Case Report

Closure of Recalcitrant Macular Hole after Choroidal Neovascularization

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Abstract

Purpose: To report the closure of a recalcitrant macular hole (MH) following the development of choroidal neovascularization.

Methods: A 67-year-old female patient in this case report was diagnosed with a MH and operated twice, but anatomical closure of MH could not be achieved. The patient was followed up without further treatment, as she rejected any additional procedure.

Results: Six months later, a lesion consistent with choroidal neovascularization appeared in the central macula, and the recalcitrant MH closed spontaneously. The MH defect remained closed in the following years.

Conclusion: Besides being a new example of the presence of choroidal neovascularization after MH surgery, the most important aspect of this case report is to report the closure of a recalcitrant MH following the development of choroidal neovascularization.

Keywords: Choroidal neovascularization, Macular hole, Vitrectomy

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Introduction

Macular hole (MH) is one of the conditions associated with the abnormal posterior vitreous detachment that causes full-thickness tissue loss, including the internal limiting membrane (ILM) and photoreceptor layer in the central macula.[1] The anatomical success rate in MH surgery is high with ILM peeling and gas tamponade. For persistent cases, reinstillation of a gas tamponade is a widely used treatment option; however, the closure rates are not as high as primary surgery.[2] Endophthalmitis, retinal pigment epithelium (RPE) abnormalities, macular edema, retinal detachment, or choroid neovascularization (CNV) are some surgical complications that limit anatomical and functional outcomes.[3]

In the literature, several CNV cases occurring after successful MH surgery have been reported.[3-5] In this presented case, CNV developed after two unsuccessful MH surgeries, and the presence of CNV contributed to the simultaneous closure of this recalcitrant MH defect. This case report aims to draw attention to the closure of a recalcitrant MH following the development of chorioretinal structural changes associated with CNV.

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Case Report

A 67-year-old female patient was referred with a complaint of low vision in her left eye. The best corrected visual acuity (BCVA) values were 20/20 in the right eye and 20/200 in the left eye. Intraocular pressure values were within the normal limits, and there was no abnormal finding in anterior segment examinations with slit-lamp biomicroscopy. On dilated fundus examination, the right eye was completely normal, and a large MH appearance was observed in the left eye [Figure 1]. There was no history of ocular trauma, surgery, chronic disease, or

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drug use. Systemic hypertension for 5 years was present as the only comorbidity. Surgery was planned for the idiopathic MH in the left eye.

One week later, uneventful phacoemulsification, posterior chamber intraocular lens implantation, pars plana vitrectomy, ILM peeling after brilliant blue staining, and perfluoropropane (C_3F_8) gas tamponade were successfully performed under local anesthesia. In addition to standard postoperative medical care including antibiotic, steroid, and cycloplegic eye drops, postoperative face-down positioning was recommended for 1 week. No intraoperative or postoperative complication was observed. At the postoperative 2nd-month visit, intraocular gas completely resorbed, but BCVA was 20/200, and the large MH appearance persisted [Figure 1]. Because of no anatomical and functional improvement, a reoperation was planned for the patient. One month later, the ILM peeling area was widened after brilliant blue staining, and a free ILM flap was created and placed over the MH to cover it. Finally, intraocular C_3F_8 gas tamponade was successfully performed. The same postoperative care procedure was repeated, and no complication was observed. Two months after the second surgery, BCVA was still 20/200, and the same large MH appearance persisted [Figure 1].

Six months after the second surgery, the patient presented with a small decrease in BCVA to 20/400. An elevated mass under the MH edges consistent with CNV was observed on dilated fundus examination and a closed MH on optical coherence tomography [Figure 2]. The patient was followed up monthly without treatment, as she rejected any additional procedure. Two months later, CNV remained limited to that area, and the MH remained closed. Similar optical coherence tomography findings and intraretinal cysts were observed 2 months later [Figure 3].

Four years after the last visit, the BCVA was still 20/400 without any treatment. Closed MH, fibrovascular changes associated with CNV lesion, and intraretinal cysts were observed on optical coherence tomography [Figure 4]. Written informed consent was obtained from the patient to share her medical records and photographs for academic purposes.

Discussion

Pathogenesis and risk factors for CNV after MH surgery are not completely understood. Preexisting drusen and age-related degenerative changes in Bruch's membrane are important risk factors for the development of CNV after MH surgery.[6] RPE abnormalities secondary to MH surgery, induced by intraocular manipulation, direct trauma, light toxicity, and inflammatory processes, are other important predisposing factors for the development of CNV.[3,4,7,8] Nevertheless, CNV is a rare condition after MH surgery, and only a minority of surgery-related RPE abnormalities progress to CNV.[4,5] Banker *et al*.[5] reported that RPE abnormalities occurred in 33% of 95 patients who underwent MH surgery, and CNV developed in only one of them. Use of trypan blue or indocyanine green could be another factor in the development of CNV secondary to MH surgery because those dyes are associated with toxicity to the neurosensory retina and RPE.[9,10] Triamcinolone acetonide or brilliant blue are known as safer options, and covering the MH site with viscoelastic material before staining may limit dye-related toxicity.[11,12] In this regard, using brilliant blue in

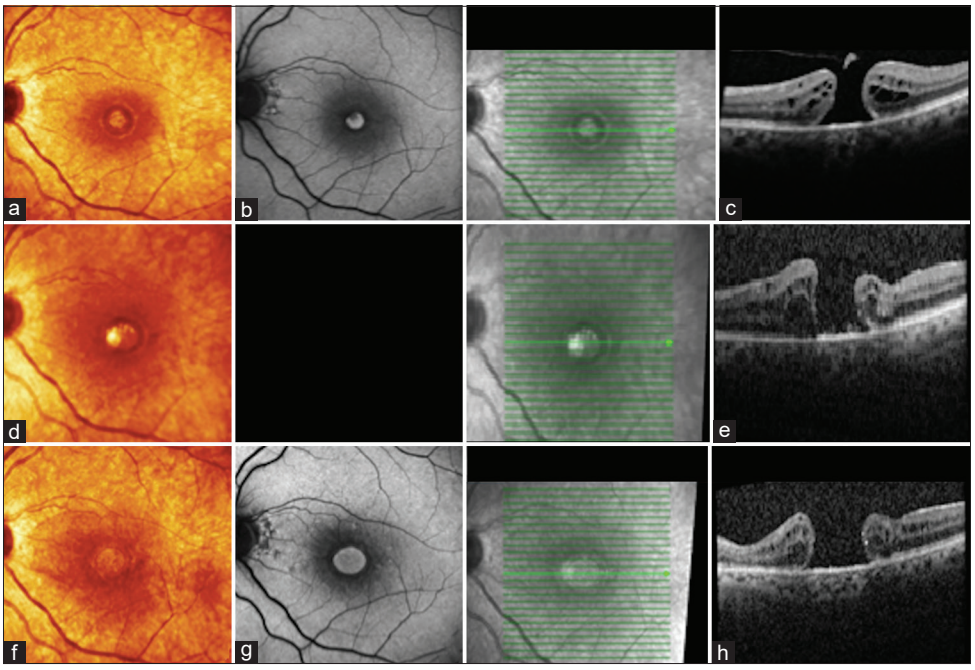

Figure 1: First line: before the first surgery: (a) colored fundus photography; (b) fundus autofluorescence; (c) optical coherence tomography. Second line: before the second surgery: (d) colored fundus photography; (e) optical coherence tomography. Third line: 2 months after the second surgery: (f) colored fundus photography; (g) fundus autofluorescence; (h) optical coherence tomography

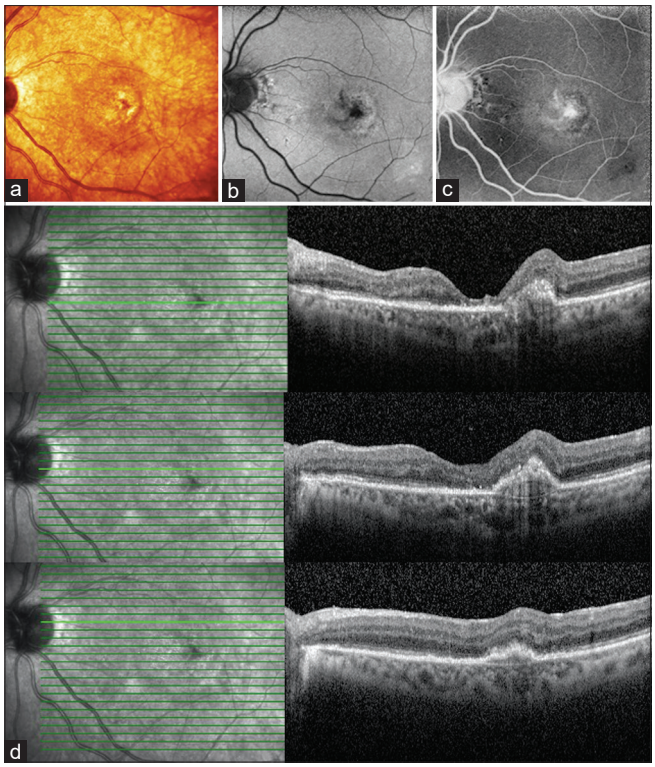

Figure 2: Six months after the second surgery, the macular hole was closed after choroidal neovascularization: (a) colored fundus photography; (b) fundus autofluorescence; (c) fundus fluorescein angiography; (d) optical coherence tomography.

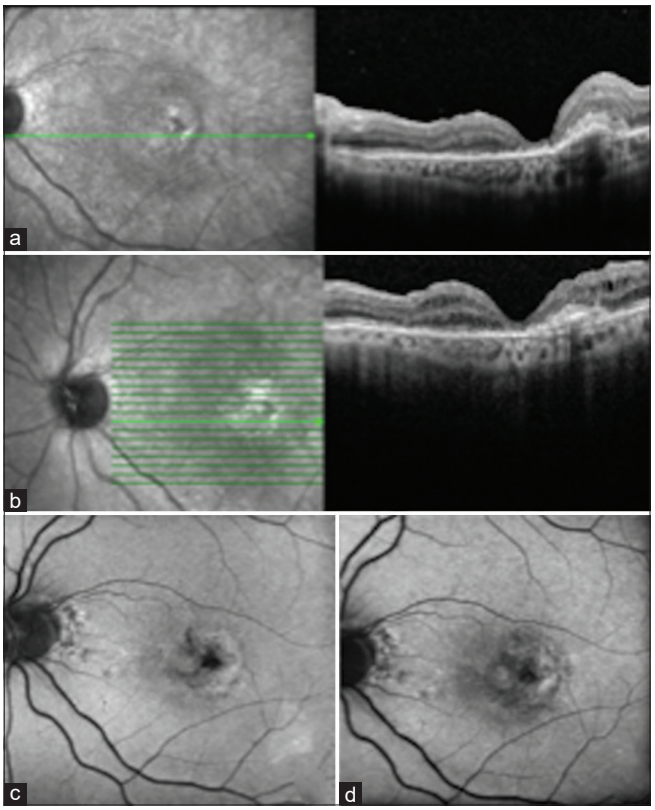

Figure 3: (a) Optical coherence tomography 8 months after the second surgery. (b) Optical coherence tomography 10 months after the second surgery. (c) Fundus autofluorescence 8 months after the second surgery. (d) Fundus autofluorescence 10 months after the second surgery. 4

repeated surgery was no additional risk for the development of CNV in this patient. In this presented patient, the absence of preexisting drusenoid changes was clearly demonstrated with different imaging modalities.

Tabandeh *et al*.[3] reported that nine eyes of eight patients who underwent successful MH surgery developed CNV. They concluded that most CNV lesions occurred in the foveal region, but the central foveal area corresponding to the previous MH site was spared. CNV can occur as early as 6 weeks after MH surgery.[3] They also found that another common feature of the lesions was a predominantly classic appearance on fluorescein angiography, with a ≤2 macular photocoagulation study (MPS) disc diameter.[3] The characteristics of CNV in this patient were fully consistent with the literature. The lesion was diagnosed 6 months after the second MH surgery, and the <2 MPS disc diameter lesion had a predominantly classic appearance.

Most of the CNVs that occur after MH surgery are associated with successful surgeries, and only a small number occur after unsuccessful surgeries.[3] In fact, Spies and Messner[13] reported a case of CNV formation in a patient with untreated MH who had no other risk factors for CNV. At this point, treatment strategies directly aim to regress CNV, regardless of whether MH surgery has been successful or whether the patient has had surgery.[3] Focal laser photocoagulation and photodynamic treatment have been performed to regress CNV, but functional outcomes were not favorable in CNV after MH surgery.[3,13,14] Oh *et al*.[6] reported that after 3-monthly intravitreal ranibizumab injections, the CNV lesion and visual acuity did not change significantly. In this reported case, CNV developed after unsuccessful MH surgery and partially regressed spontaneously within months, without anti-vascular endothelial growth factor treatment. The MH defect remained closed in the following years; however, visual acuity did not significantly increase.

The most important aspect of this case is to report the closure of a recalcitrant MH following the development of chorioretinal structural changes caused by CNV. In the literature, limited reports have clearly shown the macroscopic effects of CNV on retinal structure. In one, CNV occurred after unsuccessful MH surgery complicated with retinal detachment after intravitreal ranibizumab injection. The CNV lesion regressed, and the MH remained open.[15] In contrast, recalcitrant MH closure occurred in this case, following the development of CNV. This can be associated with more extensive retinal morphological remodeling. The new vessels in CNV are highly permeable and cause retinal edema and neuroretinal degeneration. Fibrovascular changes and endothelial-to-mesenchymal transition can be involved in the development and course of CNV.[16] In this process, some endothelial-specific markers such as vascular endothelial growth factor receptor-2 and vascular endothelial cadherin are down-regulated, while some mesenchymal markers,

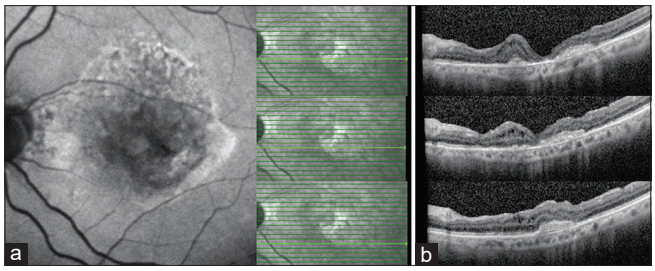

Figure 4: (a) Fundus autofluorescence 4 years after the last visit. (b) optical coherence tomography 4 years after the last visit showing closure of macular hole associated with macular disciform scar

such as vimentin, α-smooth muscle actin, and type I and type III collagens, are up-regulated in the endothelial cells.[17,18] Fibrovascular membrane-like structures and their contractions are likely responsible for the closure of recalcitrant MH. Nevertheless, the hypothesis of endothelial-to-mesenchymal transition in CNV is a novel concept and should be clarified by further experimental and animal studies.

In summary, CNV is an unusual condition after MH surgery, and only a minority of cases has occurred after unsuccessful surgery. The closure of recalcitrant MH may occur following the development of chorioretinal structural changes caused by CNV.

Declaration of patient consent

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

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Conflicts of interest

There are no conflicts of interest.

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