Macular hole formation and spontaneous closure after vitrectomy for rhegmatogenous retinal detachment documented by spectral-domain optical coherence tomography: Case report and literature review

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This case report describes macular hole (MH) formation and spontaneous closure after vitrectomy for rhegmatogenous retinal detachment (RRD) repair. A 58-year-old man referred with a macula-off superior RRD, in whom vitrectomy was performed. MH with vitreomacular traction (VMT) caused by the posterior vitreous cortex remnants developed 2 weeks after vitrectomy. Four weeks postoperatively, optical coherence tomography revealed resolution of the VMT and spontaneous closure of MH without providing any treatment. This is the first report of an MH formation and spontaneous closure after vitrectomy for RRD. This suggests that the VMT mediated by the posterior vitreous cortex remnants has an important role in the development of secondary MH.

Key words: Macular hole, optical coherence tomography, posterior vitreous cortex, rhegmatogenous retinal detachment, spontaneous closure, vitrectomy, vitreomacular traction

Macular hole (MH) develops from tangential anteroposterior traction by the vitreous.[1] This is supported by histopathological studies and optical coherence tomography (OCT).[2,3] In several diseases, MH may develop after vitrectomy.[4]

Brown first reported secondary MH after rhegmatogenous retinal detachment (RRD) repair in 1998.[5] Previous several studies described that MH can develop after vitrectomy.[6,7] However, we found no previous reports on MH formation and spontaneous closure after vitrectomy for RRD.

This report describes the first description of MH and spontaneous closure after vitrectomy for RRD with clear documentation by OCT and provides an understanding of the mechanisms of secondary MH after vitrectomy.

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Case Report

A 58-year-old man with decreased vision (20/200) and inferior visual field defect in his left eye referred to our hospital. Fundoscopy, OCT, and B-scan ultrasonography showed a macula-off superior RRD with a superior horseshoe-like tear [Fig. 1]. The Pars plana vitrectomy (23 gauge) was performed after phacoemulsification and intraocular lens implantation. At the end of the surgery, fluid-air exchange, endolaser photocoagulation, and gas tamponade (SF6, 20%) were performed by the author. The patient was instructed to maintain a supine position for 7 postoperative days.

After 2 weeks, the postoperative best-corrected visual acuity (BCVA) had decreased to 20/400, which was lower than the preoperative BCVA, even though the entire retina was attached. OCT showed an MH with vitreomacular traction (VMT) caused by the posterior vitreous cortex remnants [Fig. 2]. We decided to monitor the patient's progress without providing any other treatment.

Four weeks after the vitrectomy, OCT revealed spontaneous resolution of the VMT as well as the posterior vitreous cortex remnants and MH [Fig. 3]. Further, his vision improved to 20/100. Finally, the MH had completely closed [Fig. 3] and his BCVA was 20/40 after 6 weeks.

Discussion

The development of MH after RRD repair is a rare occurrence. The prevalence of the development of secondary MH after RRD repair ranges from 0.24% to 0.5% in literature.[5,7] Brown first reported secondary MH after RRD.[5] Recent studies reported secondary MH formation after vitrectomy for RRD repair.[6,7]

The mechanism of secondary MH after vitrectomy for RRD is not well known. Several studies support at least 2 pathogenic mechanisms for MH formation in the vitrectomized eyes.[5-8] One mechanism is related to cystoid macular edema, without vitreoretinal traction. In this mechanism, cystoid degeneration develops, the small cysts merge together to form a larger cyst, and the larger cyst can directly rupture to form a full-thickness MH.[7,8] The other mechanism is vitreofoveal tangential traction, which may play a role in the development of full-thickness MH.[4,6,7] The vitreofoveal traction such as an epiretinal membrane (ERM) results in a subfoveal cyst, which subsequently develops into a full-thickness MH. ERM was found to be one of the underlying pathologies in the development of MH after vitrectomy.[4,6] Kumagai *et al*.[6] reported 47 cases of secondary MH after vitrectomy, showing

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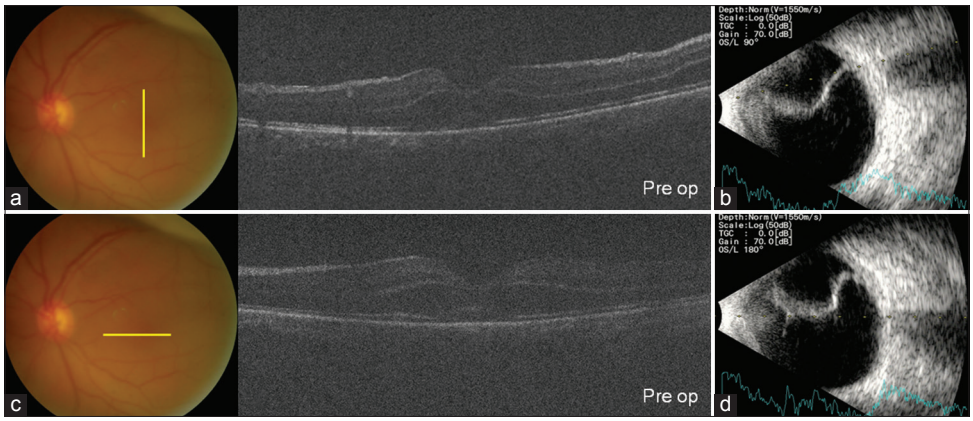

Figure 1: Preoperative fundus photograph, optical coherence tomography (a and c), and B-scan ultrasonography (b and d) superior retinal detachment with vitreous haziness due to vitreous hemorrhage and cataract

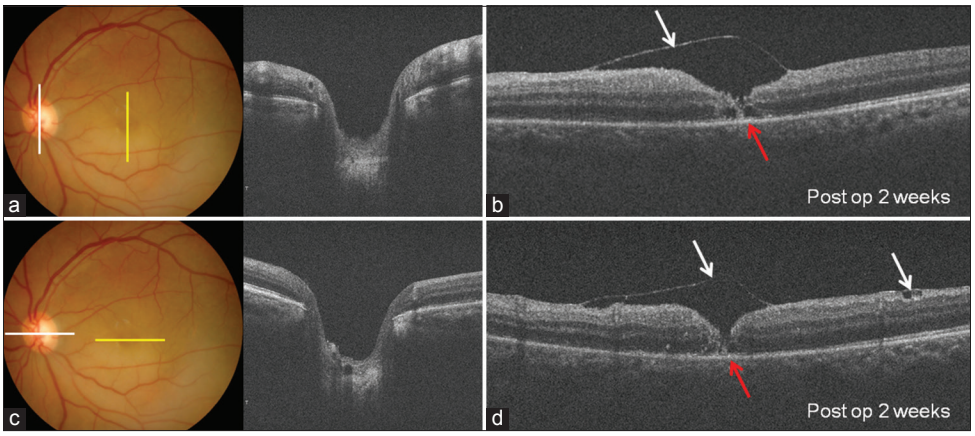

Figure 2: Two weeks after vitrectomy, the posterior vitreous cortex around the disc region (a and c) was removed, but the remnant posterior vitreous cortex and vitreomacular traction remained present in the macular region (white arrow) (b and d). Along with the posterior vitreous cortex remnants, a macular hole was found (red arrow) (b and d)

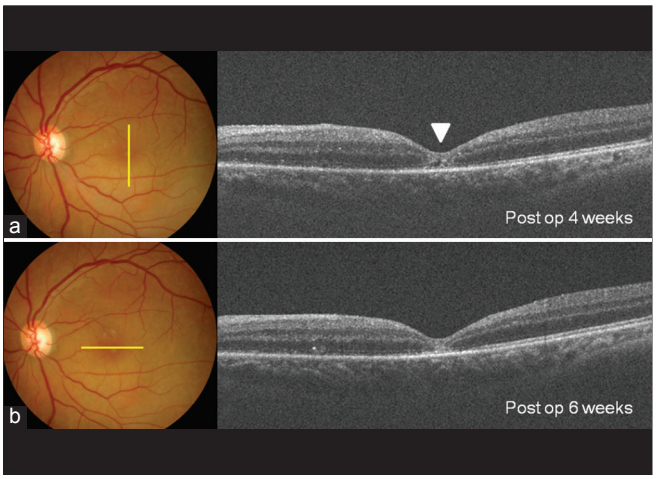

Figure 3: Four weeks after the surgery, the remnant posterior vitreous cortex and vitreomacular traction had regressed spontaneously (a). The white arrowhead indicates the closed macular hole with bridging (a). At 6 postoperative weeks, the macular hole had completely closed (b)

that all eyes diagnosed with secondary MH had an ERM or membrane-like tissue.

This is similar to the proposed mechanism for the MH related VMT. Vitreomacular adhesion is observed after partial posterior vitreous detachment (PVD), when a portion of the posterior vitreous remains attached to the macula. The tangential contraction of the vitreous cortex remnants layer can lead to macular distortion and edema and to the formation of MH.[9] Recent studies have reported that removal of VMT through enzymatic vitreolysis can assist MH closure.[9,10]

In this case, as shown in Figs. 2 and 3, the postvitreous cortex around the disc region was removed, but due to insufficient PVD induction, remnant posterior vitreous cortex remained in the macular region. We believe that induced VMT was the cause of MH and spontaneous removal of VMT lead to the closing of MH.

Summary

Similar mechanisms could have caused secondary MH after vitrectomy in our case. VMT by the posterior vitreous cortex remnants plays an important role in the development of secondary MH, and the release of VMT may have been the main reason for the eventual closure of the MH. Our findings conclusively provide an understanding of the mechanisms of secondary MH after vitrectomy.

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Conflicts of interest

There are no conflicts of interest.

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