CORRESPONDENCE

Ultrastructural Features of Tissue Removed During Idiopathic Macular Hole Surgery

EDITOR:

IN THE ARTICLE, "ULTRASTRUCTURAL FEATURES OF TISsue Removed During Idiopathic Macular Hole Surgery," by H-S. Yoon, H. L. Brooks, A. Capone, Jr, N. L. L'Hernault, and H. E. Grossniklaus (Am J Ophthalmol 122:67–75, July 1996), the authors present ultrastructural features of epiretinal membrane tissue and internal limiting lamina removed during surgery for 12 cases of idiopathic macular holes. Using the reappraised classification system of Gass,[1] there were two stage II holes, seven stage III holes, and three stage IV holes. Recently Madreperla and associates[2] reported that pseudo-opercula removed during vitrectomy surgery for idiopathic macular holes were made up of mostly fibrous astrocytes and Müller cells, supporting the idea that the pseudo-opercula are devoid of neural retinal tissue. However, the two reported cases from Madreperla were relatively chronic in duration with symptoms of 1 to 2 years in duration, bringing up the possibility of degeneration of neural retinal tissue secondarily after formation of the operculum. The two most important cases from this paper by Yoon and associates are the stage II macular holes with acute symptoms of 3 months (case 1) and 2 weeks (case 3). In these cases, there was again no evidence of neural retinal tissue with internal limiting membrane in both cases, and fibrous astrocytes with native vitreous collagen in one case.

In the schematic diagram depicting a possible mechanism for macular hole development (Figure 9 in reference 3), the authors show contraction of fibrocytes in stage I with a foveolar detachment. In this figure, the fovea is still shown to have a slight depression centrally. Based on clinical findings, this is often incorrect, as in many stage I macular holes the fovea is actually elevated with surrounding tractional striae sloping downward from the central foveal elevation, similar in appearance to a volcano. Additionally, using ultrasonographic studies, I have demonstrated actual foveal elevation or thickening in 50% of stage I holes.[4,5] The foveal elevation or thickening in stage I macular holes has been confirmed by optical coherence tomography in four cases of stage I macular holes (Duker, Reichel, and Puliafito, unpublished data, Vitreous Society, London, England, August 1995). Optical coherence tomography also demonstrated a significant cystic change within the area of foveal elevation in stage I macular holes.

Based on ultrasonographic studies of the vitreous, I believe that the vitreous traction forces are more complex than only tangential traction parallel to the retina. I think that there is also oblique vitreofoveal fibers within the cortical vitreous gel with an anterior-posterior vector component of traction, as well as the tangential vector component of traction parallel to the retina (see Figure 4 in reference 4). This traction with an anterior-posterior vector component may be responsible for the actual elevation of the central fovea in stage I macular holes, as traction parallel to or on the retinal surface, as depicted by the authors would not likely result in actual elevation of the central fovea. Additionally, optical coherence tomography has also demonstrated oblique vitreous fibers tangentially inserting into the central foveal elevation in stage I macular holes (Duker, Reichel and Puliafito, unpublished data, Vitreous Society, London, England, August 1995). The authors of the optical coherence tomography paper believe this may be the partially detached posterior hyaloid inserting into the fovea, but on the basis of my ultrasonographic studies,[4] this is much more often vitreous gel fibers within the cortical vitreous gel. Guyer and Green[6] have speculated that the tangential traction on the macula causing macular holes may be related to cellular proliferation in and on the inner surface of

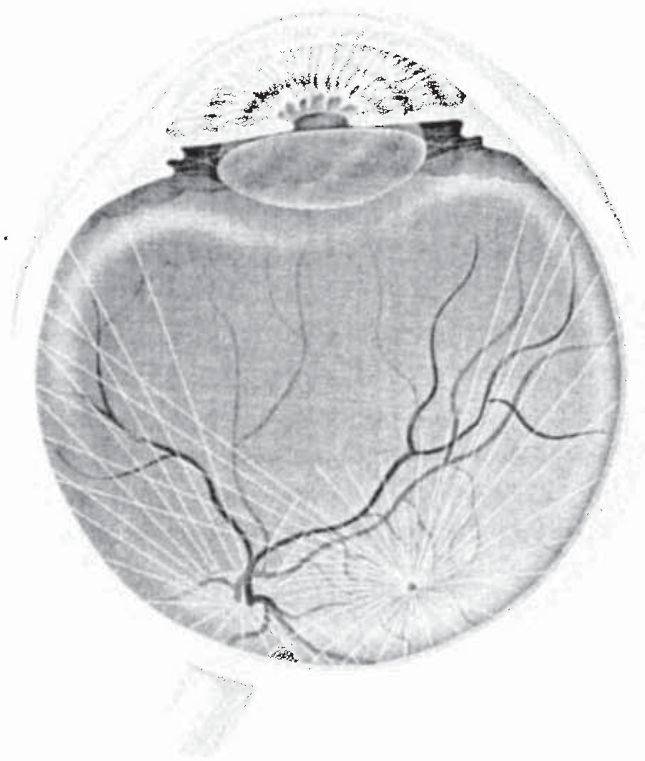

Figure. Schematic diagram based on ultrasonographic studies of vitreous fiber anatomy in eyes with macular holes showing vitreous fiber insertion into the fovea and into the disk. Note the vitreous fiber insertion into the fovea tangential and parallel to the retina, as well as obliquely oriented vitreofoveal cortical vitreous fiber insertion with an anterior-posterior component to the traction.

the thin prefoveal cortical vitreous gel. These cells within the gel may contract and result in realignment of cortical vitreous fibers. I speculate that the realignment of fibers within the attached cortical vitreous may make the oblique fibers inserting into the fovea bore linearly aligned and thus more echogenic. This contraction of cells within the prefoveal cortical vitreous could then result in tangential circumferential traction parallel to the retina, as well as tractional forces on the fovea with an anterior-posterior traction component due to oblique insertion of vitreous fibers into the fovea (Figure).

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AUTHOR REPLY

I AGREE WITH DR KOKAME THAT THERE IS OFTEN ANTERior-posterior as well as tangential traction of the vitreous to the fovea in the evolution of idiopathic macular holes. I appreciate Dr Kokame's analogy of this causing the fovea to appear like a volcano, especially since Dr Kokame is located in Hawaii.

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Rate of Progression in Open-angle Glaucoma Estimated From Cross-sectional Prevalence of Visual Field Damage

EDITOR:

IN THE ARTICLE, "RATE OF PROGRESSION IN OPEN-ANGLE Glaucoma Estimated From Cross-sectional Prevalence of Visual Field Damage," by H. Quigley, J. Tielsch, J. Katz, and A. Sommer (Am J Ophthalmol 122:355–363, Sept 1966), the authors conclude that the "rate of progressive visual field loss is not sufficient to lead to bilateral blindness in the majority of those affected" with open-angle glaucoma. They performed regression analyses on Goldmann visual field data obtained from 151 persons with open-angle glaucoma "to estimate the average rate of progressive field loss and blindness in glaucoma."