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Case Report

Retinal Nerve Fiber Layer Defect and Paracentral Scotoma after Internal Limiting Membrane Peeling with a Nitinol Loop

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Internal limiting membrane (ILM) peeling is an important maneuver in vitrectomy for macular holes (MHs). A nitinol loop is a surgical instrument designed to create an edge on the ILM and peel the ILM safely and consistently. The effect of using a nitinol loop for ILM peeling on the retina is not clear. We report here on a case of an idiopathic full-thickness MH in an adult woman, in whom retinal damage was revealed after her ILM was peeled using a nitinol loop.

Key words: macular hole, macular surgery, internal limiting membrane peeling, nitinol loop

Internal limiting membrane (ILM) peeling is an important maneuver in vitrectomy for macular holes (MHs) and epiretinal membranes [1,2]. The development of ILM visualization methods using dyes such as indocyanine green and brilliant blue G as well as improvements in surgical instruments have made ILM peeling safer and easier. However, ILM peeling remains difficult because the ILM is transparent and extremely thin. One of the most difficult maneuvers in ILM peeling is creating an edge on the ILM to grasp with forceps. Conventional methods for doing so include both the pinch-and-grab technique using forceps, and an ILM incision with a V-lance. However, these methods pose the risk of retinal damage and retinal hemorrhage [3], and thus a safer and more certain method for creating an edge on the ILM is needed.

A nitinol loop is a surgical instrument made of a nickel/titanium alloy that is designed to safely and reliably create an edge on the ILM. This loop features a serrated structure designed so that the loop reaches a depth of approx. Eighty-five% of the thickness of the ILM when it is placed in contact with the retinal surface (Fig. 1). Therefore, this loop can be used to partially peel the ILM by placing it on the retinal surface and sliding it slightly. Because of its structural properties, this loop is considered to pose little risk of damaging the retinal nerve fiber layer (RNFL) when creating an edge on the ILM. However, there have been no detailed reports on the presence or absence of retinal damage following surgeries in which this nitinol loop is used.

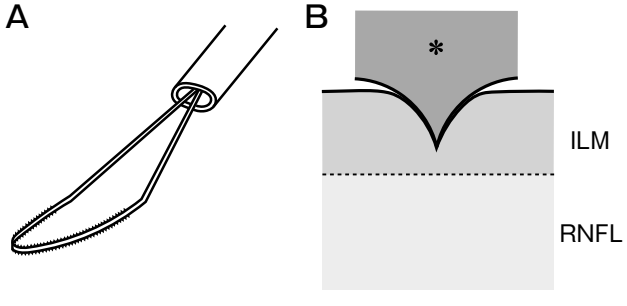

Fig. 1 Schematic image of a nitinol loop (A). The tines of a nitinol loop (*) are designed to penetrate no deeper than 85% of the ILM and not into the RNFL (B).

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We report here a case of idiopathic MH in which we peeled the ILM using a nitinol loop and then evaluated the presence of retinal damage using microperimetry and B-mode scan and en face optical coherence tomography (OCT) images.

Case Report

The patient was a 55-year-old Japanese woman who underwent an examination for sudden central scotoma in her right eye. At the initial presentation, her best-corrected visual acuity (BCVA) was 20/66 OD. Aside from an incipient cataract, no abnormalities were observed in the anterior segment. A full-thickness MH in the right eye was confirmed via fundoscopy and OCT (DRI OCT-1 Atlantis, Topcon, Tokyo, Japan; Fig. 2A-C). Microperimetry (MAIA; CenterVue, Padua, Italy) detected reduced sensitivity at the measurement point in the vicinity of the patient's MH (Fig. 2D).

We performed a microincision vitrectomy (25 gauge) combined with phacoemulsification and aspiration, and we implanted an intraocular lens. The ILM was stained with 0.25 mg/mL brilliant blue G solution (Coomassie BBG 250; Sigma-Aldrich, St. Louis, MO, USA). We used a nitinol loop (Finesse™ Flex Loop, Alcon Grieshaber, Schaffhausen, Switzerland) to partially and gently peel the ILM approx. 1 disc diameter away from the fovea on the temporal superior side of the macula (Fig. 3). The ILM in the retinal vascular arcades was then peeled using 25Ga end gripping forceps (Grieshaber Revolution DSP ILM forceps, Alcon Grieshaber). We performed fluid-20% sulfur hexafluoride gas exchange before ending the surgery. The patient remained in a prone position for 3 days after the surgery.

As shown in Fig. 2E, F, the MH was closed at 1 week

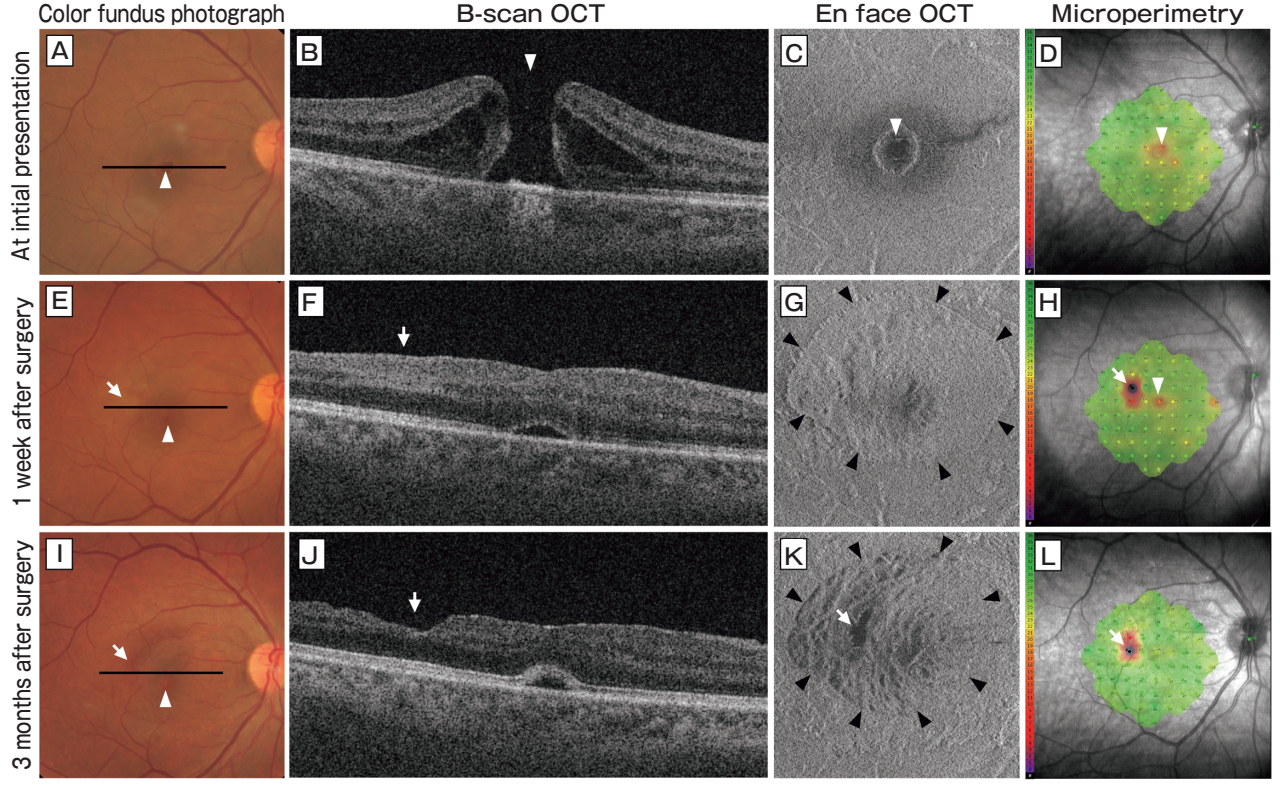

Fig. 2 The RNFL defect observed in the present case following vitrectomy surgery with ILM peeling using a nitinol loop. Color fundus photographs (**A**, **E**, **I**), B-scan OCT (**B**, **F**, **J**), en face OCT (**C**, **G**, **K**), and microperimetry (**D**, **H**, **L**) at the initial presentation (A-D), 1 week after surgery (E-H), and 3 months after surgery (I-L). The white arrowheads indicate an MH (A-C), a closed MH (E, I), and reduced sensitivity in the vicinity of the MH (D, H). The white arrows indicate RNFL swelling (E, F), paracentral scotoma (H, L), and the RNFL defect (I-K). The area surrounded by black arrowheads indicates the ILM peeling area (G) and CMDSs (K).

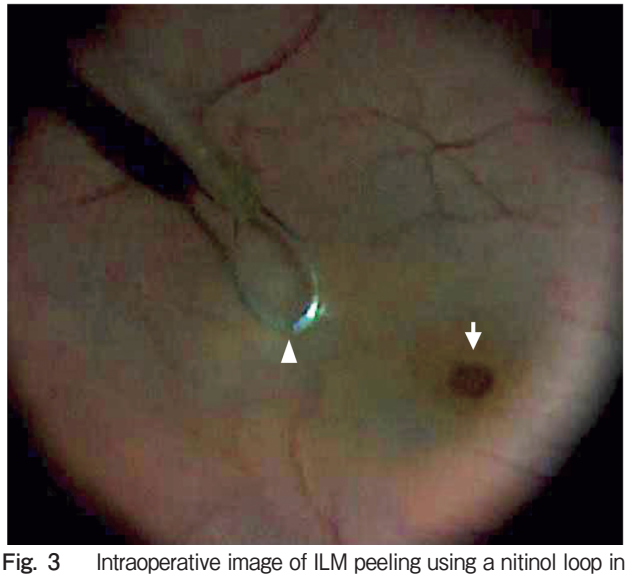

Fig. 3 Intraoperative image of ILM peeling using a nitinol loop in the present patient. The nitinol loop is in contact with the temporal side of the macula (white arrowhead). The white arrow indicates the MH.

after surgery, and at that time the patient's BCVA had improved to 20/40. A B-mode scan OCT image revealed hyper-reflective change, which we considered to reflect RNFL edema corresponding to the site where the nitinol loop was used to create an edge on the ILM (Fig. 2F). However, an en face OCT image at the ILM level revealed no apparent abnormal findings at the site where the nitinol loop was used (Fig. 2G). Not only did microperimetry detect reduced sensitivity in the vicinity of the MH, which was observed prior to surgery; it also detected a paracentral scotoma corresponding to the nitinol loop contact site (Fig. 2H).

As shown in Fig. 2I, J, fundus photography and B-mode scan OCT imaging at 3 months after the surgery showed thinning of the RNFL corresponding to the nitinol loop contact site. En face OCT imaging revealed an RNFL defect corresponding to the nitinol loop contact site as well as concentric macular dark spots (CMDSs) in the area where the ILM was peeled (Fig. 2K). A CMDS indicates a dissociated optic nerve fiber layer (DONFL) [4, 5].

Although microperimetry revealed a persistent paracentral scotoma corresponding to the nitinol loop contact site, reduced sensitivity was not observed in the area of the CMDSs (Fig. 2L). The reduced sensitivity in the vicinity of the MH disappeared, and the patient's BCVA was 20/20 at 3 months after surgery. During the 3-month follow-up, the patient has not been aware of the paracentral scotoma.

Discussion

We report a case where ILM peeling was performed for an MH with the use of a nitinol loop, leading to an RNFL defect and reduced retinal sensitivity corresponding to the nitinol loop contact site. These changes indicate that the use of a nitinol loop to create an edge on the ILM may have damaged the RNFL.

In our patient's case, the changes over time observed at the nitinol loop contact site in the RNFL resembled the changes in the DONFL. According to a previous study, an early change in the DONFL is transient RNFL swelling observed for approx. 1 week after ILM peeling [6]. At approx. 3 months after ILM peeling, the DONFL appears as CMDSs when viewed by en face OCT [4]. In B-mode scan OCT images, CMDSs appear as a focal defect of the RNFL. In the present case, we observed high-intensity changes at 1 week after surgery, which were considered to reflect RNFL swelling corresponding to the nitinol loop contact site (Fig. 2F). At 3 months after surgery, apart from the CMDSs, we observed an RNFL defect corresponding to the nitinol loop contact site (Fig. 2K). The only difference between the nitinol loop-induced RNFL defect and the DONFL was that the nitinol loop-induced RNFL defect was detected by microperimetry as a paracentral scotoma, whereas the DONFL did not demonstrate reduced macular sensitivity (Fig. 2L). These findings suggest that a nitinol loop-associated RNFL defect occurs by the same mechanism as a DONFL and is more severe than a DONFL.

This study was limited by its single-case nature and the possibility that the retinal damage was caused by a surgical technique and not by the nitinol loop. However, our results indicate that the use of a nitinol loop to create an edge on the ILM may cause retinal damage, as it did in the present case. We thus recommend that nitinol loops be used carefully in order to avoid severe vision loss. For this aim, we suggest that the nitinol loop be used (1) in the temporal region of the macula to avoid damaging the papillomacular bundle, (2) at the edge of the area where the ILM is to be peeled off, and (3) as few times as possible. In addition, the design of the nitinol loop may need to be improved in order to make it a safer surgical instrument (e.g., by

reducing the rigidity and making the tines of the loop shorter and duller).

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