funding Sources

The authors declare that no funds, grants or other support was received during the preparation of the manuscript.

Conflict of interest

The authors state that they have no conflict of interest that could influence their view on the subject matter or materials described and discussed in this manuscript.

Disclaimer

The opinions presented in this article are those of the authors and do not necessarily represent those of their institutions.

Ethical Statement

This study involved human subjects and was approved by the Ethics committee of the Oculus medical center (approval No. 01/01/26). Informed consent was not obtained due to the retrospective nature of the study. This study did not include animal experiments.

Data Availability Declaration

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Abbreviations

ERM, epiretinal membrane; OPT, optical coherence tomography; PPV, pars plana vitrectomy; RPE, retinal pigment epithelium; VMT, vitreomacular traction.

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