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## **Macular Hole Following Phakic Intraocular Lens Implantation: a Case Report**

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### **Abstract**

#### **BACKGROUND**

Phakic intraocular lens (pIOL) implantation has been commonly prescribed and is considered as a safe and effective option for correcting high myopia. However, it is associated with multiple complications.

#### **CASE SUMMARY**

This report describes a case of full-thickness macular hole (MH) in a patient with a history of bilateral pIOL implantation for the correction of myopia of -12.00 diopters in both eyes seven months ago. The MH closed after pars plana vitrectomy with internal limiting membrane removal, and the best-corrected visual acuity improved to 20/40 in the left eye.

#### **CONCLUSION**

In rare cases, MH can occur following pIOL. In this present case report, we analyzed the formation process of MH following the surgery and emphasized that it is important to inform highly myopic patients about the risk of MH occurrence, while being aware of the symptoms of this complication.

**Key Words:** Case report; Macular hole; Phakic intraocular lens; Pars plana vitrectomy; High myopia; Impaired visual acuity

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**Core Tip:** Although phakic intraocular lenses have been commonly prescribed and are safe and effective, they are still associated with multiple complications. One of the rare complications is a full-thickness macular hole, occurring after phakic intraocular lens implantation. Therefore, it is important to inform highly myopic patients about the risk of MH occurrence and be attentive to the symptoms of this complication.

## **INTRODUCTION**

Phakic intraocular lens (pIOL) implantation has been commonly prescribed since the 1980s. It is accepted as a safe and effective option for correcting high myopia in patients ineligible for corneal refractive surgery.<sup>1</sup> However, complications associated with pIOL include the reduced density of central endothelial cells, development of pigmentary dispersion, cataracts, and glaucoma<sup>1</sup>. In addition, a few cases of vitreoretinal complications, such as giant retinal tears and retinal detachment, have been reported<sup>2</sup>.

**1** We present a rare occurrence of macular hole (MH) as a complication of pIOL implantation for the treatment of high myopia.

## **7 CASE PRESENTATION**

### ***Chief complaints***

A 29-year-old female presented with a ten-day history of impaired visual acuity (VA) and distorted vision in the left eye.

### ***History of present illness***

The patient had undergone pIOL implantation in the posterior chamber for the correction of myopia of -12.00 diopters in both eyes seven months prior to the onset of symptoms described above. PIOL implantation improved the vision of the patient whose VA was 20/20 in both eyes after the treatment.

### ***History of past illness***

The patient had no history of systemic illness or obvious changes in the retina or vitreous before the pIOL implantation.

### ***Personal and family history***

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### ***Physical examination***

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The initial examination showed that the best corrected visual acuity (BCVA) of this patient was 20/20 in the right eye and 20/100 in the left eye with normal intraocular pressure. The axial length was 27.18 mm in the right eye and 27.00 mm in the left eye. The slit-lamp examination showed that the pIOL was present in both eyes and an unremarkable anterior segment in the rest of the area. The fundusoscopic examination revealed a round-shaped MH in the left eye [Fig. 1a], and the appearance of a tessellated fundus with a tilted disc in bilateral eyes.

#### *Laboratory examinations*

None

#### *Imaging examinations*

An optical coherence tomography (OCT) revealed a full-thickness MH in the left eye with surrounding subretinal fluid and perifoveal cystic changes at the rim of the hole [Fig. 1d], which had a diameter of 520 mm horizontally and 482 mm vertically. In addition, vitreoretinal traction that was induced by partial posterior vitreous detachment (PVD) was documented at the fovea, as well as in the area close to the superior vascular arcade, with an increased retinal thickness and retinal layer splitting [Fig. 1c]. It should be noted that these findings were not present in the preoperative OCT examination of both eyes [Fig. 2]. The fundus autofluorescence image of the left eye revealed hyper-autofluorescence surrounding the MH [Fig. 1b].

#### **FINAL DIAGNOSIS**

Full-thickness MH following bilateral pIOL implantation.

#### **TREATMENT**

The patient underwent 23-gauge three-port pars plana vitrectomy with triamcinolone-assisted PVD induction. Subsequently, the internal limiting membrane was removed by staining with 0.5% indocyanine green followed by gas tamponade (12% C3F8).

## OUTCOME AND FOLLOW-UP

The MH closed at two weeks after the operation [Fig. 1e] and the BCVA had improved to 20/40.

## <sup>2</sup> DISCUSSION

Most MHs are idiopathic and frequently occur in the elderly and women. However, they may also be correlated to high myopia, trauma, and surgeries, including vitrectomy, pneumatic retinopexy, scleral buckling, laser-assisted *in situ* keratomileusis, and cataract surgery<sup>3,4</sup>. In rare cases, MH can occur following pIOL<sup>5,6</sup>.

To our knowledge, three cases have been described in two previous reports<sup>5,6</sup>. In one of these cases, the MH had a similar morphology as the traumatic MH with a rhomboid-shaped appearance. In addition, defects in the internal limiting membrane were present, and a lack of subretinal fluid and cystic formation was observed. The remaining two cases had similar features as an idiopathic MH with a round-shaped appearance and edema in the perifoveal retina surrounded by subretinal fluid. The present case had characteristics similar to the latter MHs.

Previous case reports have proposed that a MH might have already existed and was misdiagnosed before pIOL implantation; otherwise, it might be a part of the natural course of high myopic change or induced by the surgery<sup>7</sup>. In the present case, preoperative OCT examination revealed normal morphology at the fovea in both eyes, without PVD, posterior staphyloma, or foveoschisis, which differs from myopic MH. Therefore, it is likely that the PVD formation was related to the surgery and subsequently led to the occurrence of MH<sup>8,9</sup>.

As suggested by Gass, during the PVD process, MH formation is initiated by the tangential traction of the premacular vitreous cortex at the retinal interface, which then leads to foveal dehiscence that progresses from foveolar detachment to a mature full-thickness MH<sup>10</sup>. Highly myopic patients tend to show PVD at a younger age, and the surgery might accelerate the process. Luna *et al* have demonstrated that PVD with

vitreous liquefaction was not present prior to LASIK, but was documented afterwards in 24%–50% of highly myopic eyes<sup>11</sup>. In recent years, in-depth research on posterior precortical vitreous pocket (PPVP) has proved that the aqueous can flow into the PPVP through the Cloquet's canal<sup>10</sup>, therefore, we speculated that the flow of aqueous humor caused by fluctuation of intraocular pressure during the surgery was drained into the premacular space, which initiated the process of PVD.

<sup>3</sup> Early in the PVD process, the vitreous detaches from the perifoveal area, but maintains attachment at the central fovea, the optic disc, and the vascular arcade area, where vitreoretinal attachments are stronger. Over time, assisted by vitreous movement, anteroposterior tractional forces at the fovea were generated, leading to pathogenic vitreous traction. Mori *et al* presented the hypothesis <sup>4</sup> that dynamic traction, attributable to vitreous movement associated with ocular saccades, is the most important type of vitreomacular traction in MH formation<sup>12</sup>. A larger PPVP in highly myopic eyes is associated with a greater level of concentrated force and a more pronounced shift in the force vector direction of the anteroposterior tensile stress<sup>10</sup>. The traction force varies depending on the configuration and angle of vitreous insertion into the peripheral retina<sup>12</sup>. Therefore, as the OCT revealed, the vitreofoveal attachment was released in the right eye [Fig. 3], which led to a slight abnormality of retinal morphology. In contrast, the MH was formed in the left eye.

Furthermore, focal cellular proliferation of the vitreous face and weak adherence of the retinal pigment epithelium-photoreceptor should not be neglected. In addition, <sup>1</sup> the effect of various refractive surgeries on choroidal thickness (CT) has been reported in recent years. Fang *et al.* evaluated the changes in choroid thickness after pIOL surgery, and found that the CT in the foveal was significantly increased at three months after the surgery, and then gradually recovered<sup>13</sup>. In all three cases, which had similar features as the present case, the MH formed at 4, 7, and 18 mo after the surgery. Zeng *et al* considered that choroidal thinning might be one of the reasons for the development of MH<sup>14</sup>. Therefore, the fluctuation of CT might play a role in the formation of MHs; however, the precise relationship needs to be elucidated by more clinical studies.

## **CONCLUSION**

It remains difficult to determine the risk factors for MH formation after refractive surgery. Thus, further case-control studies are needed to address this question. Although the incidence rate of MH occurrence is low, the present case report emphasizes that it is important to inform patients about the risk of MH occurrence and be aware of the symptoms of this complication. The patients can then receive assistance promptly and efficiently after the onset of MH formation.



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