Graefe's Archive for Clinical and Experimental Ophthalmology (2021) 259:2235–2241
https://doi.org/10.1007/s00417-021-05183-3

RETINAL DISORDERS

Macular holes following vitrectomy for rhegmatogenous retinal detachment: epiretinal proliferation and spontaneous closure of macular holes

Akinori Uemura[1] · Noboru Arimura[1] · Keita Yamakiri[1] · Kazuki Fujiwara[2] · Eri Furue[2] · Taiji Sakamoto[2]

Received: 10 February 2021 / Revised: 20 March 2021 / Accepted: 1 April 2021 / Published online: 20 April 2021

Abstract

Purpose To describe the characteristics and management of full-thickness macular holes (MHs) that develop after pars plana vitrectomy for rhegmatogenous retinal detachment (RD).

Methods Retrospective, interventional, consecutive case series. Patients who developed secondary full-thickness MHs after prior pars plana vitrectomy for RD over a 6-year period were included. The main outcome measures included optical coherence tomography (OCT) findings and the clinical course of full-thickness MHs.

Results A total of 11 eyes of 11 consecutive patients were included in the study. The mean age of the patients was 58.8 years (range, 47–70 years). The median time between RD repair and MH diagnosis was 36 months (range, 1 month–11 years). The fovea was attached to 10 eyes (91%) at the time of RD repair. OCT demonstrated epiretinal proliferation (EP) at the hole margin in 10 eyes (91%). MH spontaneously closed in 7 eyes (63%) but reopened in 5 eyes. A total of 7 eyes (63%) required a vitrectomy to repair the MHs. All MHs were closed at the last follow-up visit.

Conclusion Full-thickness MHs after pars plana vitrectomy for RD have features that are distinct from that of typical idiopathic MH. The presence of EPs is common, and MHs are prone to spontaneous closure and reopening. These findings suggest that EP may be associated with spontaneous hole closure and that long-term follow-up is necessary even if the MHs close spontaneously.

Key Message:

- Macular holes are known to rarely develop following pars plana vitrectomy for rhegmatogenous retinal detachment.
- The presence of epiretinal proliferation around a hole is common, and smaller holes tend to close spontaneously. Epiretinal proliferation may be associated with the spontaneous closure of the hole.
- Long-term follow-up is necessary because the closed hole may potentially reopen.

Keywords Epiretinal proliferation · Macular hole · Optical coherence tomography · Retinal detachment · Spontaneous closure · Vitrectomy

This article is part of a topical collection on Macular Hole.

✉ Akinori Uemura
uemura-a15@kch.kagoshima.jp

[1] Department of Ophthalmology, Kagoshima City Hospital, 37-1 Uearata-cho, Kagoshima-shi, Kagoshima 892-8580, Japan

[2] Department of Ophthalmology, Kagoshima University Graduate School of Medicine and Dental Sciences, Kagoshima, Japan

Introduction

It has been reported that a full-thickness macular hole (MH) may develop in secondary situations such as after rhegmatogenous retinal detachment (RRD) surgery, in the presence of a pre-existing complete posterior vitreous detachment, or even after pars plana vitrectomy (PPV) for retinal disorders [1–4]. The incidence of MH after RRD repair has been

reported to be approximately 1%, which includes patients with scleral buckling and pneumatic retinopexy procedures for retinal reattachment [5–9].

Anteroposterior traction by the posterior hyaloid on the foveal area has been widely accepted as a major causative factor for the development of idiopathic MH. Atypical MHs such as those that occur in the setting of a previous PPV, where there is no anteroposterior vitreous traction, raise questions regarding the pathogenesis of MH formation [1]. In patients with MH formation after PPV for RRD, several possible associated factors, including the presence of epiretinal membrane (ERM), macula-off RD, recurrent RD, and high myopia have been reported [10, 11]. However, the pathogenesis of MH formation in the setting of a previous vitrectomy remains unclear.

While most secondary MHs can be closed by an additional vitrectomy [4, 6–8, 12], spontaneous closure has rarely been reported in eyes with MH after PPV for RRD [9, 11, 13–15]. Due to the small number of patients with spontaneous closure of the MH, the factors that contribute to this remain unknown. Based on our clinical experience, we speculated that spontaneous closure might be more prevalent in MH after PPV for RRD than previously thought. However, there have been few previous reports on the prevalence of spontaneous hole closure and its possible associations.

The association between spontaneous MH closure and epiretinal proliferation (EP) has been reported in patients with MH presenting with EP [16]. EP is a distinct clinical entity from traditional ERM that is classically associated with lamellar MHs [17–20] and is observed on optical coherence tomography (OCT) as a homogenous material with medium reflectivity and varying thickness. Although the presence of EP has been reported in patients with MH after PPV for RRD [15], its prevalence and association with spontaneous hole closure have not been fully elucidated in the literature.

In this study, we report a consecutive case series of patients who developed MH after RRD repair and investigate the characteristics and clinical course of MHs, particularly the prevalence of spontaneous closure of MH and its association with EP.

Method

This study followed the tenets of the Declaration of Helsinki and was approved by the institutional review board of Kagoshima City Hospital. The clinical records of consecutive patients who underwent PPV for RRD and subsequently developed full-thickness MH between April 2014 and March 2020 were reviewed for this retrospective study. Patients with MH detected during PPV or immediately after gas absorption were excluded from the study.

From the medical records of these patients, demographic and clinical data including age, sex, the affected eye, refractive error, best-corrected visual acuity (BCVA) at the time of RRD repair, the status of the macula at RRD repair, surgical procedure for RRD, number of surgeries performed for RRD, the time interval between initial vitrectomy for RD and MH diagnosis, BCVA at time of MH diagnosis, the status of the MH at the final visit were collected, and the follow-up period after MH diagnosis were recorded.

The diagnosis of full-thickness MH was confirmed using spectral-domain OCT (Cirrus; Carl Zeiss Meditech and Spectralis; Heidelberg Engineering, Heidelberg, Germany).

Measurements of the MH size (minimum and base) were performed with the "caliper" function of the Heidelberg instrument. All OCT images were carefully reviewed by at least two independent retinal specialists.

OCT imaging was also used to differentiate EP from classic or typical ERMs. Classical ERM tissue was described as a thinner, irregular, and hyperreflective line on the inner retinal surface, occasionally accompanied by areas of hyporeflective space between the membrane and the inner retina, while EP was defined as thicker preretinal material with homogenous medium reflectivity. The presence of lamellar MH was defined according to the following OCT findings: the presence of irregular foveal contour, separation of the layers of the neurosensory retina, and absence of full-thickness macular defects [21].

The BCVA was recorded at each visit and reported in the Snellen fraction, which was converted into the logarithm of the minimal angle of resolution values for statistical analysis. A Wilcoxon rank-sum test was used for the continuous variables. A p value of less than 0.05 was considered statistically significant.

Results

A total of 11 eyes of 11 consecutive patients with a mean age of 58.8 years (range, 47–70 years) were included in the study. The mean age at diagnosis of RRD was 54.0 years (range, 38–67 years). In the cohort, 5 of the 11 eyes (45%) had high myopia (refractive error greater than − 6 diopters or axial length greater than 26.5 mm). At the time of RRD surgery, the macula was attached to 10 (91%) of the 11 eyes. All patients underwent vitrectomy for primary RRD with or without simultaneous cataract surgery. The vitreous cavity was filled with sulfur hexafluoride (SF6) gas in all eyes. No additional intervention (such as encircling placement, internal limiting membrane [ILM] peeling or use of perfluorocarbon liquid) was performed. In all eyes, the retina was reattached after a single operation. The mean Snellen BCVA was 20/30 at the time of RRD presentation and 20/20 after surgical repair of RRD (Table 1).

Table 1 Patients' baseline characteristics

No	Age	Gender	Macula on/off	BCVA at RD repair	High myopia	Number of RD repair	BCVA post-RD repair
1	58	M	On	20/15	Yes	1	20/15
2	54	F	Off	20/100	Yes	1	20/33
3	68	M	On	20/22	No	1	20/17
4	55	M	On	20/28	No	1	20/20
5	51	F	On	20/22	Yes	1	20/20
6	70	M	On	20/50	No	1	20/17
7	70	F	On	20/20	No	1	20/22
8	56	M	On	20/20	Yes	1	20/15
9	47	M	On	20/20	Yes	1	20/22
10	61	F	On	20/25	No	1	20/20
11	56	M	On	20/20	No	1	20/17

PVD, posterior vitreous detachment; *BCVA*, best-corrected visual acuity; *RD*, retinal detachment

The median interval between RRD repair and MH diagnosis was 36 months (range, 1–137 months). MH was diagnosed more than 1 year after RRD repair in 10 (91%) of the 11 patients. The mean BCVA was 20/35 at the time of MH diagnosis, and 7 (63%) eyes had a visual acuity of at least 20/40 (Table 2). The mean (± standard deviation [SD]) diameter of the MH (minimum linear diameter) was 229 ± 164 µm (range, 88–593 µm). OCT examination revealed ERM in all eyes (100%) and EP in 8 (73%) of the 11 eyes at the time of MH presentation. During the subsequent follow-up period, EPs were found in two additional eyes; therefore, the presence of EPs was confirmed in 10 eyes (91%). All MHs with EP developed more than 12 months after RRD repair. In two eyes, EP was observed on OCT before MH formation (Fig. 1).

Seven (63%) of the 11 MHs in the current study showed at least one episode of spontaneous closure during the follow-up period. The time to spontaneous closure ranged from 1 to 4 months, with a mean of 2 months. The median diameter at MH diagnosis was 135 µm in seven eyes with spontaneous hole closure and 387 µm in four eyes without closure; the difference was statistically significant ($p = 0.023$). Six (86%) of the seven eyes with spontaneous MH closure had OCT evidence of EP.

Among the seven patients with spontaneous closure of MH, five had recurrent MH but two of them closed again spontaneously; therefore, a total of seven eyes underwent a second vitrectomy with ILM peeling and gas injection. At the final visit, MH was closed in all patients and the mean Snellen BCVA was 20/24 (range, 20/40–20/17), with a mean (± SD) follow-up period of 28.6 ± 17.7 months from the time of MH diagnosis (range, 4–68 months) (Table 2). Three of the four eyes with spontaneous MH closure showed the appearance of degenerative lamellar MH (Fig. 2).

Discussion

This consecutive case series included 11 patients who developed full-thickness MHs after vitrectomy for RRD. The results showed at least one episode of spontaneous closure of MH occurred in seven (63%) of the 11 eyes. Furthermore, ten eyes (91%) had EP at the hole margin on OCT; of these, this was seen at the time of MH presentation in eight eyes, and during the follow-up period in two eyes. To the best of our knowledge, this is the first study to show a high frequency of spontaneous hole closure and presence of EP in eyes with MH after vitrectomy for RRD.

EPs were first noted as a thicker or denser ERMs and were described as features specific to lamellar MH [17, 18]. Pang et al. focused their attention on lamellar hole-associated EP, an entity considered to be characteristic of lamellar MH [19]. They suggested that EP originates primarily from the proliferation of Müller cells onto the inner retina. This proliferation is not specific to lamellar MH, as it was also observed in eyes with full-thickness MH and ERM [22]. Although the prevalence of EP in idiopathic MH has been reported to be between 7.9 and 26.5% [23–26], our results showed that EP was observed at a high frequency in secondary MHs after RRD repair. It is difficult to determine whether the MHs occurred before the development of EP or these holes developed as a consequence of EP. Interestingly, we noticed that EPs were present in two eyes before the development of MHs. Several reports have documented the presence of EPs before MH formation in vitrectomized eyes [16, 27]. As EP itself is known not to induce a traction effect such as distortions of the underlying normal retinal tissue, it is unlikely that the presence of EP is directly involved in MH formation [19]. However, it cannot be denied that EP plays an important role in the mechanism of MH formation.

Table 2 Development of the MH and morphological characteristics

No	Interval to MH diagnosis (months)	BCVA at MH diagnosis	EP	ERM	MH size minimum (μ)	Spontaneous closure	Reopen of MH	Surgery for MH	BCVA at MH repair	MH closure	Final BCVA	Follow-up from MH diagnosis (months)
1	80	20/20	Yes	Yes	135	Yes	Yes	Yes	20/50	Yes	20/20	48
2	12	20/28	Yes*	Yes	183	Yes	No	No	NA	Yes	20/33	33
3	38	20/20	Yes*	Yes	88	Yes	Yes +	No	NA	Yes	20/17	38
4	1	20/50	No	Yes	211	Yes	Yes	Yes	20/200	Yes	20/40	68
5	36	20/40	Yes	Yes	147	Yes	Yes +	No	NA	Yes	20/22	16
6	137	20/33	Yes	Yes	111	Yes	Yes	Yes	20/66	Yes	20/33	20
7	33	NA	Yes	Yes	593	No	-	Yes	20/66	Yes	20/20	37
8	33	20/22	Yes	Yes	237	No	-	Yes	20/40	Yes	20/17	14
9	119	20/66	Yes	Yes	173	No	-	Yes	20/133	Yes	20/40	4
10	104	20/50	Yes	Yes	538	No	-	Yes	20/50	Yes	20/25	25
11	23	20/25	Yes*	Yes	109	Yes	No	No	NA	Yes	20/17	12

*EP was observed during follow-up, not at the time of MH diagnosis

+There was a reopening of MH, but it closed again spontaneously

MH, macular hole; *BCVA*, best-corrected visual acuity; *EP*, epiretinal proliferation; *ERM*, epiretinal membrane; *NA*, not available

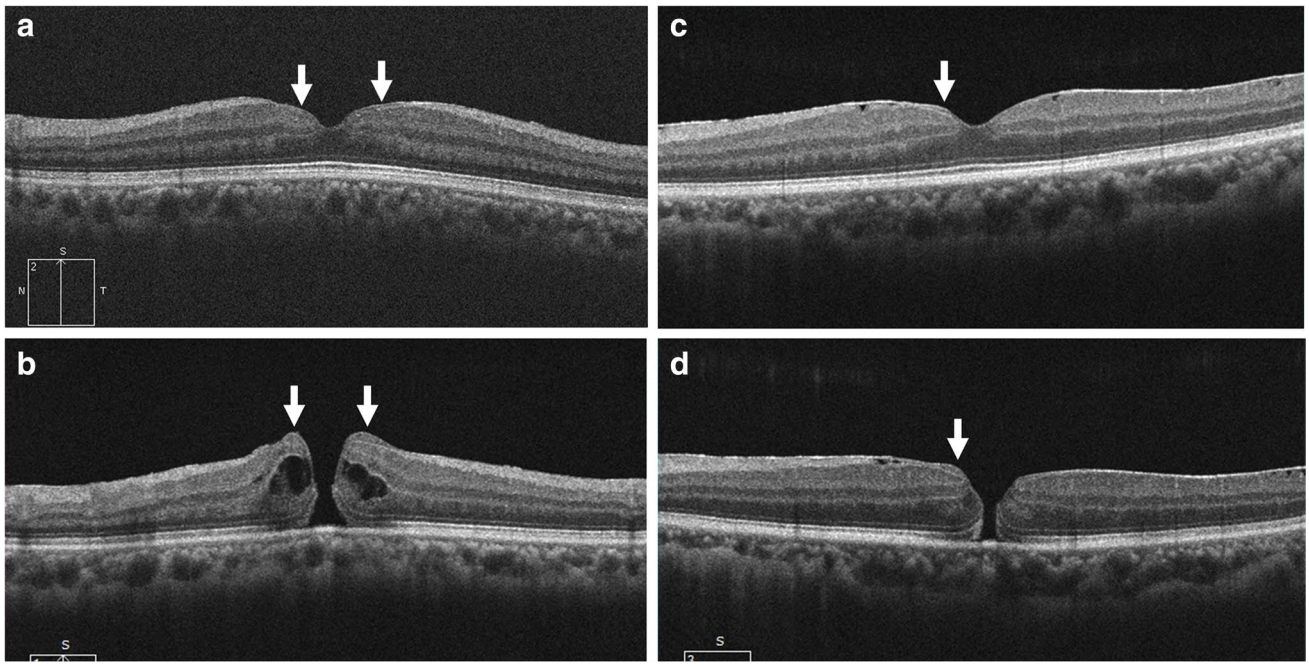

Fig. 1 The presence of epiretinal proliferation (EP) before the development of MH. **a** (Case 11) OCT image at 12 months after vitrectomy for RD. EP was present at the fovea (arrow) with an extensive classic epiretinal membrane (ERM). **b** (Case 11) Two years after vitrectomy for RD. Small MH developed with OCT evidence of perifoveal EP (arrow). **c** (Case 8) OCT image at 12 months after vitrectomy for RD. EP was present over the fovea (arrow). **d** (Case 8) Thirty-three months after vitrectomy for RD, an MH developed with OCT evidence of EP at the edge of the hole (arrow)

The current study also found a high rate of spontaneous MH closure. There have been few reports regarding the spontaneous closure of MH after RRD repair. Fabian et al. reported that one of seven eyes with MH after PPV for RRD experienced spontaneous hole closure [9]. Khurana et al. reported that in two of 25 eyes, MHs spontaneously closed [11]. Reopening after spontaneous closure of the MH was observed in 5 eyes in our series, and two of them had MH reclosure. There have been similar reports of repeated opening and spontaneous closure of MHs after vitrectomy [14, 15]. These findings indicate that there is a tendency for spontaneous closure and reopening in MHs that present after RRD repair. When spontaneous hole closure occurs, the macular structure usually assumes a lamellar MH configuration [16]. In fact, three of four eyes with spontaneous closure of MH at the last visit showed a lamellar MH appearance. Although these facts suggest that MH may be derived from the previously existing lamellar MH, none of the eyes in our series showed presence of lamellar MH before MH formation.

The relationship between EP and spontaneous MH closure is poorly understood. In the current study, four of the seven eyes with spontaneous hole closure had EP at MH diagnosis, and six of 10 eyes with EP had spontaneous MH closure. Previous reports have also shown that spontaneous closure is not uncommon in MH with EP, and when spontaneous closure occurs, tissues with density similar to that of EP were observed filling the hole [16, 22]. These results indicate that EP may play an important role in the spontaneous closure of MH. Bringmann et al. suggested that spontaneous closure of small MHs is mediated by the active mechanisms of Müller cells [28]. Since EP involves retinal glial cells, specifically Müller cells [20], the migration and proliferation of Müller cells might be associated with the formation of EP leading to spontaneous hole closure [22].

There is no consensus on when to operate on an MH that does not close spontaneously. The average time from MH diagnosis to spontaneous closure was 2 months in the current study. Other reports have also shown spontaneous hole closure within 1–2 months [9, 13–15, 27]. Additionally, eyes with spontaneous hole closure had smaller hole sizes than that of eyes without closure. Given that smaller MHs are more likely to close spontaneously and have relatively good vision, immediate surgical intervention may be avoided [14, 27]. Nevertheless, if MHs do not close spontaneously within 2 or 3 months, or if the MH size increases with deterioration of vision, surgical intervention may be indicated. However, in the present study, three of seven eyes with spontaneously closed MHs eventually required vitrectomy due to reopening of the hole. Even in eyes with MHs that close spontaneously, there is still a risk that the hole will reopen in the future.

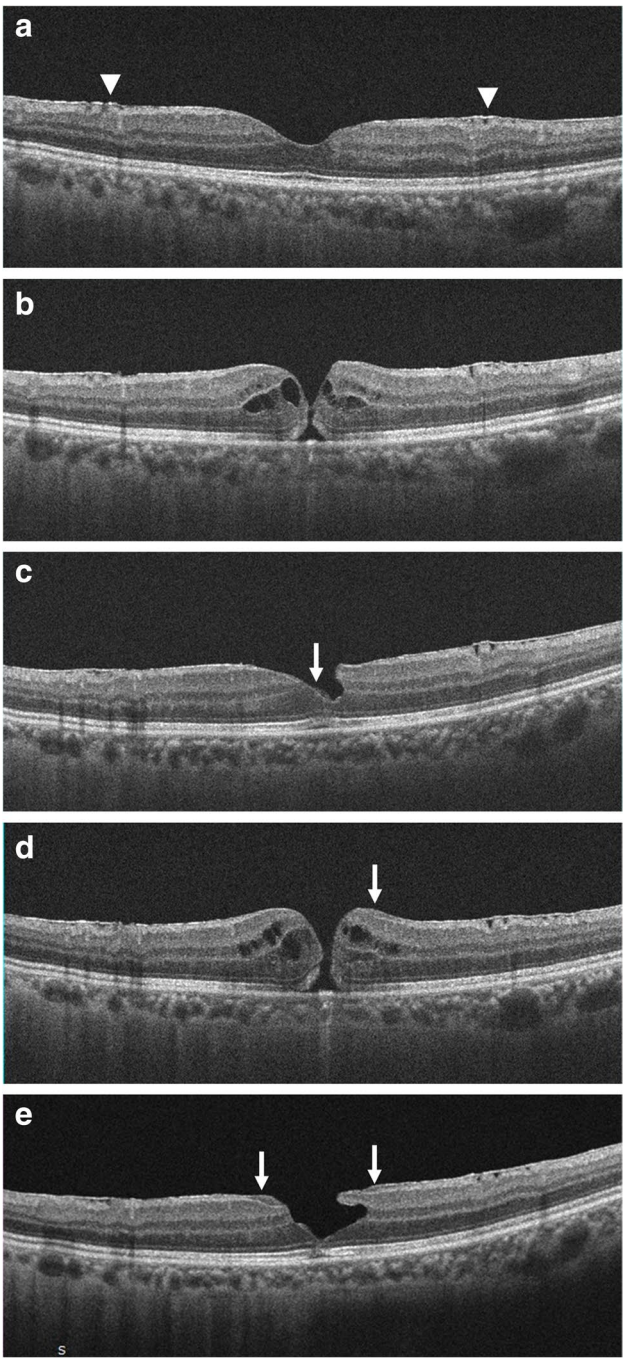

Fig. 2 The sequence of OCT images over time from case 3 with spontaneous closure of a MH after vitrectomy for RD. **a** OCT image at 2 years after vitrectomy for RD showing high retinal surface reflectivity from ERM (arrowhead). **b** Thirty-eight months after vitrectomy. A small full-thickness MH with cystic cavities developed. Vision was 20/20. **c** Forty months after vitrectomy. The hole closed with OCT evidence of EP (arrow). **d** Four months later, the hole reopened, with persistent EP at the hole edge (arrow). **e** Three years after the initial development of MH. OCT showing a lamellar MH appearance with dehiscence of the outer retinal layer. Vision was 20/20

This indicates that early vitrectomy may be an alternative to waiting for spontaneous closure [29].

There are several limitations to the present study. The study's retrospective nature, the small number of patients, and the variable duration of follow-up might have limited the power of the results to support our statement. Moreover, as not all patients were routinely examined after RRD repair, the time interval between RRD repair and MH diagnosis may have been overestimated. Furthermore, although four of the 11 eyes in the present study had spontaneous hole closure at the last visit, we cannot eliminate the possibility that these MHs will reopen in the future.

In conclusion, our study demonstrates that the presence of EP was common in MHs occurring after vitrectomy for RRD. In addition, although spontaneous closure of MH was observed in more than half of the eyes, most of these holes reopened and eventually required surgery. These findings suggest that EP may be associated with spontaneous closure of MH and that long-term follow-up is necessary, regardless of whether the MH closes spontaneously.

Declarations

Ethical approval This study was conducted with the approval of the institutional review board (IRB) of the Kagoshima City Hospital. All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was not necessary for the analysis of the medical records due to the retrospective design of the study.

Conflict of interest The authors declare no competing interests.

References

1. Smiddy WE (1993) Atypical presentations of macular holes. Arch Ophthalmol 111:626–631
2. Gordon LW, Glaser BM, Ie D, Thompson JT, Sjaarda RN (1995) Full-thickness macular hole formation in eyes with a pre-existing complete posterior vitreous detachment. Ophthalmology 102:1702–1705
3. Lipham WJ, Smiddy WE (1997) Idiopathic macular hole following vitrectomy: implications for pathogenesis. Ophthalmic Surg Lasers 28:633–639
4. Lee SH, Park KH, Kim JH, Heo JW, Yu HG, Yu YS, Chung H (2010) Secondary macular hole formation after vitrectomy. Retina 30:1072–1077
5. Brown GC (1988) Macular hole following rhegmatogenous retinal detachment repair. Arch Ophthalmol 106:765–766
6. Moshfeghi AA, Salam GA, Deramo VA, Shakin EP, Ferrone PJ, Shakin JL, Fastenberg DM (2003) Management of macular holes that develop after retinal detachment repair. Am J Ophthalmol 136:895–899

7. Benzerroug M, Genevois O, Siahmed K, Nasser Z, Muraine M, Brasseur G (2008) Results of surgery on macular holes that develop after rhegmatogenous retinal detachment. Br J Ophthalmol 92:217–219
8. Garcia-Arumi J, Boixadera A, Martinez-Castillo V, Zapata MA, Fonollosa A, Corcostegui B (2011) Macular holes after rhegmatogenous retinal detachment repair: surgical management and functional outcome. Retina 31:1777–1782
9. Fabian ID, Moisseiev E, Moisseiev J, Moroz I, Barak A, Alhalel A (2012) Macular hole after vitrectomy for primary rhegmatogenous retinal detachment. Retina 32:511–519
10. Medina CA, Ortiz AG, Relhan N, Smiddy WE, Townsend JH, Flynn HW Jr (2017) Macular hole after pars plana vitrectomy for rhegmatogenous retinal detachment. Retina 37:1065–1072
11. Khurana RN, Wykoff CC, Bansal AS, Akiyama K, Palmer JD, Chen E, Chang LK, Major JC Jr, Wu C, Wang R, Croft DE, Wong TP (2017) The association of epiretinal membrane with macular hole formation after rhegmatogenous retinal detachment repair. Retina 37:1073–1078
12. Kumagai K, Ogino N, Furukawa M, Larson E, Uemura A (2008) Surgical outcomes for patients who develop macular holes after pars plana vitrectomy. Am J Ophthalmol 145:1077–1080
13. Kim JY, Park SP (2015) Macular hole formation and spontaneous closure after vitrectomy for rhegmatogenous retinal detachment documented by spectral-domain optical coherence tomography: case report and literature review. Indian J Ophthalmol 63:791–793
14. Sridhar J, Townsend JH, Rachitskaya AV (2017) Rapid macula hole formation, spontaneous closure, and reopening after pars plana vitrectomy for macular-sparing retinal detachment. Retin Cases Brief Rep 11:163–165
15. Mori T, Kitamura S, Sakaguchi H, Nishida K, Ohji M (2018) Two cases of repeating recurrences and spontaneous closures of macular holes in vitrectomized eyes. Jpn J Ophthalmol 62:467–472
16. Lai TT, Chen SN, Yang CM (2016) Epiretinal proliferation in lamellar macular holes and full-thickness macular holes: clinical and surgical findings. Graefes Arch Clin Exp Ophthalmol 254:629–638
17. Witkin AJ, Ko TH, Fujimoto JG, Schuman JS, Baumal CR, Rogers AH, Reichel E, Duker JS (2006) Redefining lamellar holes and the vitreomacular interface: an ultrahigh-resolution optical coherence tomography study. Ophthalmology 113:388–397
18. Parolini B, Schumann RG, Cereda MG, Haritoglou C, Pertile G (2011) Lamellar macular hole: a clinicopathologic correlation of surgically excised epiretinal membranes. Invest Ophthalmol Vis Sci 52:9074–9083
19. Pang CE, Spaide RF, Freund KB (2014) Epiretinal proliferation seen in association with lamellar macular holes: a distinct clinical entity. Retina 34:1513–1523
20. Pang CE, Maberley DA, Freund KB, White VA, Rasmussen S, To E, Matsubara JA (2016) Lamellar hole-associated epiretinal proliferation: a clinicopathologic correlation. Retina 36:1408–1412
21. Duker JS, Kaiser PK, Binder S, de Smet MD, Gaudric A, Reichel E, Sadda SR, Sebag J, Spaide RF, Stalmans P (2013) The International Vitreomacular Traction Study Group classification of vitreomacular adhesion, traction, and macular hole. Ophthalmology 120:2611–2619
22. Tsai CY, Hsieh YT, Yang CM (2016) Epiretinal membrane-induced full-thickness macular holes: The clinical features and surgical outcomes. Retina 36:1679–1687
23. Itoh Y, Levison AL, Kaiser PK, Srivastava SK, Singh RP, Ehlers JP (2016) Prevalence and characteristics of hyporeflective preretinal tissue in vitreomacular interface disorders. Br J Ophthalmol 100:399–404
24. Lee Kim E, Weiner AJ, Ung C, Roh M, Wang J, Lee IJ, Huang NT, Stem M, Dahrouj M, Eliott D, Vavvas DG, Young LHY, Williams GA, Garretson BR, Kim IK, Hassan TS, Mukai S, Ruby AJ, Faia LJ, Capone A Jr, Comander J, Kim LA, Wu DM, Drenser KA, Woodward MA, Wolfe JD, Yonekawa Y (2019) Characterization of epiretinal proliferation in full-thickness macular holes and effects on surgical outcomes. Ophthalmol Retina 3:694–702
25. Bae K, Lee SM, Kang SW, Kim ES, Yu SY, Kim KT (2019) Atypical epiretinal tissue in full-thickness macular holes: pathogenic and prognostic significance. Br J Ophthalmol 103:251–256
26. Takahashi H, Inoue M, Itoh Y, Koto T, Hirota K, Kita Y, Hirakata A (2020) Macular dehiscence-associated epiretinal proliferation in eyes with full-thickness macular hole. Retina 40:273–281
27. Zhang W, Grewal DS, Jaffe GJ, Mahmoud TH, Fekrat S (2017) Spontaneous closure of full-thickness macular hole with epiretinal membrane in vitrectomized eyes: case series and review of literature. Ophthalmic Surg Lasers Imaging Retina 48:183–190
28. Bringmann A, Duncker T, Jochmann C, Barth T, Duncker GIW, Wiedemann P (2020) Spontaneous closure of small full-thickness macular holes: presumed role of Müller cells. Acta Ophthalmol 98:e447–e456
29. Chen HJ, Jin Y, Shen LJ, Wang Y, Li ZY, Fang XY, Wang ZL, Huang XD, Wang ZJ, Ma ZZ (2019) Traumatic macular hole study: a multicenter comparative study between immediate vitrectomy and six-month observation for spontaneous closure. Ann Transl Med 7:726