Rhegmatogenous Retinal Detachment Due to Paravascular Linear Retinal Breaks Over Patchy Chorioretinal Atrophy in Pathologic Myopia

Ling Chen, MD, PhD; Keyan Wang, MD; Daniel D. Esmaili, MD; Gezhi Xu, MD, PhD

Objective: To characterize posterior paravascular linear retinal breaks over areas of patchy chorioretinal atrophy as a cause of retinal detachment among patients with pathologic myopia.

Methods: In this retrospective case series, we evaluated 10 pathologically myopic eyes having rhegmatogenous retinal detachment associated with posterior paravascular linear retinal breaks.

Results: Ten eyes with posterior paravascular linear retinal breaks and retinal detachment were identified from January 1, 2008, to July 31, 2009. The retinal breaks were most frequently found along the inferotemporal vascular arcade, followed by the superotemporal arcade. The length of the breaks ranged from 0.25 to 1 disc diameter, and their distance from the optic disc ranged from 1 to 5 disc diameters. These paravascular linear retinal breaks have distinct clinical characteristics, including a strong propensity to occur over areas of patchy chorioretinal atrophy, a linear shape that is oriented parallel to the adjacent retinal vessels, and a tendency to result in progressive retinal detachment. Vitrectomy with gas tamponade was performed in all cases, and retinal reattachment was achieved in 9 cases by a single operation.

Conclusions: Paravascular linear retinal breaks over areas of patchy chorioretinal atrophy represent a distinct clinical entity that can result in a special category of retinal detachment among patients with pathologic myopia. These breaks are apt to elude detection before surgery, and a careful search along the posterior vascular arcades during vitrectomy may help to detect these abnormalities. Pars plana vitrectomy with photocoagulation and intraocular tamponade may lead to a resolution of such rhegmatogenous retinal detachments.

Arch Ophthalmol. 2010;128(12):1551-1554

Pathologic myopia is one of the leading causes of blindness in the world. It is characterized by excessive axial elongation with progressive degeneration of the posterior pole. Various sequela, such as staphyloma, macular hole, and chorioretinal atrophy, have been widely recognized. Recently, other abnormalities affecting the posterior pole have been described, such as paravascular retinal microfolds, intraretinal cysts, lamellar holes, inner retinal cleavage, and foveoschisis. Detection of these retinal pathologic conditions helps to understand or explain the progressive degenerative changes of pathologically myopic eyes.

Retinal detachment represents a major vision-threatening complication of pathologic myopia and is associated with peripheral retinal tears or macular holes. However, retinal detachments with undetected breaks have been frequently reported in the literature. In such cases, the surgical success rate was suboptimal because the detection of retinal breaks is critical for the successful treatment of retinal detachments. Herein, we report an unusual type of posterior retinal break that can easily escape diagnosis. These breaks have a linear shape, lie parallel to the adjacent retinal vessels of the posterior vascular arcades, are exclusively located over areas of patchy chorioretinal atrophy, and result in progressive retinal detachment among patients with pathologic myopia.

Methods

Study Design

This was a retrospective case series of 10 consecutive patients with paravascular linear retinal breaks seen at Eye and Ears, Nose, and Throat Hospital, Fudan University, Shanghai, China, from January 1, 2008, to July 31, 2009. Before data collection began, permission for medical record review was obtained by the institutional review board of the hospital.
INCLUSION AND EXCLUSION CRITERIA

Patients with pathologic myopia and rhegmatogenous retinal detachment were included in the study if they had posterior paravascular linear retinal breaks, complete baseline clinical data, a minimum of 6 months of follow-up, and a video recording of their surgery. Patients with insufficient documentation were excluded from the study. Baseline clinical data included patient demographics, Snellen visual acuity, ocular examination findings, ocular axial length, and refractive error. Follow-up data included Snellen visual acuity, ocular examination findings, and retinal reattachment results.

SURGICAL PROCEDURE

Standard 3-port pars plana vitrectomy was performed using retrobulbar anesthesia by an experienced surgeon (G.X.). All eyes underwent phacoemulsification to enable clear visualization of the fundus and to improve postoperative Snellen visual acuity, followed by vitrectomy. The core vitreous was first removed, paying particular attention to visualizing any vitreo-retinal adhesions and freeing the posterior hyaloid membrane with active suction. All focal or diffuse vitreoretinal adhesions and epiretinal membranes were removed with the help of triamcinolone acetate. The retinal periphery, macula, and vascular arcades were carefully examined to identify retinal breaks. Circumferential retinopexy was performed around breaks and associated patchy atrophy using an argon laser, and the vitreous cavity was filled with perfluoropropane (C3F8) gas. Postoperative follow-up examinations were performed at day 1 and week 1, followed by every 2 weeks for the first 3 months and then every 2 to 6 months thereafter.

RESULTS

Included in the study were 10 patients with rhegmatogenous retinal detachment due to posterior paravascular linear retinal breaks (n=8), a combination of paravascular linear retinal breaks with a macular hole (n=1), or a peripheral horseshoe tear (n=1). The mean patient age was 60 years (age range, 51-70 years). Seven patients were women, and 3 patients were men. Both eyes were equally myopic in all cases, and the mean spherical equivalent correction in the study eye was −12.1 diopters (D) (range, −7.0 to −18.0 D). The mean axial length was 30.0 mm (range, 28.6-34.1 mm). Preoperative Snellen visual acuity ranged from hand motions to 20/400 lines. The mean duration of retinal detachment was 1 month (range, 5 days to 2 months). Macular involvement was observed in all cases. Retinal detachment limited to the posterior pole was identified in 4 eyes, and total retinal detachment was observed in 6 eyes. A macular hole or a horseshoe tear was observed in 2 cases before surgery. A full-thickness paravascular linear retinal break was discovered in 1 case by optical coherence tomography before surgery (Figure). No paravascular linear retinal breaks were detected in the other 7 cases, despite diligent presurgical evaluation.

In all study eyes, the posterior hyaloid was present in the posterior pole and was tightly attached to the retinal vessels. Retinal detachment was more frequently seen in the temporal quadrants (in 7 of 10 eyes). The prolifer-
we examined the linear retinal breaks they were exclusively located along the posterior vascular arcades over areas of patchy chorioretinal atrophy. These breaks are apt to be missed and therefore, we recommend looking for linear retinal breaks along the posterior vascular arcades especially over areas of patchy chorioretinal atrophy. In contrast to little linear retinal breaks found along the temporal vascular arcades, these juxtapapillary microholes were generally round, smaller than the diameter of the adjacent retinal vessels, and mainly situated in the lower nasal quadrant at a distance of 1 to 2 disc diameters from the optic nerve. Moreover, exclusive distribution of these microholes over areas of patchy chorioretinal atrophy was not observed, and the resultant retinal detachments were shallow and stationary. Therefore, we believe that rhegmatogenous retinal detachments due to paravascular linear retinal breaks and patchy chorioretinal atrophy represent a specific category.

The detection of paravascular linear retinal breaks can be challenging before and during surgery. The main obstacles to identification include a lack of contrast due to underlying chorioretinal atrophy, obscureness of breaks within retinal folds, and the fact that the paravascular location in the posterior pole is generally not thoroughly searched for retinal breaks. We recommend looking for linear retinal breaks along the posterior vascular arcades, especially over areas of patchy chorioretinal atrophy, by gently flattening the retina with the assistance of a surgical instrument such as an extrusion needle (Figure). Unlike iatrogenic breaks induced during surgery, which commonly manifest in the retinal periphery as a horseshoe tear and may be associated with bleeding, the breaks described herein are linear in shape, appear to be exclusively located along the posterior vascular arcades over areas of patchy chorioretinal atrophy, and do not bleed. Retinal reattachment by vitrectomy with laser and gas tamponade was successful in 9 of 10 cases with a single operation. Further study is needed to verify this result.

To our knowledge, this is the first report of paravascular linear retinal breaks over areas of patchy chorioretinal atrophy causing rhegmatogenous retinal detachment among patients with pathologic myopia. In this series, these breaks were exclusively located along the posterior vascular arcades and over areas of patchy chorioretinal atrophy. Their distinct clinical characteristics include a linear shape with an axis parallel to the adjacent retinal vessels, a tendency to occur at the edges of posterior staphylomas, and a potential to result in progressive retinal detachment. These breaks are apt to elude detection before surgery, and careful examination along the posterior vascular arcades during vitrectomy helps to detect such abnormalities and to increase the success rate of surgery.

The most striking observation in this study was the exclusive location of paravascular linear retinal breaks over areas of patchy chorioretinal atrophy. In the absence of direct clinicopathologic correlation, we can only speculate about the pathogenesis of this association. Patchy chorioretinal atrophy has been associated with disappearance of the choriocapillaris and with damage to the retinal pigment epithelium. The outer retina over areas of patchy chorioretinal atrophy can be thinner, and this may correspond to retinal rarefaction around the retinal vessels, as demonstrated by histopathologic examinations of autopsied eyes. It is possible that the atrophic chorioretinal changes seen herein may have promoted the development of retinal breaks.

The linear shape and axis parallel to the adjacent retinal vessels suggest that unequal traction is being exerted from different directions. The propensity for breaks to develop at the margins of staphylomas, which represent the portion of sclera most strongly affected by excessive axial elongation in pathologic myopia, may contribute to the pathogenesis of these breaks. Moreover, the exclusive paravascular location indicates that the formation of retinal breaks may be associated with the status of the retinal vessels. It has been suggested that retinal arteriolar sclerotic changes and the inflexibility of the retinal vessels in patients with pathologic myopia could generate inward tractional force. The inflexibility of these vessels and the associated tangential contraction on the adjacent retina could be other important contributors to the formation of paravascular linear retinal breaks.

During surgery, we also observed that the vitreous was adhesive to the retina surrounding the linear retinal breaks. This suggests that vitreous traction is another important cause of retinal break development. Therefore, we hypothesize that the combination of axis elongation, vitreoretinal traction, tangential tension from adjacent vessels, and chorioretinal atrophic changes creates the unique shape and specific location of paravascular linear retinal breaks.

Several previous studies have reported retinal detachment due to juxtapapillary microholes. In contrast to the bigger linear retinal breaks found along the temporal vascular arcades in our study, these juxtapapillary microholes were generally round, smaller than the diameter of the adjacent retinal vessels, and mainly situated in the lower nasal quadrant at a distance of 1 to 2 disc diameters from the optic nerve. Moreover, exclusive distribution of these microholes over areas of patchy chorioretinal atrophy was not observed, and the resultant retinal detachments were shallow and stationary. Therefore, we believe that rhegmatogenous retinal detachments due to paravascular linear retinal breaks and patchy chorioretinal atrophy represent a specific category.

To our knowledge, this is the first report of paravascular linear retinal breaks over areas of patchy chorioretinal atrophy causing rhegmatogenous retinal detachment among patients with pathologic myopia. In this series, these breaks were exclusively located along the posterior vascular arcades and over areas of patchy chorioretinal atrophy. Their distinct clinical characteristics include a linear shape with an axis parallel to the adjacent retinal vessels, a tendency to occur at the edges of posterior staphylomas, and a potential to result in progressive retinal detachment. These breaks are apt to elude detection before surgery, and careful examination along the posterior vascular arcades during vitrectomy helps to detect such abnormalities and to increase the success rate of surgery.

The linear shape and axis parallel to the adjacent retinal vessels suggest that unequal traction is being exerted from different directions. The propensity for breaks to develop at the margins of staphylomas, which represent the portion of sclera most strongly affected by excessive axial elongation in pathologic myopia, may contribute to the pathogenesis of these breaks. Moreover, the exclusive paravascular location indicates that the formation of retinal breaks may be associated with the status of the retinal vessels. It has been suggested that retinal arteriolar sclerotic changes and the inflexibility of the retinal vessels in patients with pathologic myopia could generate inward tractional force. The inflexibility of these vessels and the associated tangential contraction on the adjacent retina could be other important contributors to the formation of paravascular linear retinal breaks.

During surgery, we also observed that the vitreous was adhesive to the retina surrounding the linear retinal breaks. This suggests that vitreous traction is another important cause of retinal break development. Therefore, we hypothesize that the combination of axis elongation, vitreoretinal traction, tangential tension from adjacent vessels, and chorioretinal atrophic changes creates the unique shape and specific location of paravascular linear retinal breaks.

Several previous studies have reported retinal detachment due to juxtapapillary microholes. In contrast to the bigger linear retinal breaks found along the temporal vascular arcades in our study, these juxtapapillary microholes were generally round, smaller than the diameter of the adjacent retinal vessels, and mainly situated in the lower nasal quadrant at a distance of 1 to 2 disc diameters from the optic nerve. Moreover, exclusive distribution of these microholes over areas of patchy chorioretinal atrophy was not observed, and the resultant retinal detachments were shallow and stationary. Therefore, we believe that rhegmatogenous retinal detachments due to paravascular linear retinal breaks and patchy chorioretinal atrophy represent a specific category.

The detection of paravascular linear retinal breaks can be challenging before and during surgery. The main obstacles to identification include a lack of contrast due to underlying chorioretinal atrophy, obscureness of breaks within retinal folds, and the fact that the paravascular location in the posterior pole is generally not thoroughly searched for retinal breaks. We recommend looking for linear retinal breaks along the posterior vascular arcades, especially over areas of patchy chorioretinal atrophy, by gently flattening the retina with the assistance of a surgical instrument such as an extrusion needle (Figure). Unlike iatrogenic breaks induced during surgery, which commonly manifest in the retinal periphery as a horseshoe tear and may be associated with bleeding, the breaks described herein are linear in shape, appear to be exclusively located along the posterior vascular arcades over areas of patchy chorioretinal atrophy, and do not bleed. Retinal reattachment by vitrectomy with laser and gas tamponade was successful in 9 of 10 cases with a single operation. Further study is needed to verify this result.
In conclusion, we report a unique type of rhegmatogenous retinal detachment due to paravascular linear retinal breaks distributed over areas of patchy chorioretinal atrophy along the posterior vascular arcades among patients with pathologic myopia. A careful search for such breaks along the posterior vascular arcades during vitrectomy may help to detect these abnormalities, especially in cases without identifiable retinal breaks before surgery.

Submitted for Publication: January 29, 2010; final revision received March 13, 2010; accepted March 26, 2010. Correspondence: Gezhi Xu, MD, PhD, Eye and Ears, Nose, and Throat Hospital and Department of Ophthalmology, Shanghai Medical School, Fudan University, Shanghai 200031, China (xugezhi@sohu.com).

Author Contributions: Drs Chen, Wang, Esmaili, and Xu contributed equally to this work.

Financial Disclosure: None reported.

Funding/Support: This study was supported by grant GZX/No.2007CB512205 from the National Basic Research Program of China, by grant GZX/No.PKJ2008-2008-113(3):298-403 from the National Basic Research Program of China, and by 211 Talents Start-up Program of China, by grant GZX/No.2007CB512205 from the National Basic Research Program of China.

REFERENCES