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Crescent-Shaped Retinal Defects Associated With Membrane Peeling With a Diamond-Dusted Membrane Scraper

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Abstract

Membrane peeling is a common procedure for treating diseases of the vitreoretinal interface, such as macular holes and epiretinal membranes; however, potential complications include inner retinal dimples and inner retinal optic neuropathy. The current case series describes five patients who developed large, crescentic inner retinal defects after membrane peeling with diamond-dusted membrane scrapers. The changes visualized by en face optical coherence tomography were outside the fovea and followed the expected contours of membrane scrapers being used intraoperatively. The visual acuities at the last follow-up were 20/40 or better in all five patients.

INTRODUCTION

Peeling internal limiting membranes (ILMs) and epiretinal membranes (ERMs) is commonly performed in vitreoretinal diseases; however, potential complications include visual field loss, retinal tears, inner retinal dimples, inner retinal optic neuropathy, and nerve fiber layer injury.¹ In 1997, Tano et al. introduced a 2-mm, flexible silicone-tipped cannula covered with diamond fragments to provide a gentle, frictional force on the retinal surface to initiate an ILM edge for subsequent peeling.²

The following case series describes five patients who developed crescentic inner retinal defects after membrane peeling. Indocyanine green (ICG) dyes were used to stain the membranes, diamond-dusted membrane scrapers (DDMS) were used to initiate flaps, and end-grasping forceps were used to peel.

CASE DESCRIPTIONS

The retrospective review was approved by the institutional review board of the University of Miami Miller School of Medicine and was compliant with the Health Insurance Portability and Accountability Act of 1996. All five patients underwent 23-gauge pars plana vitrectomy with membrane peeling. The clinical features are summarized in the Table. Patients were between the ages of 62 and 81 years and were followed for 31 to 125 months. Three patients

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had ERMs and two had macular holes (MHs). The preoperative visual acuity ranged from 20/60 to 20/100, and the vision at the last visit ranged from 20/25 to 20/40. All five patients developed new crescentic inner retinal defects postoperatively (Figure). In addition, two patients with a history of macular degeneration developed outer retinal defects. Only one patient with a history of ocular hypertension had a visual field performed in the postoperative period; the cecocentral scotoma did not correlate solely with the crescentic inner retinal defect.

DISCUSSION

Architectural changes in the retina are not uncommon after membrane peeling. Retinal dimples developed in 86% of patients in one study and were hypothesized to result from inner retinal trauma with subsequent regeneration and remodeling.³ In addition, inadvertently grasping the retina may disrupt axoplasmic flow and cause localized nerve fiber layer injury, resulting in small, temporary hyporeflexive arcuate striae.⁴

The large, permanent, crescentic inner retinal defects in the current case series were noted postoperatively on optical coherence tomography (OCT) and progressed slowly over time. Since the structural changes occurred outside the fovea, they did not adversely affect the visual outcomes. The final best-corrected visual acuity was 20/40 or better in all five patients with a follow-up of 2 to 10 years.

The exact etiology of the crescent-shaped retinal defects is unclear; however, they likely resulted from trauma during membrane peeling. Three biomechanical forces may result in damage: scraping, pulling, or pushing the retina. On histopathology, the DDMS has been shown to cause sharp, linear, partial thickness grooves in the ILM.⁵ Repeated scraping in the same area may create larger and deeper grooves. ICG has also been associated with deeper cleavage planes and retinal toxicity during membrane removal.⁶ Furthermore, peeling the ERM and ILM can avulse the Muller footplates.¹ Excessive downward pressure can damage photoreceptors, retinal pigment epithelium (RPE), and/or choroid. The outer retinal atrophy in the AMD patients may have resulted from progression of the macular degeneration or from downward force during surgery.

The crescentic inner retinal defects likely fall on the same spectrum of postoperative retinal trauma as inner retinal dimples. The DDMS, ICG, and ILM forceps are invaluable tools in membrane peeling; however, they may be associated with postoperative retinal and RPE changes. Recently, abrading the ILM with a DDMS has been suggested as an alternative to traditional membrane peeling to close macular holes.⁶ In the future, intraoperative OCT may assist the surgeon in assessing the force and traction on the retinal layers during macular surgery.

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Figure.

Optical coherence tomography (OCT) images of inner retinal defects after membrane peeling. (A) Left: The preoperative OCT of patient 1 on the left demonstrates a smooth, regular inner retinal contour. Right: The OCT obtained 31 months later demonstrates inner retinal loss inferior to the fovea. (B) Left: Patient 2 had a full-thickness macular hole. Right: The OCT 1 year later clearly shows a crescentic inner retinal defect inferior to the fovea. (C) Left: The OCT of patient 3 was obtained 7 months after the membrane peel. Although the outer retina shows drusen and age-related macular degeneration (AMD), the inner retina demonstrates a crescentic inner retinal defect that progressively worsened over time. Right: The OCT obtained 3.5 years later shows significantly greater loss of the inner retina. (D) Left: The OCT of patient 4 was obtained 9 years after the initial membrane peel and demonstrates closure of the macular hole with a crescentic loss of the inner retina superiorly. Right: One year later, the crescentic defect is slightly larger. (E) Left: Patient 5 had a history of neovascular AMD, for which she received multiple injections of anti-vascular endothelial growth factor drugs. Although there is irregularity of the outer retina with a subfoveal choroidal neovascular membrane, the inner retina was intact. Right: After membrane

peeling, the patient developed progressive inner retinal atrophy. The OCT was obtained a year later.

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Clinical Course: Patient Demographics, Surgical Techniques, and Clinical Outcomes

TABLE

Case	Age	M/F	Eye	Ocular History	Indication	Lens	Pre-Op BCVA	Surgery	Instruments used	Defect Location	Postop BCVA	Follow-up (months)
1	78	M	OS	Lattice degeneration	ERM	Pha-kic	20/80	CE/IOL/23G PPV/ICG/IVTA/MP	ICG, DDMS, ILM forceps	Inferior	20/25	31
2	62	F	OS	VMT	FTMH	Pha-kic	20/100	23G PPV/ICG/MP/FAX/C3F8	ICG, DDMS, ILM forceps	Inferior	20/40	31
3	60	M	OD	Dry AMD ERM s/p PPV/MP OHTN	ERM	IOL	20/60	IOL repositioning/23G PPV/ICG/MP/IVTA	ICG, DDMS, ILM forceps	Inferior	20/30	47
4	81	M	OS	None	FTMH	Pha-kic	20/100	23G PPV/ICG/MP/FAX/C3F8	ICG, DDMS, ILM forceps	Superior	20/25	125
5	79	F	OD	Neovascular AMD	ERM	IOL	20/80	23G PPV/ICG/MP/FAX/C3F8	ICG, DDMS, ILM forceps	Inferior	20/25	79

AMD = age-related macular degeneration; BCVA = best-corrected visual acuity; CE = cataract extraction; C3F8 = perfluoropropane gas; DDMS = diamond-dusted membrane scraper; ERM = epiretinal membrane; FAX = fluid-air exchange; FTMH = full-thickness macular hole; ICG = indocyanine green; IOL = intraocular lens; ILM = inner limiting membrane; IVTA = intravitreal triamcinolone; M/F = males/females; MP = membrane peel; OD = right eye; OS = left eye; OHTN = ocular hypertension; PPL = pars plana lensectomy; 23G PPV = 23-gauge pars plana vitrectomy; VMT = vitreomacular traction