

VITREOMACULAR ADHESION AND THE DEFECT IN POSTERIOR VITREOUS CORTEX VISUALIZED BY TRIAMCINOLONE-ASSISTED VITRECTOMY

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Purpose: To study the vitreomacular adhesion and the contractile force of posterior hyaloid, which are shown in triamcinolone acetonide (TA)-assisted pars plana vitrectomy (PPV).

Design: Interventional case series.

Methods: Twenty-eight eyes with diabetic macular edema (DME) without posterior vitreous detachment (PVD) received TA-assisted PPV. Surgical PVD was performed by an aspiration of vitrectomy probe, and the dynamic changes of posterior vitreous cortex and residual vitreous cortex were evaluated.

Results: A premacular defect was formed in the detached posterior vitreous cortex during surgical PVD in 27 of 28 eyes. Immediately thereafter, the small defect expanded into a large hole in the detached posterior vitreous cortex in all cases. A residual vitreous cortex was left on the macula in 22 eyes.

Conclusions: These observations demonstrate a firm vitreoretinal adhesion in the central macula and suggest that the enlargement of the defect of posterior vitreous cortex may be extrusion of vitreous out through the premacular dehiscence into the preretinal space, or a tangentially contractile force may exist in the posterior vitreous cortex. Both macular adhesion and the traction of vitreous cortex might contribute to the pathogenesis of DME and other vitreomacular disease.

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The interaction between posterior vitreous cortex and retina plays a pivotal role in the pathogenesis of various ocular diseases. Especially in macular disease, the adherence of vitreous to the macula is con-

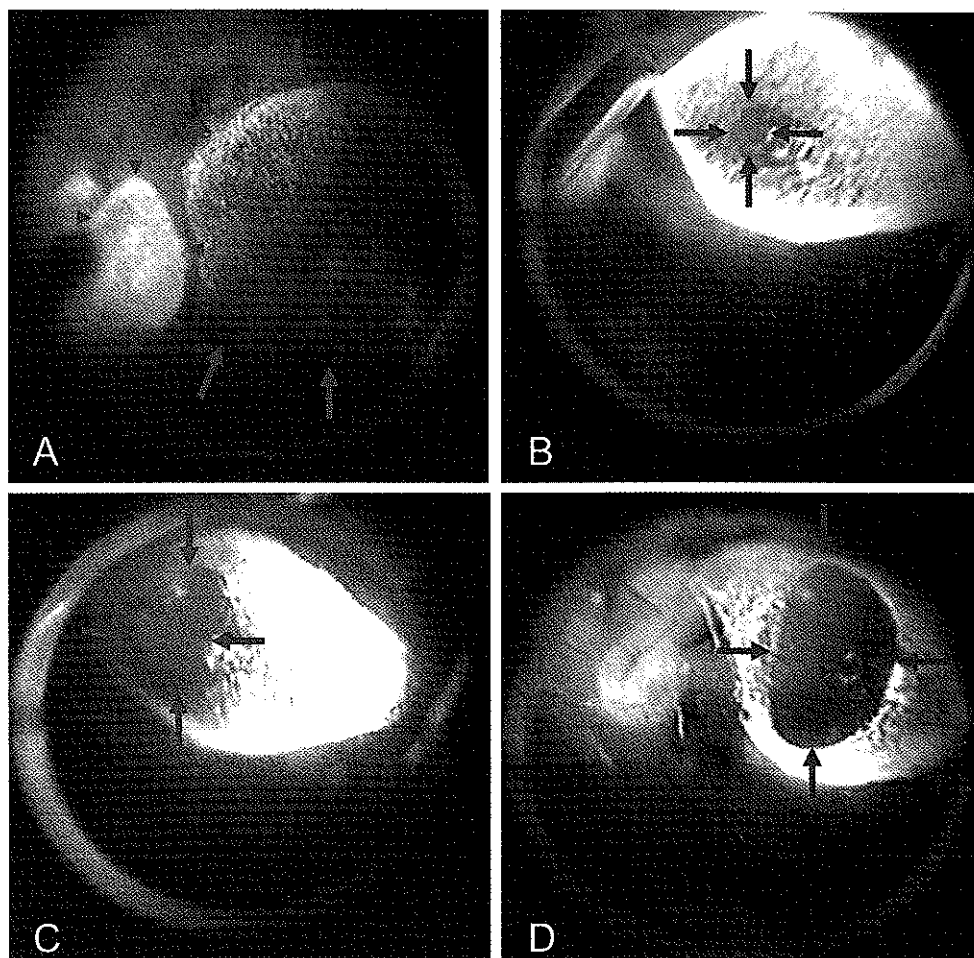
sidered to be the primary pathology, which is frequently found in macular hole and vitreomacular traction syndrome.¹⁻⁷ Through this focal adherence, a contractile force is transmitted to the macula, resulting in macular edema or hole formation. Until now, this contractile force has been assumed to be generated by two mechanisms: one is dynamic vitreous tractional force during ocular rotations and the other is tractional force from the elastic nature of posterior vitreous cortex.^{1,2} The latter idea is supported by the finding of a trampoline-like posterior vitreous detachment (PVD) in ultrasonography and/or optical coherence tomography (OCT).^{1,5,6,8} However, no apparent evi-

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Fig. 1. A-D, Intraoperative findings of the formation of premacular oval defect in the detached posterior hyaloid cortex. **A**, The premacular liquefied space (arrows) and another liquefied space on the optic disk (arrowheads) adjacent to the premacular space are stained white. **B**, Small defect is formed in the posterior hyaloid cortex around the fovea (arrows). **C**, The defect in the posterior hyaloid cortex (arrows) is enlarged as the posterior hyaloid cortex is separating from the retina. **D**, The formed oval defect in the posterior hyaloid cortex (arrows) enlarges its size much larger than the remaining vitreous cortex at the fovea (arrowheads).



dence of contractile force of vitreous cortex has yet to be shown directly in a clinical study.

Recently, we performed a triamcinolone acetonide (TA)-assisted pars plana vitrectomy (PPV) to visualize the vitreous.⁹⁻¹² This method allows a clear observation of the gel structure of the vitreous body, especially that of the relationship between posterior hyaloid and retinal surface. Using this technique, we found the dynamic change of posterior hyaloid cortex during surgical PVD. This observation provides evidence of the contractile potential of posterior vitreous cortex, and provides insight to the mechanism of diabetic macular edema and various macular diseases.

Patients and Methods

This study comprised 28 eyes in 25 patients (14 women and 11 men) with diabetic macular edema. Patients underwent TA-assisted PPV at Kagoshima University Hospital between December 2002 and November 2003. Patients with predisposing factors, such as significant posterior vitreous detachment, prolifer-

ative membrane with or without tractional retinal detachment, and massive vitreous hemorrhage, were excluded. Possible advantages and disadvantages of treatment were explained to patients and fully informed consent was obtained. The study was carried out with the approval of Kagoshima University Hospital Review Board, and was performed in accordance with the ethical standards laid down in the 1989 Declaration of Helsinki.

TA-assisted PPV was performed by the previously described method.⁹⁻¹² Briefly, standard three sclerotomies were created. Cataract surgery and/or intraocular lens insertion was done when needed. Prior core vitrectomy was performed, and TA solution was sprayed onto the vitreous to visualize the vitreous gel. TA solution (Kenacort-A, Bristol Pharmaceuticals KK, Tokyo, Japan) was made by the previously described method.⁹⁻¹² The posterior vitreous cortex was separated from the retina by active aspiration with a vitrectomy probe or a soft cannulated extrusion needle around the optic disk. This procedure was performed

gently and carefully so as not to damage the posterior vitreous cortex (Figure 1). After surgical PVD, the residual vitreous cortex was resected and endolaser photocoagulation and/or fluid-gas exchange were performed when needed. The intraoperative findings were recorded immediately after surgery, and these records were reviewed postoperatively.

Results

After injection of TA suspension, the premacular liquefied space was clearly visualized as a white structure in all 28 eyes. The posterior vitreous around the fovea seemed to lose the construction of gel and formed only thin layer of vitreous cortex. Another liquefied space was confirmed on the optic disk adjacent to the premacular space. These findings were similar to previous autopsy findings called bursa premacularis or posterior vitreous pocket and Martegiani's space^{13,14} (Figure 1A).

In most eyes (27 eyes), although vitreoretinal separation could be done gently and smoothly, there was a firm adherence of the vitreous cortex to the fovea. As a result, a small premacular defect was formed in the detached posterior vitreous cortex (Figure 1B). Immediately thereafter, the small defect became as large as two to three disk diameters (Figure 1, C and D). Interestingly, there was a small island of residual vitreous cortex left on the macula in 22 eyes, which corresponded to the posterior vitreous cortex defect, while six eyes did not show any residual vitreous cortex on the macula. In three eyes, a large portion of posterior vitreous cortex was left on the posterior area, which appeared to be identical to the posterior wall of the vitreous pocket. Only one eye had no defect in the posterior vitreous cortex when surgical PVD was made. All surgical procedures were performed without any serious problems. Intraoperative retinal tear was formed unintentionally, which was treated by laser endophotocoagulation (3 eyes).

Discussion

In this study, we observed that a small foveal defect of the posterior hyaloid was formed at the beginning of a surgical PVD maneuver and this defect was enlarged into round or oval hole immediately. The present observation has two important implications. First, there is a strong adherence of the vitreoretinal contact in the central macula. A small island of posterior vitreous cortex was frequently left on a macular area after surgical PVD. The OCT images in a previous study reveal the existence of vitreomacular adhesion by the observation of natural process of PVD in

healthy eyes.⁸ Kishi et al reported that vitreous cortex remnants were present on the retinal surface in the macular area in 26 (44%) of 59 eyes by scanning electron microscopic study of autopsy eyes with PVD.¹⁵ It is interesting that this residual vitreous cortex on the macula was observed more frequently in surgical PVD (79% in this study) than in spontaneous PVD. Because an eye without PVD might have a stronger vitreoretinal adhesion than one with PVD, it is plausible that an eye without PVD has more chance to have a residual vitreous cortex on the macula after surgical PVD than an eye with spontaneous PVD. As previously suggested, the premacular vitreous cortex remains attached to the retina after PVD, with the results that round defect of posterior vitreous is formed.^{16,17} Remnants of vitreous cortex on the macula after PVD may provide a scaffold for cell proliferation and might thereby play a role in the formation of epiretinal membrane.

Second, the small defect in posterior vitreous cortex expanded greatly immediately after its formation. As previous pathologic studies suggested, an equally plausible hypothesis is that this enlargement is caused by extrusion of vitreous out through the premacular dehiscence into the preretinal space.^{18,19} Otherwise, there may be a strong tangential force from the posterior vitreous cortex. If a tangential contractile force exists in a membrane (e.g., rubber membrane), the defect grows into a larger hole, as observed in this study. We cannot exclude that enlargement of the hole in the posterior vitreous cortex was induced in part by surgical manipulation or fluid currents through the newly formed hole. However, these observations suggest that a constant tangential contractile force is present in the vitreous cortex.

When a continuous vitreoretinal interface is separated in a small area for some reason, this separation will spread to the surrounding area due to this contractile force. However, the separation will stop at the area where vitreoretinal adhesion is stronger than the contractile force of the posterior hyaloid. Considering the firm adherence of vitreous and macula observed in this study, the macula is probably such an area. A trampoline-like structure observed by ultrasonographic study or OCT analysis might reflect this process.^{1,8} Once this vitreoretinal separation produces enough space to fill vitreous fluid between posterior hyaloid and retinal surface, the additional dynamic vitreous tractional force during ocular rotations acts to further promote vitreoretinal separation. These static and dynamic tractional forces likely work together to cause a variety of vitreomacular pathologies and may help explain why vitrectomy is beneficial in many cases.

There are, of necessity, clear limitations to the present study. This is an observational case series and a randomized case selection was not performed. Evaluation of residual vitreous cortex and posterior vitreous cortex was not done in a perfectly objective manner. Above all, this is a study of diabetic macular edema eyes. Nonetheless, it is likely that the patho-anatomic change occurs similarly in both nondiabetic and diabetic eyes. TA-assisted PPV vividly shows dynamic changes on the vitreoretinal surface, which will allow better understanding of various ocular diseases.

References

1. Johnson MW, Van Newkirk MR, Meyer KA. Perifoveal vitreous detachment is the primary pathogenic event in idiopathic macular hole formation. *Arch Ophthalmol* 2001;119:215-222.
2. Kishi S, Hagimura N, Shimizu K. The role of the premacular liquefied pocket and premacular vitreous cortex in idiopathic macular hole development. *Am J Ophthalmol* 1996;122:622-628.
3. Gass JDM. Idiopathic senile macular hole: its early stages and pathogenesis. *Arch Ophthalmol* 1988;106:629-639.
4. Gass JDM. Reappraisal of biomicroscopic classification of stages of development of a macular hole. *Am J Ophthalmol* 1995;119:752-759.
5. Gaudric A, Haouchine B, Massin P, et al. Macular hole formation: new data provided by optical coherence tomography. *Arch Ophthalmol* 1999;117:744-751.
6. Chauhan DS, Antcliff RJ, Rai PA, et al. Pappilovitreal traction in macular hole formation: the role of optical coherence tomography. *Arch Ophthalmol* 2000;118:32-38.
7. Guyer DR, Green WR. Idiopathic macular holes and precursor lesions. In: Franklin MR, ed. *Retina and Vitreous*. Amsterdam, the Netherlands: Kugler Publications; 1993:135-162.
8. Uchino E, Uemura A, Ohba N. Initial stage of posterior vitreous detachment in healthy eyes of older persons evaluated by optical coherence tomography. *Arch Ophthalmol* 2001;119:1475-1479.
9. Sakamoto T, Miyazaki M, Hisatomi T, et al. Triamcinolone-assisted pars plana vitrectomy improves the surgical procedures and decreases the postoperative blood-ocular barrier breakdown. *Graefes Arch Clin Exp Ophthalmol* 2002;240:423-429.
10. Sonoda KH, Sakamoto T, Enaida H, et al. Residual vitreous cortex after surgical vitreous separation visualized by intravitreal triamcinolone acetonide. *Ophthalmology* 2004;111:226-230.
11. Enaida H, Sakamoto T, Ueno A, et al. Submacular deposition of triamcinolone acetonide after triamcinolone-assisted vitrectomy. *Am J Ophthalmol* 2003;135:243-246.
12. Enaida H, Hata Y, Ueno A, et al. Possible benefits of triamcinolone-assisted pars plana vitrectomy for retinal diseases. *Retina* 2003;23:764-770.
13. Worst JG. Cisternal systems of the fully developed vitreous body in the young adult. *Trans Ophthalmol Soc UK* 1977;97:550-554.
14. Kishi S, Shimizu K. Posterior precortical vitreous pocket. *Arch Ophthalmol* 1990;108:979-982.
15. Kishi S, Demaria C, Shimizu K. Vitreous cortex remnants at the fovea after spontaneous vitreous detachment. *Int Ophthalmol* 1986;9:253-260.
16. Kishi S, Shimizu K. Oval defect in detached posterior hyaloid membrane in idiopathic premacular fibrosis. *Am J Ophthalmol* 1994;118:451-456.
17. Hikichi T, Takahashi M, Trempe CL, et al. Relationship between premacular cortical vitreous defects and idiopathic premacular fibrosis. 1995;15:413-416.
18. Sebag J. Age-related differences in the human vitreoretinal interface. *Arch Ophthalmol* 1991;109:966-971.
19. Sebag J. Anatomy and pathology of the vitreoretinal interface. *Eye* 1992;6:541-552.