

Vitreomacular Traction Causing Idiopathic Full-Thickness Macular Hole Determined By Spectral-Domain Optical Coherence Tomography

Vijay Kumar Srivastava¹, M Shivakumar², Pratik Kataria³, Vandana Parasar³

¹Professor and Head, Department of Ophthalmology, Rajarajeswari Medical College and Hospital, Bengaluru, Karnataka, India, ²Professor, Department of Ophthalmology, Rajarajeswari Medical College and Hospital, Bengaluru, Karnataka, India, ³Postgraduate Student, Department of Ophthalmology, Rajarajeswari Medical College and Hospital, Bengaluru, Karnataka, India

Abstract

Macular holes commonly occur as an age-related primary idiopathic condition, not associated with other ocular problems or antecedent events. The primary mechanism for idiopathic macular hole formation is the dynamic vitreofoveal traction caused by a perifoveal posterior vitreous detachment (PVD). A 56-year-old woman presented with diminution of vision and metamorphopsia in the left eye. The anterior segment examination was normal in both eyes. A dilated fundus evaluation of the left eye showed a small full-thickness macular hole (FTMH) with cystoid foveal thickening. The optical coherence tomography (OCT) showed a stage 2 small FTMH with vitreomacular traction. Amsler grid showed a central distortion while Watzke-Allen test was positive. An FTMH is a common cause for impairment of central vision. The clinical symptoms include diminution of central vision, metamorphopsia, photopsia, and micropsia. OCT is important for the assessment of extent, progression, and resolution of macular holes. The stage 1 impending macular holes are observed, as they have a better chance for spontaneous closure. The stage 2 or greater FTMHs, with a smaller chance of spontaneous closure, are treated with pars plana vitrectomy with internal limiting membrane peeling. Recently, intravitreal injection of ocriplasmin has been used for pharmacological induction of PVD.

Key words: Fovea centralis, Macula lutea, Optical coherence tomography, Retinal diseases, Retinal perforations, Visual acuity, Vitreous body, Vitreous detachment

INTRODUCTION

The clinical spectrum of a vitreomacular interface disease includes vitreomacular adhesion (VMA), vitreomacular traction (VMT), macular epiretinal membrane (ERM), full-thickness macular hole (FTMH), lamellar macular hole, and macular pseudohole.¹

The idiopathic macular hole was recognized as a unique clinical entity more than a century ago.² Macular holes commonly occur as an age-related primary idiopathic condition, not associated with other ocular problems or antecedent events.³ The tangential traction of an attached

prefoveal vitreous cortex, causing foveolar detachment and dehiscence, has been hypothesized to cause a macular hole.⁴

The optical coherence tomography (OCT) is a decisive tool in the evaluation of the extent of VMA or traction in a case of an idiopathic macular hole, as clinical examination alone may not be sufficient.⁵ This case emphasizes the importance of OCT examination in the determination of the diagnosis and the etiology of an idiopathic macular hole.

CASE REPORT

A 56-year-old woman presented with complaints of gradual, painless, progressive diminution of vision and metamorphopsia in the left eye since 5 months. On presentation, her best-corrected visual acuity was 6/60 in the left eye and 6/6 in the right eye.

Any history of trauma or ocular inflammation before the onset of symptoms was absent. No history of any systemic

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www.ijss-sn.com

Month of Submission : 11-2015
 Month of Peer Review : 12-2015
 Month of Acceptance : 01-2016
 Month of Publishing : 01-2016

Corresponding Author: Dr. Pratik Kataria, Department of Ophthalmology, Rajarajeswari Medical College and Hospital, Bengaluru, Karnataka, India. Phone: +91-9731915381. E-mail: prakat256@gmail.com

disorder was present. Gross examination of both eyes was unremarkable. Facial symmetry and ocular posture were maintained. The anterior segment examination was normal in both eyes. Intraocular pressure in both eyes was found to be within normal limits.

A dilated fundus examination of the left eye showed a small FTMH with cystoid foveal thickening (Figure 1). Rest of the fundus examination in the left eye was normal. The high-resolution spectral-domain OCT of the macula in the left eye showed a stage 2 small FTMH with VMT, along with foveal thickening and intraretinal cystic changes (Figure 2). The inner retina shows a slightly eccentric dehiscence with a centrifugal displacement of the photoreceptor layer. Amsler grid showed a central distortion while the Watzke-Allen test was positive in the left eye.

The fundus examination in the fellow eye was normal with no evidence of a vitreoretinal interface disease. A diagnosis of stage 2 FTMH with VMT in the left eye was achieved. Surgical intervention with pars plana vitrectomy and internal limiting membrane peeling was offered, but the patient elected to wait and observe for spontaneous resolution.

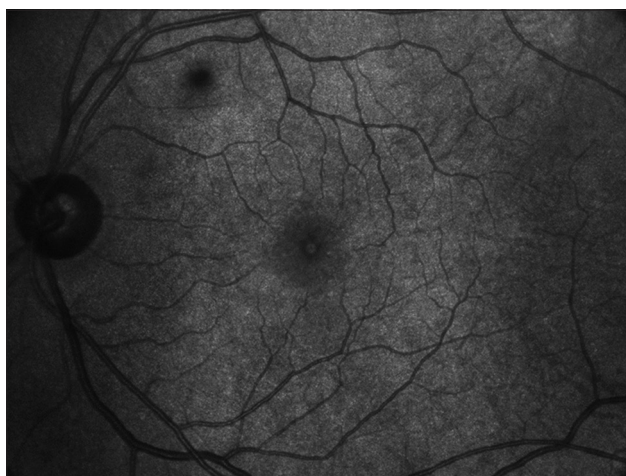


Figure 1: A red-free fundus photograph of the left eye showing a small full-thickness macular hole with cystoid foveal thickening

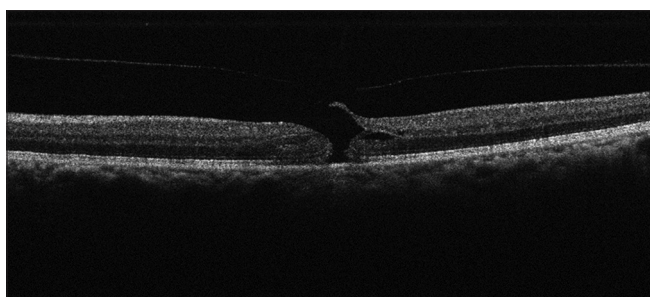


Figure 2: High resolution spectral-domain optical coherence tomography of the macula in the left eye showing a stage 2 small full-thickness macular hole with vitreomacular traction, along with foveal thickening and intraretinal cystic changes

DISCUSSION

An FTMH is a relatively common cause of impairment of central vision, most commonly occurring in females aged 60-70 years.

In eyes with accelerated vitreous liquefaction before sufficient weakening of the vitreoretinal adhesion, posterior vitreous detachment (PVD) may lead to serious complications. High-resolution imaging of the vitreoretinal interface has allowed identification of an association between PVD and the early stages of idiopathic macular hole.⁶ This has led to a hypothesis that the primary mechanism for idiopathic macular hole formation is the dynamic vitreofoveal traction caused by a perifoveal PVD.⁷

Idiopathic macular hole occasionally develops in eyes with pre-existing spontaneous or surgical PVD, which may be suggestive of alternative mechanisms of macular hole formation such as primary degeneration of inner retinal layers at the central fovea.⁸ However, the traction-induced foveal disruption occurring before or coincident with the vitreofoveal separation may lead to inner foveal damage with destabilization of the outer foveal layer.⁷

FTMHs are commonly classified into four stages:⁴

- Stage 0: VMA.
- Stage 1a: “Impending” macular hole with VMT.
- Stage 1b: “Occult” macular hole with VMT.
- Stage 2: “Small” FTMH $\leq 400 \mu\text{m}$ in diameter with VMT.
- Stage 3: “Full-size” FTMH $>400 \mu\text{m}$ in diameter with VMT.
- Stage 4: Includes full-size FTMH with complete PVD.

The clinical symptoms of VMT include slowly progressive diminution of central vision, metamorphopsia, photopsia, and micropsia, which may not correlate with clinical findings.¹ The signs of VMT can be observed clinically with slit lamp biomicroscopy, ultrasonography, and OCT.⁹ The spectral-domain OCT is an important tool for assessment of extent, progression, and resolution of macular holes. It yields valuable information regarding the ultrastructural changes at the vitreomacular interface and within the retinal layers.⁵

The stage 1 impending macular holes are observed and not treated surgically, as they have about a 50% chance for spontaneous closure with resolution of symptoms.¹⁰ Spontaneous resolution with restoration of the normal foveal contour in other cases of FTMH (stage 2-4) is very rare, occurring only in 2-4% of eyes, probably secondary to ERM formation.¹¹ The spontaneous resolution of VMT is more likely in the eyes with less vitreous surface adhesion and without ERMs.⁵

Multiple mechanisms have been proposed for the spontaneous resolution of a macular hole, including release of traction following complete detachment of the posterior hyaloid from the foveal area, formation of a contractile ERM causing closure by shrinkage, bridging of the retinal tissue across the macular hole, and proliferation of cells at the base of the macular hole.¹²

The Müller cells may play an initial role in the spontaneous closure of an FTMH with the restoration of the outer retina as the photoreceptors themselves cannot proliferate.² The centripetal traction-induced by the extension and proliferation of Müller cells may facilitate adhesion of other disrupted retinal layers including the IS/OS junction.² The astrocytes which are present in the inner retina may also be implicated in the process of spontaneous closure, as they are also known to participate in the formation of retinal glial scars.²

The stage 2 or greater FTMHs, with a small chance of undergoing spontaneous closure, are treated surgically by a pars plana vitrectomy with or without internal limiting membrane (ILM) peeling.³ Surgical intervention is usually required in eyes with a larger vitreous surface adhesion or a coexisting ERM.¹³

For many years, researchers have been investigating pharmacological tools to induce a complete PVD.¹⁴ Recently, intravitreal injection of ocriplasmin, a recombinant form of human plasmin, has been used as a monotherapy as well as an adjunct to surgical vitrectomy.¹⁵ It has been found to cause resolution of VMT with closure of macular hole in over 25% of the eyes, but the results are generally inferior to surgical vitrectomy.¹⁶

CONCLUSION

- The idiopathic macular hole represents a complication of a slowly evolving early stage of an age-related PVD.
- An OCT examination is obligatory to study both the progression and resolution of macular holes, except in straightforward cases.
- Follow-up using OCT examination enables monitoring for a spontaneous closure in early cases so that a complex surgery may be avoided. However, the risk of observation versus surgery should be explained to the patient.

- Surgical intervention with pars plana vitrectomy with ILM peeling may be considered for advanced cases, especially in cases with a larger vitreous surface adhesion or a coexisting ERM.

ACKNOWLEDGMENT

Dr. Ganesh Sathyamurthy. MS, Vitreoretinal Surgeon (Consultant), Rajarajeswari Medical College and Hospital, Bengaluru, Karnataka, India.

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How to cite this article: Srivastava VK, Shivakumar M, Kataria P, Parasar V. Vitreomacular Traction Causing Idiopathic Full-Thickness Macular Hole Determined by Spectral-Domain Optical Coherence Tomography. *Int J Sci Stud* 2016;3(10):190-192.

Source of Support: Nil, **Conflict of Interest:** None declared.