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Resolution of Myopic Macular Retinoschisis and Macular Hole With Topical Medical Therapy

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Abstract

Purpose: To report a case of myopic macular retinoschisis and full-thickness macular hole (FTMH) that both resolved with topical medical therapy alone. **Methods:** A retrospective chart review was performed. **Results:** A patient with myopic retinoschisis with an FTMH and increased symptoms of metamorphopsia was treated with topical medical therapy consisting of 1% prednisolone, 4 times daily, and 0.07% bromfenac, daily. Anatomic changes at 6 weeks included drawing together of the inner retinal edges around the MH and its subsequent closure with marked resolution of the myopic retinoschisis. **Conclusions:** Severe myopic macular retinoschisis and an FTMH both resolved with topical therapy consisting of steroidal and nonsteroidal anti-inflammatory medications. This rapid resolution contradicts current theories of the pathogenesis of myopic macular retinoschisis.

Keywords

macular hole, high myopia, myopic macular retinoschisis, retinoschisis, myopic traction maculopathy, myopic foveoschisis, prednisolone, bromfenac, topical eyedrops

Introduction

Myopic macular retinoschisis, one of the major causes of central vision loss and damage to the retina, affects 9.1% to 31.3% of highly myopic eyes.[1–6] The condition is believed to be multifactorial in origin and is also known as myopic foveoschisis and myopic traction maculopathy. Factors associated with myopic macular retinoschisis include stiffness of the inner retina as a result of retinal vessels, abnormalities of the vitreoretinal interface, and an outward protrusion of the eyeball involving the macular region and leading to staphyloma and globe ectasia.[3,4,7–11]

The mainstay of treatment for myopic macular retinoschisis is often observation. Despite marked retinal thickening and stretching, there may be integrity of the Müller cells that, while elongated and surrounded by cystic changes, allows for useful vision.[5,12] Surgery, involving vitrectomy, membrane peeling, and fluid–gas exchange, can result in improved vision[12–14]; however, surgery carries with it a risk of full-thickness macular hole (FTMH) development, which can lead to progressive retinal detachment (RD) in highly myopic eyes.[13]

Recently, medical therapy has been shown to allow closure of FTMHs without surgery.[15–17] Edema can be decreased in small MHs, those less than 200 µm in size, with treatment comprising steroids, nonsteroidal anti-inflammatory medication, carbonic anhydrase inhibitors, and antivascular endothelial growth factor (anti-VEGF) injections.[15] To our knowledge, spontaneous closure of an FTMH associated with resolution of myopic macular retinoschisis has been rarely reported.[18,19]

Case Report

A 72-year-old man presented with a 1-year duration of blurred vision in the right eye. Systemic medications taken daily included amlodipine/valsartan 10 to 160 mg, hydrochorothiazide 12.5 mg, and simvastatin 20 mg. His ophthalmic history included radial keratotomy in both eyes and previous cataract surgery with a posterior chamber intraocular lens in both eyes. The visual acuity (VA) was 20/50 OD and 20/20 OS. The axial length was 32.20 mm OD and 30.82 mm OS. A fundus examination initially showed myopic macular degeneration with lacquer cracks (Figure 1). Optical coherence tomography (OCT) showed myopic schisis with an inner layer lamellar MH and an intact outer photoreceptor layer (Figure 2A). Observation was recommended with a follow-up in 6 months.

The patient returned 6 months later with increased metamorphopsia of 2 month's duration. The fundus examination and OCT both showed an FTMH within the area of retinoschisis (Figure 2B). Treatment was initiated with 1% prednisolone acetate, 4 times daily, and 0.07% bromfenac, daily. After 6 weeks of treatment, the inner layer edges of the MH began to

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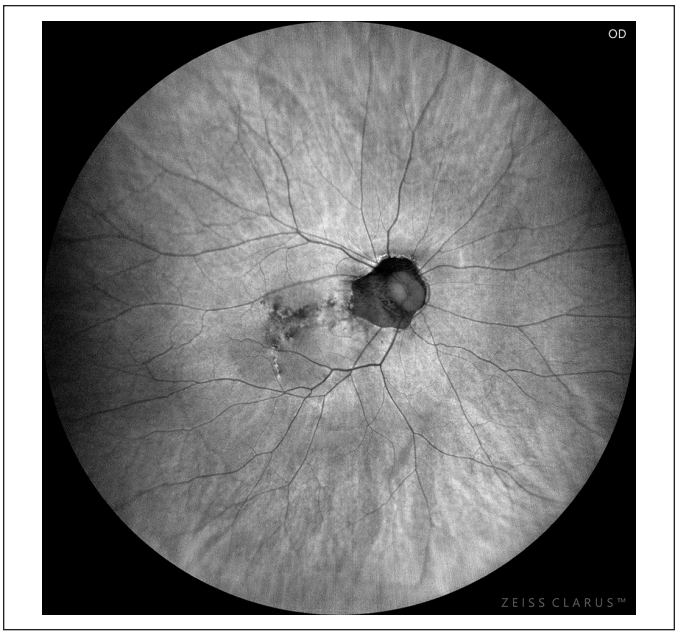

Figure 1. Fundus autofluorescence photograph of the right eye. Note the lacquer cracks and myopic fundus.

draw together (Figure 2C), but the basal diameter of the MH's outer layer increased. After 4.5 months of topical therapy, the MH closed, and the myopic retinoschisis markedly resolved with recovery of 20/20 vision (Figure 2D). An evaluation of the complete OCT scan throughout the macula found no evidence of vitreomacular traction release, visible posterior vitreous detachment (PVD), or breakage in the internal limiting membrane (ILM). Closure of the MH, along with resolution of central retinoschisis, were maintained after 7.5 months of treatment; however, mild localized retinoschisis remained superiorly (Figure 3).

Conclusions

Myopic macular retinoschisis is a marked thickening of the retina with elongation between the Henle fiber layer and the photoreceptor layer.[3,12] There are schisis cavities in many layers of the retina and elongation of Müller cells. Although the etiology is complex, associated factors include elongation and expansion of the globe in highly myopic eyes, stiffness of the inner retina associated with the vessels in the inner retina causing stretching of the retinal layers, with the outer retina maintaining its adhesion to the retinal pigment epithelium (RPE), and tractional forces on the retina due to vitreomacular traction, incomplete PVD, stiffness of the ILM, and epiretinal membranes.[3,6,13]

After starting topical medical therapy, marked resolution of the myopic macular retinoschisis and closure of the FTMH were seen. Anatomic changes, with the inner edges of the MH drawing together, have been typically found with medical therapy–induced MH closure[15] and were seen in this patient 6 weeks after beginning topical prednisolone and bromfenac. The MH closure, with a marked rapid resolution of myopic retinoschisis, was subsequently noted over a few months. This rapid change is not explained by current theories of the etiology of myopic macular retinoschisis, which have been conceived to explain its usual chronic nature.

In this patient, there was no change in globe ectasia. The theory of inner retinal stiffness with stretching of the outer retina to maintain RPE contact is not consistent with spontaneous improvement. Release of tractional forces on the retina by the detachment of preretinal cortical vitreous often noted during incomplete PVD is still a possibility, but no evidence of epiretinal membrane, resolution of vitreomacular traction, or clinical evidence of PVD were found in this patient. Breaks in the ILM have also been reported to result in rapid resolution and spontaneous regression of retinoschisis, but this also was not noted in this case, even with the entire OCT field of study being reviewed.[11]

Spontaneous resolution of myopic traction maculopathy occurs in 3.9% of cases,[11] both in eyes with and without PVD. However, spontaneous resolution of myopic macular retinoschisis has been rarely reported and has not previously been associated with an FTMH and treatment with topical medications.

Because the possibility exists of spontaneous myopic macular retinoschisis resolution without change in globe ectasia, any sudden decrease in inner retinal stiffness, or visible acute traction release or break in the ILM, there may be an unknown component that may rapidly change, explaining the rapid resolution. In this patient, closure of the MH after topical therapy and decreased fluid passage could be an important factor. Tornambe's hydration theory suggests that an ILM break in the central macula can cause paracentral edema, resulting in an MH.[20] Closure of the MH could then decrease edema in the central macula, perhaps allowing the retinoschisis to resolve. Müller cell dysfunction is another cause of macular retinoschisis that could improve and result in a relatively rapid resolution.[5] These cells also control intracellular fluid and metabolic water not associated with fluorescein angiographic leakage. A dysfunction in their structure can lead to an inflow of fluid.[21,22] Triamcinolone has been shown to decrease fluid inflow into the cells, inhibiting osmotic swelling of the cells and stimulating clearance of fluid from retinal tissue. Topical steroids could have contributed to the inhibition of osmotic swelling in the area of retinoschisis, especially with medication being allowed to diffuse into the retinal tissue by an FTMH.

Medical therapy to decrease edema, resulting in MH closure, has been noted in an increasing number of reports.[15,17,23] A key finding is the drawing together of the inner retinal edges, as noted at 6 weeks after topical medical therapy (Figure 2C). MH closure using this modality usually occurs in the treatment of small MHs, about 200 μm or less in size. In this patient's case, both surgical management and topical medical therapy were discussed. The possibility of closure of the MH with significant myopic retinoschisis was felt to be unlikely, but the patient initially chose medical therapy. Medical therapy was started as a trial to close the MH, and unexpectedly this led to resolution of the myopic macular retinoschisis, closure of the MH, and significant improvement of vision to 20/20.

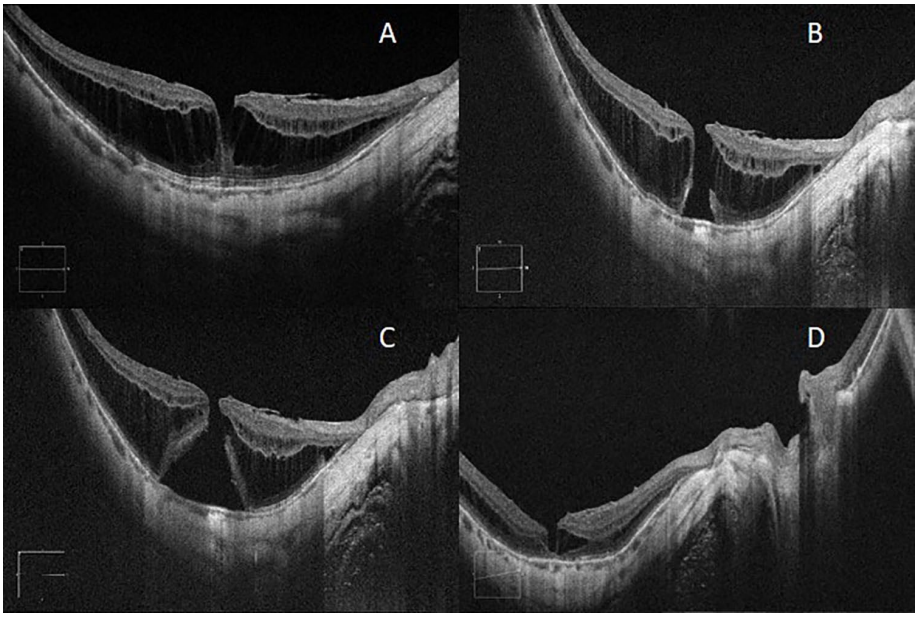

Figure 2. (A) Optical coherence tomography (OCT) of the right eye. Note the macular retinoschisis and thinning of the inner fovea with an intact outer retina. (B) OCT of the right eye shows a full-thickness macular hole (MH). The patient noted increased metamorphopsia. (C) Six weeks after beginning treatment, OCT of the right eye shows the inner edges of the MH beginning to close with enlargement of the base diameter. (D) Closure of the MH with marked resolution of macular retinoschisis.

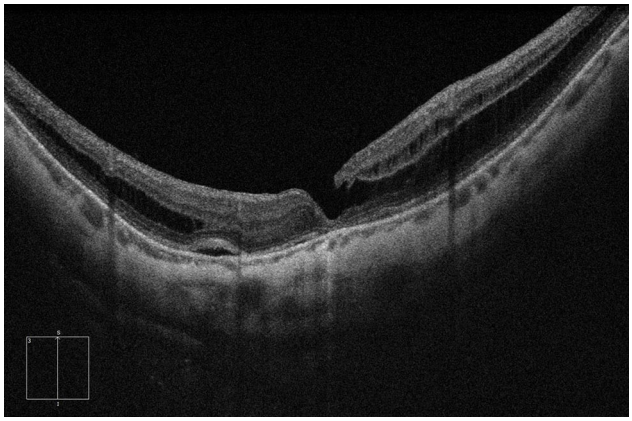

Figure 3. Inferior-superior optical coherence tomography of the right eye 7.5 months after topical treatment. Note the stable closure of the macular hole, increased superior edema, and recurrent retinoschisis.

To our knowledge, this case is the first to describe the closure of an MH and resolution of its associated myopic macular retinoschisis after treatment with topical medication. Facilitating MH closure in myopic macular retinoschisis could be a potential treatment that has not previously been considered and requires future study.

Authors' Note

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Ethical Approval

This case report was conducted in accordance with the Declaration of Helsinki. The collection and evaluation of all protected patient health information was performed in a Health Insurance Portability and Accountability Act (HIPPA)-compliant manner.

Statement of Informed Consent

Informed consent was obtained for publication of all photographs and images included herein.

Declaration of Conflicting Interests

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