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Idiopathic macular hole with asteroid hyalosis
Two case reports

Takakuni Kitagaki, MD, Hiroyuki Suzuki, MD, PhD, Ryohsuke Kohmoto, MD, Shou Oosuka, MD,
Masanori Fukumoto, MD, PhD, Takaki Sato, MD, PhD, Takatoshi Kobayashi, MD, PhD, Teruyo Kida, MD, PhD,
Tsunehiko Ikeda, MD, PhD*

Abstract

Introduction: Asteroid hyalosis (AH) is characterized by mild vitreous-body liquefaction and a reduced likelihood of posterior vitreous detachment (PVD). Here, we report the clinical features of 2 cases of macular hole (MH) in eyes with AH. The extent of retinal vitreous adhesion at the time of vitreous surgery, the presence or absence of iatrogenic retinal breaks, and the postoperative course was examined in regard to the association with AH.

Case presentation: Case 1 involved a 67-year-old female with decreased visual acuity in her left eye. Although preoperative optical coherence tomography examination revealed complete PVD with operculum around the fovea central region, vitreoretinal adhesion was quite strong starting at the midperiphery of the fundus, and an iatrogenic retinal break was formed at the inferior site during vitreous surgery. Endophotocoagulation was performed, and no onset of retinal detachment (RD) postsurgery was observed. Case 2 involved a 74-year-old male with metamorphopsia in his right eye. Preoperative perifoveal PVD was observed, yet vitreoretinal adhesion was strong starting at the midperiphery of the fundus and multiple iatrogenic retinal breaks had formed on the inferior, superior, and temporal sites. Moreover, RD occurred during surgery. Endophotocoagulation and gas tamponade was performed; however, postoperative RD requiring multiple reoperations occurred.

Conclusion: As with normal MH, MH with AH is thought to be caused by perifoveal PVD, yet as the vitreoretinal adhesion was found to be quite strong starting at the midperiphery of the fundus, we decided to keep the artificial PVD within a range where adhesion was loose.

Abbreviations: AH = asteroid hyalosis, MH = macular hole, PVD = posterior vitreous detachment, RD = retinal detachment.

Keywords: asteroid hyalosis, iatrogenic retinal break, posterior vitreous detachment, retinal detachment, vitreous surgery

1. Introduction

In 1894, Benson[1] reported asteroid hyalosis (AH) as being a degenerative diseases of the vitreous, and indicated that in eyes with AH, particulate opacities such as phospholipids and mucopolysaccharides were scattered in the vitreous cavity, with 80% to 90% of which being unilateral. In that study, Benson[1] pointed out that the vitreous liquefaction was slight and that it was difficult for posterior vitreous detachment (PVD) to occur as

a characteristic of eyes with AH,[2–6] as well as that it may complicate epiretinal membrane and macular edema.[7–10] To the best of our knowledge, there has only been 1 previous report indicating the occurrence of a macular hole (MH) in eyes with AH.[9] Here, we report the clinical features of 2 cases with AH that underwent vitreous surgery for an MH.

2. Case presentation

2.1. Case 1

Case 1 involved a 67-year-old female who presented with the primary complaint of decreased visual acuity (VA) in her left eye. Initial fundoscopy examination revealed poor, fundus visibility due to AH, yet not MH was detected. However, optical coherence tomography (OCT) image clearly revealed the existence of a MH. Complete PVD with operculum around the MH had occurred (Fig. 1 A). The refraction was nearly emmetropia and the corrected VA was 0.3, and the patient had undergone cataract surgery 1 year before presentation.

Vitreous surgery was performed by first removing the core vitreous gel together with AH (Fig. 2 A), followed by triamcinolone acetonide being applied to the macula to prepare an artificial PVD from around the MH. Although the adhesion of the posterior pole was easily dissociated (Fig. 2 B), the vitreous gel was strongly adhered to the retina in a planar fashion from the mid-peripheral area of the retina (Fig. 2 C) and the boundary line of the vitreoretinal adhesion was raised in a "tent-like" shape during the artificial PVD creation over the entire circumference. One iatrogenic retinal break
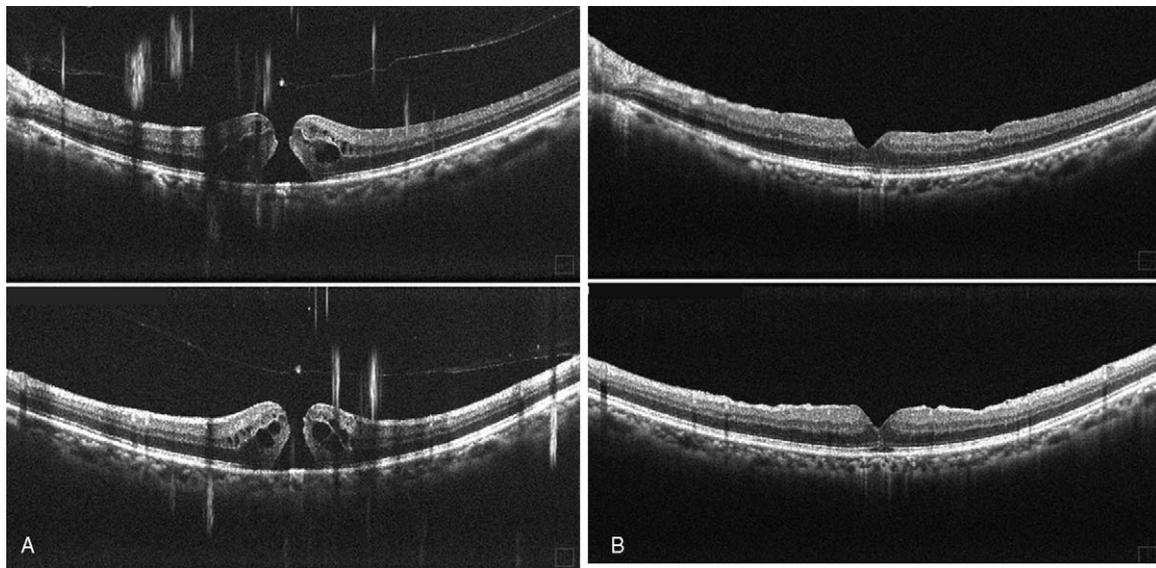
Figure 1. Optical coherence tomography (OCT) imaging of Case 1. Complete posterior vitreous detachment (PVD) with operculum around the macular hole (MH) occurred preoperatively (A). After vitreous surgery, the MH closed and the patient's corrected visual acuity (VA) improved to 0.7 (B).

was formed at the 6 o'clock position of the lower mid-peripheral area, so endophotocoagulation was performed at that same area. Next, the inner limiting membrane surrounding the MH was stained with Brilliant Blue G (Fig. 2 D) and peeled off, and a fluid-air exchange and gas tamponade by 20% sulfur hexafluoride (SF6) was performed. Postsurgery, the MH closed and the patient's corrected VA improved to 0.7 (Fig. 1 B).

2.2. Case 2

Case 2 involved a 74-year-old male who presented with the primary complaint of metamorphopsia in his right eye. Similar to Case 1, initial fundoscopy examination revealed poor fundus visibility due to AH and no MH was detected. However, and as with Case 1, OCT image clearly revealed the existence of a MH.
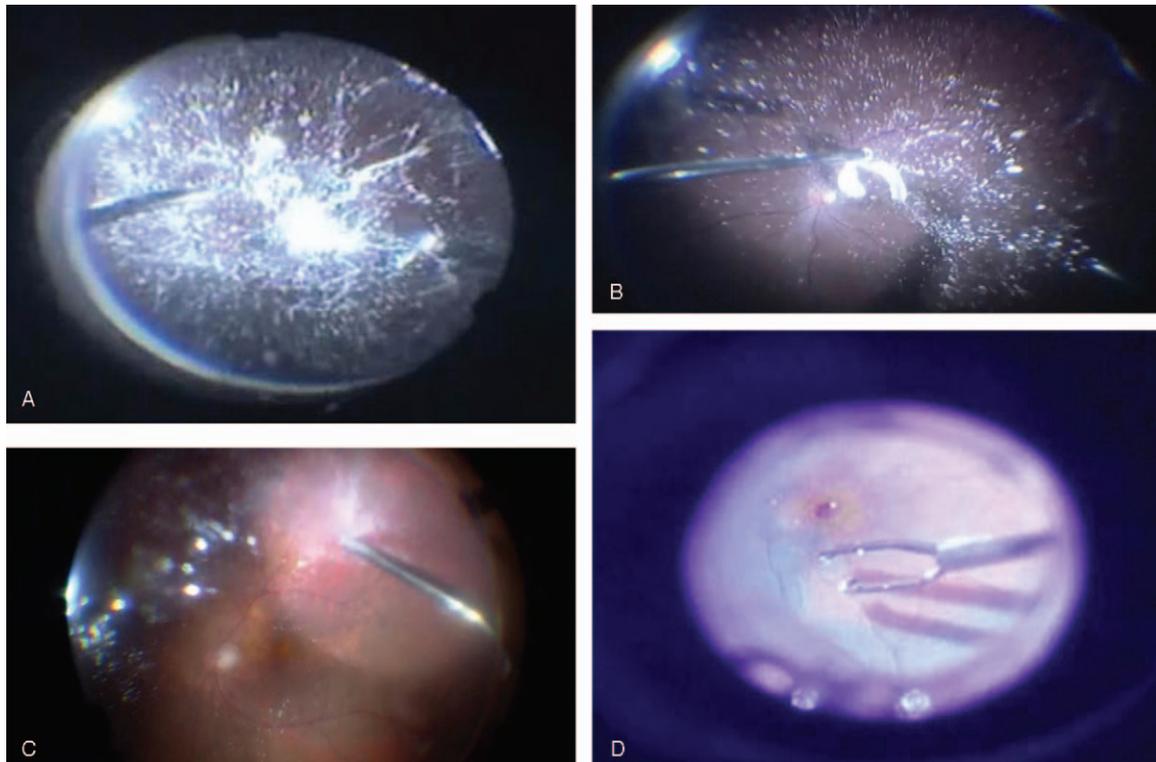
Figure 2. Intraoperative findings of the left eye of Case 1. The core vitreous gel together with AH was removed (A), thus making it easy to create an artificial PVD from around the MH (B). Vitreous gel was strongly adhered to the retina in a planar fashion from the mid-peripheral area of the retina (C), and an iatrogenic retinal break was formed at the 6 o'clock direction of the lower mid-peripheral area. After that, the inner limiting membrane stained with Brilliant Blue G was peeled off (D).

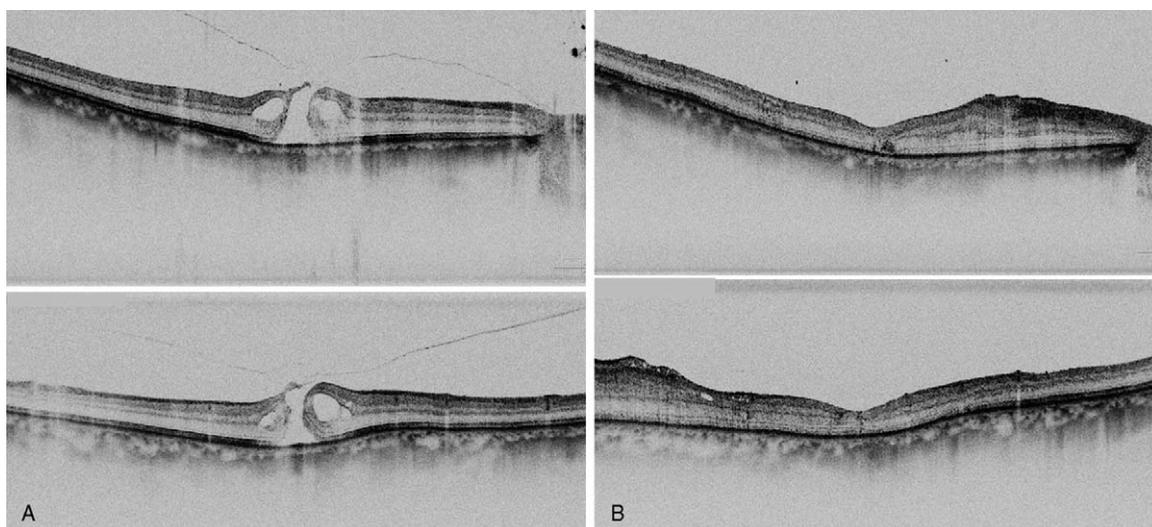**Figure 3.** OCT imaging of Case 2. Preoperative image showing perifoveal PVD (A). Vitreous surgery resulted in closure of the MH; however, the patient's corrected VA remained at 0.3 (B).

Moreover, perifoveal PVD had occurred around the MH (Fig. 3 A). The refraction was mild myopia, and his corrected VA was 0.2.

For treatment, we performed ultrasonic phacoemulsification and intraocular lens implantation, and the core vitreous gel was excised. Triamcinolone acetonide was applied to the macula and artificial PVD was created from around the MH to the periphery. Similar to Case 1, the vitreous gel was firmly adhered to the retina in a planar fashion from the mid-peripheral area, and we found that the boundary line of vitreoretinal adhesion had been raised in a "tent-like" shape at the time of creating the artificial PVD. Multiple iatrogenic retinal breaks were formed on the inferior, superior, and temporal sides, complicated with localized retinal detachment (RD). Further artificial PVD creation was difficult, and the peripheral area was limited to vitreous shaving. Next, removal of the inner limiting membrane around the MH, pneumatic retinal replacement, endophotocoagulation around the retinal break, and a 20% SF6 gas tamponade was performed. Postsurgery, RD reoccurred from the inferior side (Fig. 4 A), so surgical resection of the residual vitreous gel and a silicone oil tamponade was performed (Fig. 4 B). The silicone oil was then removed. Postsurgery, restoration of the retina was achieved and the MH became closed, yet the patient's corrected VA remained at 0.3 (Fig. 3 B).

3. Discussion

Although AH alone rarely causes a reduction of VA, diseases such as diabetic retinopathy, macular edema, and epiretinal membrane, etc., can result in a deterioration of fundu visibility, and sometimes make it undetectable. Even in cases in which fundus visibility is poor due to AH, the state of the macular area can be clearly visualized via OCT imaging, which is a very useful tool for diagnosis.[9] OCT is said to be affected by opacity of the optic media, but in cases of AH, clear tomographic images of the retina are relatively easy to obtain because light passes through the spaces between asteroid bodies. In the 2 cases in this present study, a MH was not detected during the preoperative fundus examination. However, the MH was clearly detectable via OCT imaging. Case 1 was a patient with a stage 4 MH. Case 2 was a patient with a stage 3 MH, which allowed for vitreous adhesion
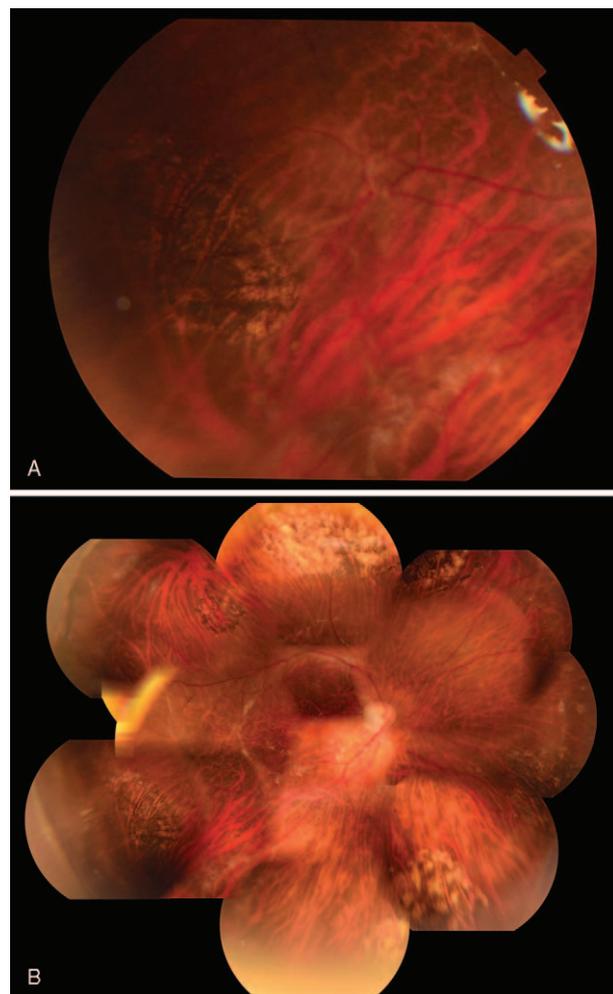

Figure 4. Fundus photographs of Case 2. After the initial surgery, retinal detachment reoccurred from the inferior side (A), and the patient underwent surgical resection of the residual vitreous gel and a silicone oil tamponade (B).

at the edge of the MH; however, perifoveal PVD had occurred around the MH. These findings were thought to be similar to common cases of an idiopathic MH. Even in the findings observed during vitreous surgery, the adhesion of the posterior vitreous membrane around the MH and the retina was relatively loose, and it was comparatively easy to make an artificial PVD for the posterior pole. Therefore, the pathogenesis of a MH in eyes with AHs was presumed to be nearly the same as that of a usual case of a patient with a MH. The difference from a normal MH case was that the vitreoretinal adhesion was very strong, starting at the mid-peripheral region. When artificial PVD was prepared by vitreous-cutter aspiration, we found that in both cases, the retina at the boundary site of vitreoretinal adhesion was linearly elevated around the entire circumference. In addition, the adhesions were stronger in the mid-peripheral region, and in our 2 cases, iatrogenic retinal breaks were formed due to excessive traction, and in Case 2, localized RD was given during surgery. The findings of some previous studies have indicated that iatrogenic retinal breaks can occur in the vicinity of the equator during vitrectomy surgery for an MH.[11,12] However, in our 2 cases, the iatrogenic retinal breaks were found to have formed at the posterior pole, not at the equator. In Case 2, as vitreous resection around the multiple iatrogenic retinal breaks was insufficient at the initial operation, RD recurred by retraction of the residual vitreous gel.

It is well known that AH usually occurs in eyes in which the posterior vitreous body is undetached. In a study by Topilow et al,[4] examination via slit-lamp microscopy and a frontal lens revealed that of 16 examined eyes, 10 had no PVD, 4 had partial PVD, and only 2 had complete PVD, and that the rate of PVD was clearly lower than that of patients of the same age. Wasano et al[3] reported that in 59 AH eyes that were examined, there was clearly less complete PVD and more partial PVD compared with the controls. On the contrary, Mochizuki et al[5,13] reported that vitreous gel remained on the retinal surface even in a case of seemingly appearing PVD at AH, and that "vitreous separation" (i.e., vitreoschisis) was observed. In the 2 cases in this present study, preoperative OCT imaging revealed perifoveal PVD, and we theorize that the partial PVD led to the onset of the MH. However, no PVD was generated around the macula, and in both cases, adhesion of the surface was continuous up to the periphery of the fundus. Similar findings to those in AH eyes are also seen in diseases other than that of a MH, especially in cases with proliferative diabetic retinopathy accompanied by AH, in which there are many cases where the vitreoretinal adhesion is extremely strong and the level of surgical difficulty is higher.[14] Although there are numerous unclear aspects as to why vitreoretinal adhesions are strengthened in eyes with AH, it has been reported that there is an increase of inflammation-related cells, such as macrophages, in the vitreous of AH[4] and that prolonged mild inflammation might accelerate the manifestation of the adhesion molecules at the vitreoretinal interface.

As to the macular diseases that can accompany AH, numerous studies have reported macular edema and epiretinal membrane, etc.[7–10] Although many of those macular diseases are reported to have often been caused by the primary disease, such as diabetic retinopathy, the study by Hwang et al[9] is the only previous report related to AH accompanied by MH. As in this present study, they reported a case of a stage 3 MH in which partial PVD had occurred in the posterior pole and in which partial PVD had also occurred in the optic disc head site. In addition, the findings at the time of vitreous surgery indicated that artificial PVD around the MH is relatively easy and that closure of the MH was obtained postsurgery, thus leading to the improvement of VA. However, Hwang et al[9] provided no description in their study as to the extent of vitreoretinal adhesion at the peripheral region.

On the basis of the above findings, we theorize that MH with AH is caused by perifoveal PVD as its pathogenic mechanism, in the same manner as in cases of normal MH. However, vitreoretinal adhesion starting at the mid-periphery was found to be very strong in our 2 cases, so we determined that artificial PVD should be limited to the area of loose adhesion.

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Author contributions

Conceptualization: Takakuni Kitagaki, Hiroyuki Suzuki, Masanori Fukumoto, Teruyo Kida, Tsunehiko Ikeda.
Data curation: Takakuni Kitagaki, Hiroyuki Suzuki, Ryohsuke Kohmoto, Shou Oosuka, Masanori Fukumoto, Takaki Sato, Takatoshi Kobayashi, Teruyo Kida, Tsunehiko Ikeda.
Formal analysis: Takakuni Kitagaki, Hiroyuki Suzuki, Ryohsuke Kohmoto, Shou Oosuka, Takaki Sato, Takatoshi Kobayashi, Teruyo Kida, Tsunehiko Ikeda.
Investigation: Takakuni Kitagaki, Hiroyuki Suzuki, Ryohsuke Kohmoto, Shou Oosuka, Takaki Sato, Teruyo Kida, Tsunehiko Ikeda.
Methodology: Takakuni Kitagaki, Hiroyuki Suzuki, Ryohsuke Kohmoto, Takatoshi Kobayashi, Teruyo Kida, Tsunehiko Ikeda.
Project administration: Takakuni Kitagaki, Hiroyuki Suzuki, Tsunehiko Ikeda.
Resources: Takakuni Kitagaki.
Supervision: Teruyo Kida, Tsunehiko Ikeda.
Visualization: Hiroyuki Suzuki.
Writing – original draft: Takakuni Kitagaki.
Writing – review & editing: Teruyo Kida, Tsunehiko Ikeda.

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