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Vitreous Changes After Treatment of Retinopathy of Prematurity

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Purpose: To investigate the vitreous findings in patients with cicatricial retinopathy of prematurity (ROP) who underwent retinal cryopexy and/or photocoagulation during the acute phase of the disease.

Methods: Vitreous findings were evaluated in 15 patients (29 eyes) with cicatricial ROP by slit-lamp biomicroscopy and indirect ophthalmoscopy.

Results: The ocular examination revealed that all eyes had extensive vitreous liquefaction that affected a large segment of the vitreous. A great deal of fibrillar condensation of the vitreous was present in membrane-like vitreous fibers that traversed the vitreous cavity to the periphery of the degenerating retina. These vitreous changes were most marked in the areas in which retinal cryopexy and/or photocoagulation had been performed. Despite advanced liquefaction, the posterior cortical vitreous was not separated from the retina in any eyes.

Conclusion: In eyes with ROP that underwent retinal cryopexy and/or photocoagulation during the acute phase of the disease, the vitreous was abnormal, which may contribute to vitreoretinal traction that eventually leads to retinal breaks and detachment. **Jpn J Ophthalmol 1999;43:543–545**

Key Words: Cryopexy, photocoagulation, retinopathy of prematurity, vitreous changes.

Introduction

In cicatricial retinopathy of prematurity (ROP), rhegmatogenous retinal detachment is the major complication that threatens vision in older children or young adults.[1,2] In 1971, Faris et al[3] reported the vitreous findings in patients with mild cicatricial ROP, previously referred to as retrolental fibroplasia. The resultant vitreous changes that included vitreous gel liquefaction and posterior vitreous detachment (PVD) were extremely rare. Vitreous bands and membranes traversing the liquefied vitreous cavity were also observed. The authors suggested that their results demonstrated firm vitreoretinal adhesion, an important factor in the pathogenesis of the fundus. Formation of retinal breaks is thought to result from slow, continuous traction of the vitreous gel at sites of abnormal vitreoretinal adhesion.[1–3]

More than a quarter century has passed since that study, and during that time the treatment for the acute phase of the disease has changed. It is now accepted that retinal cryopexy and photocoagulation prevent advanced extraretinal fibrovascular proliferation in acute ROP.[4,5] To the best of our knowledge, no study has reported the vitreous findings in eyes with cicatricial ROP after the above treatment was accepted as efficacious. In the present study, we evaluated the vitreous findings in 15 patients with cicatricial ROP who underwent retinal cryopexy and/or photocoagulation during the acute phase of the disease.

Materials and Methods

Twenty-nine eyes of 15 patients (9 boys, 6 girls) with cicatricial ROP who underwent cryopexy and/or photocoagulation during the acute phase of the disease were studied. The mean patient age (±stan-

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dard deviation [SD]) was 9.0 ± 3.2 years (range = 6–17 years). The spherical equivalent was −3.0 ± 4.5 diopters (D) (range = 0.5 to −15.0 D). The gestational age at birth and the birth weight were 27 ± 1.4 weeks (range = 24–30 weeks) and 1039 ± 208 grams (range = 602–1372 grams), respectively.

During the acute phase in all eyes, ROP progressed beyond stage 3 (moderate); in 7 eyes, ROP progressed to stage 4A.[6,7] Retinal photocoagulation was performed in 18 eyes, cryopexy was performed in 8 eyes, and both were performed in 3 eyes. An ocular examination including the measurement of the best-corrected visual acuity, slit-lamp biomicroscopy, and indirect ophthalmoscopy, was performed.

The vitreous condition was studied and documented with a vitreous observation system that consisted of a slit-lamp microscope, a preset lens, a monochromatic CCD camera, and a computerized unit for image processing and storage.[8] The CCD camera was connected to the slit-lamp, and the vitreous image, which was transmitted from the CCD camera, was viewed on a television monitor. The image was then saved in the computer.

Results

During the ocular examination in 3 eyes, a mass of opaque tissue was observed in the fundus periphery with some localized retinal detachment, and the optic disc was dragged toward the tissue. In the remaining 26 eyes, a mass of opaque tissue was seen in the fundus periphery without retinal detachment; attenuated retinal vessels and a retina that was pale in the periphery were observed. All eyes had extensive liquefaction that affected a large segment of the vitreous (Figure 1); changes such as these are quite rare in normal eyes of the same age.[9]

Fibrillar condensation of the vitreous was present in membrane-like vitreous fibers that traversed the vitreous cavity to the periphery of the deteriorating retina (Figure 2). These vitreous changes were seen in a large segment of the vitreous; however, they were most prevalent in the areas in which retinal cryopexy and/or photocoagulation had been performed. Optically empty spaces in the vitreous over the chorioretinal atrophic scars after treatment were generally larger in eyes treated with cryopexy, because the chorioretinal atrophic lesions were commonly larger in eyes that underwent cryopexy than in eyes that underwent photocoagulation. These optically empty spaces in the vitreous were traversed by condensed vitreous strands. Firm vitreoretinal adhesion was observed at the margin of the peripheral deteriorating retina. Some condensed vitreous strands seemed to originate from the opaque tissue in the fundus periphery with or without retinal detachment and advance to the posterior lens. Despite advanced liquefaction, the posterior cortical vitreous was not separated from the retina in any eyes (Figures 1 and 2).

Discussion

In eyes with cicatricial ROP that underwent retinal cryopexy and/or photocoagulation during the acute phase of the disease, marked vitreous liquefaction without PVD was observed, especially in the retinal areas that underwent treatment. The results of the present study confirm that increased vitreous liquefaction without PVD occurs in eyes with cicatricial ROP, as reported by Faris et al.[3] These vitreous changes are abnormal in the first or second decade of life.[9]

Because the efficacy of the treatment to prevent advanced extraretinal fibrovascular proliferation in

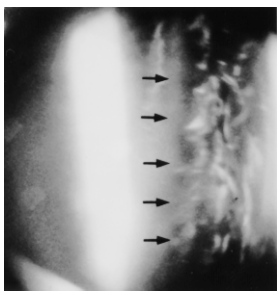

Figure 1. Vitreous photograph. Extensive fibrillar condensation of vitreous cortex (arrows) is present in liquefied vitreous.

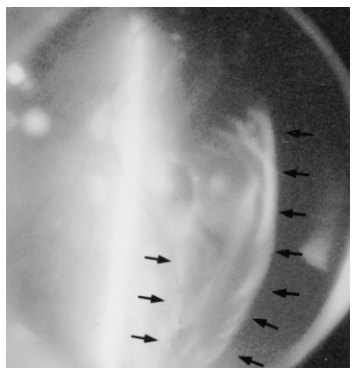

Figure 2. Vitreous photograph. Thick membrane-like vitreous fiber (arrows) traverses vitreous cavity to periphery of deteriorating retina.

acute ROP was not well established at the time that Faris et al reported their results, no patients in their study were treated with retinal cryopexy or photocoagulation.[3] Retinal cryopexy and photocoagulation are thought to induce vitreous liquefaction.[10] Although the present study did not compare the vitreous findings in eyes with cicatricial ROP between those that underwent treatment and those that did not, and because the vitreous changes were most marked in the areas in which the treatment had been performed, the advanced vitreous liquefaction observed in the present study most likely results from the disease itself and the treatment.

Foos[11] demonstrated that in the acute phase of stage 3, the vitreous overlying the ridge became opalescent with linear condensation, and then an optically empty pocket developed over the ridge. It was suggested that this pocket of liquefaction collapsed, and, consequently, synchysis of the central vitreous developed, which was observed in the current study. Foos also speculated that the fibrovascular proliferation surrounding the ridge destroyed the overlying vitreous by causing degradation of the hyaluronic acid and condensation of the vitreous collagen. de Juan et al[12] examined the historical and ultrastructural characteristics of 27 membranes removed during vitreous surgery to treat stage 4 or 5 ROP; and reported that the retinal surface was perforated by many glial microvillous protrusions that had focally dissolved the internal limiting lamina and migrated into the vitreous cavity.

These findings suggest that the proliferative membrane is securely attached to the retina. The vitreous manifestations of familial exudative vitreoretinopathy are different from those of cicatricial ROP. The vitreous condensation and vitreous strands attached to the deteriorating peripheral retina are also observed in this disorder. However, vitreous liquefaction occurs infrequently,[13] which differs from the vitreous changes of cicatricial ROP confirmed in the present study.

Eyes with cicatricial ROP that had undergone cryopexy and/or photocoagulation during the acute phase of the disease had the following characteristics: vitreous changes, such as marked vitreous liquefaction; vitreous membranes traversing the liquefied vitreous cavity; firm vitreoretinal adhesion at the margin of the peripheral deteriorating retina; and the low incidence of PVD despite advanced liquefaction. Such characteristics may contribute to vitreoretinal traction that eventually leads to retinal breaks and detachment. Periodic examination of these patients is necessary, even though their vision is not threatened during the acute phase of the disease.

The authors have no commercial or proprietary interest in any aspect of this study.

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