Rapid macular hole formation and closure in a vitrectomized eye following rhegmatogenous retinal detachment repair

Ratnesh Ranjan, George Joseph Manayath, Udayasree Avadhani, Venkatapathy Narendran

Abstract:
The development of a full-thickness macular hole (FTMH) after pars plana vitrectomy (PPV) for rhegmatogenous retinal detachment (RRD) repair is a rare occurrence. We report the first case of rapid MH formation with internal limiting membrane (ILM)-like hyper-reflective bridging membrane under silicone oil (SO) meniscus, noted on the first postoperative day (POD) postvitrectomy for RRD. A 57-year-old man presented with best-corrected visual acuity (BCVA) of perception of light with immature cataract and total RRD in the left eye. He underwent cataract extraction with intraocular lens implantation with uneventful PPV, endolaser to primary break, and 360° periphery followed by injection of 1000 Cst SO. On the first POD, retina was attached in all quadrants with SO in situ. However, a dark red spot was noted at macula which was confirmed as FTMH with a bridging linear hyper-reflectivity under SO meniscus on optical coherence tomography (OCT). Repeat OCT, at 2 weeks and 3 months, revealed closed MH under the SO meniscus with BCVA improving to 6/60. This is the first reported case of very rapid FTMH formation with an ILM-like linear hyper-reflectivity following vitrectomy for RRD and highlights the possible contributory role of multiple nonconventional factors for rapid FTMH development and fibrinous membrane-assisted closure under SO tamponade.

Keywords:
Full-thickness macular hole formation, rhegmatogenous retinal detachment, silicon oil tamponade

Introduction

The development of a macular hole (MH) after rhegmatogenous retinal detachment (RRD) repair is a rare occurrence and has been reported after scleral buckling (SB), pneumatic retinopexy, and pars plana vitrectomy (PPV). The prevalence of MH development after RRD repair varies from 0.5% to 2%, and the majority of MHs have been reported following SB surgery. The pathophysiology is believed to be due to the existence of vitreous in eyes undergoing SB, which causes anteroposterior vitreofoveal traction after surgery, thereby facilitating the MH development. However, MH formation following vitrectomy for RRD repair, where vitreofoveal traction is deemed to be absent or minimal, remains a conundrum. Most of the MHs in vitrectomized eyes have been reported to have delayed onset, after several weeks to months post-PPV. The contraction of subclinical residual cortical vitreous, or gradual glial tissue proliferation followed by contraction, has been postulated to cause delayed-onset MH after PPV for RRD. We report a case of rapid-onset full-thickness MH (FTMH) following vitrectomy for RD repair, noted on the 1st postoperative day (POD), and an early closure of the MH under silicone oil (SO) tamponade.

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A 57-year-old man presented with the complaint of sudden-onset loss of vision in his left eye for 3 months. His best-corrected visual acuity (BCVA) was 6/6 in the right eye and perception of light in the left eye. Anterior-segment examination showed immature cataract (nuclear sclerosis Grade II) in both eyes. Fundus examination showed normal findings in the right eye and total RRD with a single break at 4 o’clock hour position with posterior vitreous detachment in the left eye. The patient underwent cataract extraction with intraocular lens implantation in the left eye combined with 25G micro-incision PPV, perfluorocarbon liquid (PFCL) injection, vitreous-base shaving, endolaser to primary break, and 360° periphery followed by fluid–air exchange and injection of 1000 Cst SO.

On the first POD, retina was attached in all quadrants with SO in situ. However, a round, dark red spot suggestive of FTMH was noted at the macula. Optical coherence tomography (OCT) confirmed a FTMH with an internal limiting membrane (ILM)-like linear hyper-reflectivity bridging the MH, under the SO meniscus [Figure 1]. The patient was kept under observation with standard postoperative medications. Postoperatively at 2 weeks, his BCVA improved to 6/60 and retina remained attached with SO in situ. Repeat OCT imaging revealed a closed MH under the SO meniscus, with a central macular thickness of 159 µm [Figure 2a]. During his last visit at 3 months, MH remained closed as confirmed on OCT [Figure 2b]. On both occasions, the bridging hyper-reflective membrane was absent on subsequent scans. The patient was advised SO removal and is yet to undergo the procedure.

**Discussion**

Lee et al. proposed two potential mechanisms for the pathogenesis of postvitrectomy secondary MH formation as epiretinal membrane-related “tangential traction type” and cystoid macular edema (CME)-related “cystoid degeneration type.”[4] Tangential traction by remnant vitreous contraction or by retinal glial tissue proliferation and contraction, over the ILM, may play a role in the development of a FTMH. In the cystoid degeneration type, multiple small cysts merge with one another to form a large cyst, which ruptures to form a FTMH.[4,5]

Rahman et al. reported five cases of FTMH in eyes that had previously undergone vitrectomy for RD, diagnosed from 2 weeks to 4 months post-PPV. They suggested residual undetected preretinal cortical vitreous contraction inducing tangential traction as the plausible cause.[5] Shibata et al. reported a delay of 29.6 ± 26.6 months for MH formation in vitrectomized eyes. They concluded that glial migration and proliferation followed by the contraction of glial plaques or membranes requires a longer time in vitrectomized eyes.[2] However, early development of MH on the first POD and the absence of any residual cortical vitreous noted on high-resolution spectral domain-OCT ruled out this commonly suggested “tangential traction-type” mechanism of MH formation in our case.

To the best of our knowledge, this is the first report of MH development documented on the first POD following vitrectomy for RRD. In the largest series reported by Schlenker et al., among the 18 cases of MH development in vitrectomized eyes post-RD repair, 2 cases developed MH as early as 2 days postvitrectomy, while 5 more cases...
developed within 5–10 days. A degenerative form of CME was advocated as an inciting factor for early MH formation. They also suggested that reattachment of such swollen and distorted macula may stretch the fovea and thereby may change the tractional dynamics, enough to induce spontaneous umbo dehiscence. Degenerative changes and thinning of the macula have been reported in long-standing macula-off RRD due to hypoxia and deficiency of nutrients provided by retinal pigment epithelium–choriocapillaris complex, which may also predispose to MH formation. This explains the fact that MH formation following RRD repair is more common in macula-off cases. Similarly, our case also had a long-standing macula-off RRD with a duration of 3 months.

Iatrogenic trauma during vitrectomy might be another possible mechanism for triggering the MH formation, especially for cases seen in immediate postoperative period. Lee et al. suggested that an interval of 3 months between the primary vitrectomy and MH formation in 60% of patients indicates a possible iatrogenic mechanism. Tsilimbaris et al. also advocated that the proximity of MH diagnosis to vitrectomy for RRD would indicate the role of possible surgical manipulations and consequent inflammatory processes. The iatrogenic mechanism of MH formation includes mechanical trauma resulting from forceful jet of PFCL or SO directed inadvertently over the fovea, or accidental instrument touch, phototoxicity from endoilluminator, and SO toxicity to retinal tissue. However, in our case, mechanical trauma to fovea by inadvertent injection of PFCL or SO jet, or by direct instrument touch, was absent intraoperatively. Phototoxicity can cause immediate damage to retinal tissue. However, no case of phototoxicity-induced MH following the use of endoilluminator in extramacular surgeries has been reported so far. Studies have shown significant thinning of inner retinal layers in vitrectomized eyes with SO tamponade compared to gas-filled eyes, but such changes were noted several months after SO removal.

In our case, FTMH was noted on the first POD along with a hyper-reflective membrane with variable thicknesses over the inner retinal layers resembling ILM, beneath the SO meniscus. This membrane disappeared after 2 weeks of follow-up on anti-inflammatory medications. In addition, this hyper-reflective membrane bridging the MH had increased reflectivity beneath it compared to the area just above it, i.e., below the SO interface, and hence it could be an inflammatory fibrinous membrane rather than an intact ILM. These findings suggest toward an inflammatory pathogenic process for rapid MH development. Though the exact pathogenesis of acute postoperative MH development in this case is unclear, literature supports that this could be a manifestation of multiple contributory factors. Coalescence of microcystic spaces, ischemia-induced retinal tissue damage, phototoxicity, laser-induced inflammation, and/or SO-related inflammation and toxicity could be the contributory factors.

MH closure under SO tamponade, even without ILM peeling, has been reported in various studies. Karia et al. noted MH closure in 90% eyes during follow-up at 6 weeks after PPV and air–SO exchange without ILM peeling. In our case, MH closure was noted at 2 weeks postoperatively. The closure was probably assisted by the scaffold of fibrinous membrane or fibrin clot under which glial tissue proliferated to fill the hole.

Conclusion

We describe a heretofore unreported case of rapid FTMH formation with a bridging ILM-like reflectivity under SO meniscus, on the first POD of vitrectomy for RRD, followed by early closure under SO tamponade. The rapid MH development in this case highlights the possible contributory role of multiple nonconventional mechanisms such as cystoid macular changes, ischemic retinal tissue damage, surgical manipulation, and postsurgical inflammation. It is important to understand and recognize early MH formation as a postoperative complication of vitrectomy surgery for RRD and its natural course.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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