Spontaneous Closure of a Full-Thickness Stage 2 Idiopathic Macular Hole without Posterior Vitreous Detachment

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Key Words

Macular holes · Posterior vitreous detachment · Retina · Optical coherence tomography

Abstract

Objective: We report a 50-year-old female patient with a stage 2 idiopathic macular hole that closed spontaneously. **Method:** The case is presented on the basis of an observational case report. **Results:** The stage 2 idiopathic macular hole closed spontaneously in 6 weeks with a lamellar defect in the outer retina due to the formation of the bridging retinal tissue, but without any evidence of the common mechanisms of spontaneous closure such as posterior vitreous detachment or epiretinal membrane formation.

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Introduction

An idiopathic macular hole was identified as a unique clinical entity more than 100 years ago [1]. Most macular holes occur as an age-related primary idiopathic condition, unrelated to other ocular problems or antecedent events. The hallmark inciting event of an idiopathic macular hole formation is hypothesized to be focal shrinkage of the vitreous cortex in the foveal area [2].

Gass [3, 4] classified macular holes into 4 stages. Stage 1, also known as ‘impending macular holes,’ is characterized by a foveal cyst within the retina. Stages 2–4 include full-thickness macular holes, which are further divided into smaller holes (<400 µm in diameter

(stage 2)), holes larger than 400 μm in diameter (stage 3) and with a complete posterior vitreous detachment (stage 4).

It has been clinically established that stage 1, impending macular holes, have a 50% chance for spontaneous closure with the resolution of symptoms [5]. They are therefore observed and not surgically treated. However, spontaneous resolution with hole closure and a restoration of the normal foveal contour is very rare in full-thickness macular holes (stages 2–4). It occurs in 2–4% of the eyes [6, 7]; therefore, these cases are usually treated surgically by pars plana vitrectomy, with or without internal limiting of the membrane peeling.

We report a 50-year-old female patient with a stage 2 idiopathic macular hole that closed spontaneously. She also had a lamellar defect in the outer retina due to the formation of the bridging retinal tissue, but without any evidence of the common mechanisms of spontaneous closure such as posterior vitreous detachment or epiretinal membrane formation.

Case Report

The patient presented to us in May 2013 with a right eye vision diminution, which started 1 month ago. On examination, the best-corrected visual acuity was 20/125 in the right eye and 20/30 in the left eye. Slit lamp examination revealed a clear cornea and a pupil that was normal in size and reacting to light; early cataract in both eyes was also detected. The posterior segment showed the presence of a full thickness centric macular hole (stage 2) in the right eye. No posterior vitreous detachment was clinically identified. Further examination with the Amsler grid test demonstrated central metamorphopsia in the right eye. In the other eye, the fundus, the disc and the retinal vasculature appeared healthy; the macula was within normal limits. Intraocular pressure measured 14 mm Hg in both eyes, measured by applanation tonometry.

The Watzke-Allen test was positive for the right eye. Optical coherence tomography (OCT) was performed for the right eye, which revealed a full-thickness macular hole (stage 2) with cystic spaces suggesting an accumulation of secondary vitreous fluid and no posterior vitreous detachment (fig. 1).

Various treatment options, including conservative management, were discussed with the patient. She noticed an improvement in her visual acuity after 6 weeks. The best corrected visual acuity was checked and was 20/40 on the Snellens chart for the right eye and 20/30 for the left eye. Fundus examination of the right eye revealed no evidence of a macular hole, and the Watzke-Allen test was negative. OCT revealed the closure of the macular hole with the resolution of the cystic spaces and bridging of the inner retinal layers with lamellar defects without any posterior vitreous detachment or glial tissue proliferation (fig. 2).

The follow-up after 12 weeks showed persistent outer lamellar defects on the OCT; visual acuity improved marginally to 20/30 (fig. 3).

Discussion

In our patient, macular hole closure was confirmed by OCT examination, showing a sensory retina bridging over the former area of the macular hole with small outer layer defects. Similar mechanisms have been described previously [8–10]. The bridging of the retinal tissue allowed the resolution of the cystoid spaces by preventing the influx of

vitreous fluid into intraretinal spaces and therefore leading to a spontaneous closure of the macular hole.

Four explanations have been proposed for the spontaneous resolution of a macular hole: (1) complete detachment of the posterior hyaloid from the foveal area leading to a release of traction; (2) cell proliferation at the base of the hole; (3) formation of a contractile epiretinal membrane resulting in shrinkage and closure of the hole, and (4) bridging of the retinal tissue across the hole [11].

Out of the 4 mechanisms mentioned above, posterior vitreous detachment and epiretinal membrane formation may or may not always be evident in patients with spontaneous closure of a macular hole, but the bridging of the sensory retina and the smaller size of the macular hole appear to be the most consistently reported findings for the spontaneous closure of macular holes [12, 13]. As to the origin of the bridging of the retinal tissue, proliferation of the retinal postmitotic neurosensory cells has been proposed, but could not be identified; proliferation of glial or retinal pigment epithelial cells has also been suggested [14]. Since the concept of cell proliferation as a mechanism of macular hole closure is still speculative, the exact mechanism of how the spontaneous macular hole closure with maintained normal retinal structure occurs is still unclear.

Disclosure Statement

None of the authors have financial or proprietary interests in any material or method mentioned.

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Fig. 1. Right eye full-thickness macular hole with cystic spaces and without any posterior vitreous detachment visible on OCT.

Fig. 2. Right eye OCT resolution of a macular hole at 6 weeks with a tiny residual and an outer lamellar defect in the outer retina due to the formation of the bridging of the retinal tissue, but without any posterior vitreous detachment or epiretinal membrane formation.

Fig. 3. Right eye OCT at 16 weeks, showing a persistent outer lamellar defect decreasing in size with further bridging of the retinal tissue proliferation.