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Case report : Management of Macular Hole
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Management of Macular Hole

Abstract

***Introduction** : Macular hole is a defect in the center of the macular area of the retina. A macular hole causes loss of sharp "straight-ahead" vision and reading vision. In early stages of macular hole formation, the hole is very small and the central vision may be only sligthly blurred and distorted. As the hole enlarges, the vision becomes progressively worse. Its diagnosis is made by recognition of clinical features and diagnostic test by using the slit lamp with a handheld or fixed lens, used in either a contact or non contact fashion, the slit-beam test (Watzke-Allen sign), Fluorescein angiography, and optical coherence tomography (OCT). There are two options for macular hole therapy, either observation or surgery, depend on the stage of macular hole.*

***Objective** : To report case series of macular hole and their management.*

***Case report** : There are two cases with macular hole. Both of the case is diagnosed as macular hole. Both the patients had the same complaints, such as blurred on one side of vision. By Amsler grid examination we found metamorphopsia, and from posterior segment examination it was found macular hole. OCT examination was performed for both patient, where the first patient it was found full thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posterior hyaloid face. At the second patient we found the full thickness hole with separation of the vitreous from the macula and non present operculum on the posterior hyaloid face. Both of the patients was performed pars plana vitrectomy, a peeling of the internal limiting membrane and SF6 gas tamponade. For the first patient there was improvement of visus and by OCT re-examination we found the macula was getting back to normal. While the second patient there was no improvement of visus, and by OCT re-examination, we found macular hole still persistent.*

***Conclusion:** Pars plana vitrectomy, peeling of the internal limiting membrane and SF6 gas tamponade is carried out for macular hole stage II, III and IV, aim to close the hole, because the hole can lead to a detached retina, a sight-threatening condition. The operation may failure because the size of the hole which cause it was difficult to enclose. It may probably because the patient did not comply to maintain postoperative face down positioning.*

I. Introduction

The macula is at the back of the eye, situated in the middle of the retina. It is a light sensitive layer which converts light into signals that tell the brain what we are looking at. The macula enables us to see sharp, clear images. A hole is caused when the vitreous, the gel-like substance in the eye, shrinks and starts to come away from the retina. During this process, a hole in the macula can result. This is known as a macular hole. [1]

Full-thickness macular holes are defects involving all layers of the retina rom the internal limiting membran through the outer segment of retinal photoreceptors. Lamellar holes involve

only a portion of the retinal layers. The vast majority of macular holes are idiopathic, and are predominantly seen in emmetropic patients in the sixth or seventh decades of life, more often in women at ratio of 2-3 1. The prevalence of idiopathic macular holes has been estimated to be 3.3 per 1000. Other causes of maculare holes include trauma, high myopia,cystoid macular edema, inflammation, solar retinopathy, vitreomacular traction syndrome, traction from epiretinal membranes, and degenerative conditions of the retina. Idiopathic macular holes are bilateral in about 16% of patients. [2-5]

II. Case Report

Case 1

A 73-years old man came to Cicendo Eye Hospital (CEH) on April 15th 2011 with chief complaint of decreased vision on his right eye since two months ago. No history of trauma, red eyes, no history wearing spectacle or previous surgery found. History of hypertension and diabetes mellitus was denied. General examination was within normal limit. Ophthalmological examination revealed the uncorrected visual acuity (UCVA) of right and left eyes were 0.2 f2 Ph fixed and 0,3ph 0.4 . Ocular motility was full to all direction. Intraocular pressure (IOP) using Schiotz tonometer were 17.3 mmHg for both eyes. Anterior segment examination of both eyes were within normal limits except for cataract on both eyes.

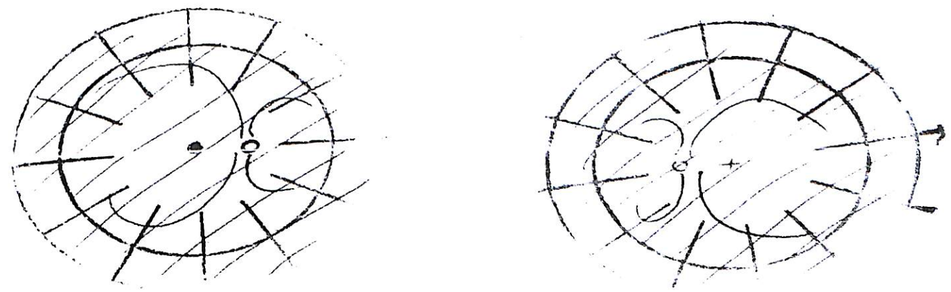

Figure 1. Fundus drawing April 15th 2011

The posterior segment of the right eye (RE) revealed clear media, round and sharp border of optic disc, cup/disc ratio was 0.3, artery/vein ratio was 2/3,tigroid and flat retina, and on macula is found macular hole, while the left eye (LE) revealed normal limits. Patient was

advised to have ocular coherence tomography (OCT). OCT of the right eye showed full thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posterior hyaloid face (figure 2). The patient was diagnosed with macular holestage 3 on RE, and imatur senile cataract on both eyes. The planed management pars planavitrectomy, peeling of the internal limiting membrane and SF6 gastamponade under general anesthesi.

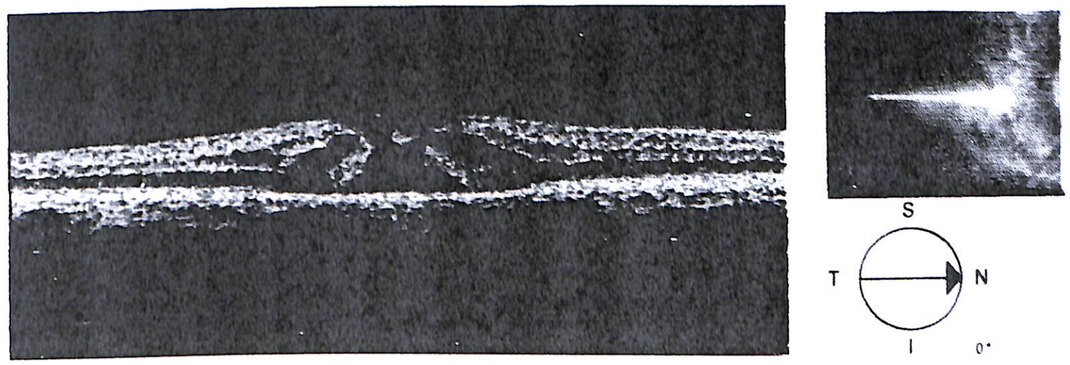

Figure 2. OCT of the right eye : full thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posterior hyaloid face.

On April 27th 2011, the patient was performed pars plana vitrectomy and peeling of the internal limiting membrane by implementation of colorization at internal limiting mebrane using methylen blue before. After that, it was performed fluid gas exchange and at the last using SF6 gas astamponade. Antibiotic cream was applied to the eye before it closed. The therapy were Ofloxacineyedrop 6 times a day, steroid eye drop 6 times a day, cyclon eye drop 3 times a day, Cyprofloxacin 500 mg 2 times a day, and Paracetamol 500 mg 3 times a day. The patients maintaned face-down positioning post operatively for 10 days.

One day post operatively the visual acuity of the right eye was hand movement. Digital IOP was normal, bulbar conjunctival hyperemis, cornea relatify clear, coa moderate, cloudy lens. Funduscopic examination revealed slighly cloudy media, round and sharp border of optic disc, cup/disc ratio and artery/vein ratio were physiologist, tigroid and flat retina and present SF6, decreased foveal reflex. The diagnosed was macular hole on RE (post pars plana vitrectomy, peeling of the internal limiting membrane and SF6), and imatur senile cataract on both eyes.

One week post operatively patients returned to control, visual acuity of the right eye were 4/60. Funduscopic examination revealed slighly cloudy media, round and sharp border of optic disc, cup/disc ratio and artery/ vein ratio were physiologyst, tigroid and flat retina and present SF6, and on macula is found macular hole, decreased foveal reflex.

One week later (two weeks post operatively), visual acuity of the right eye and left eye were 3/60 and 0.5 LogMAR respectively. Funduscopic examination revealed clear media, round and sharp border of optic disc, cup/disc ratio was 0.3, artery/vein ratio was 2/3, tigroid and flat retina, on macula was found edema and the hole was getting better. Then we took the OCT re-examination to the patient, and then it was found that the macula is getting back to normal.

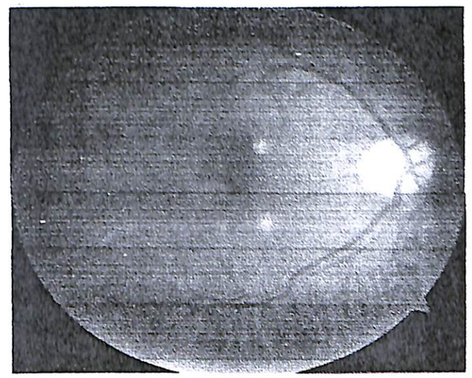

Figure 3. Fundus photograph of the RE

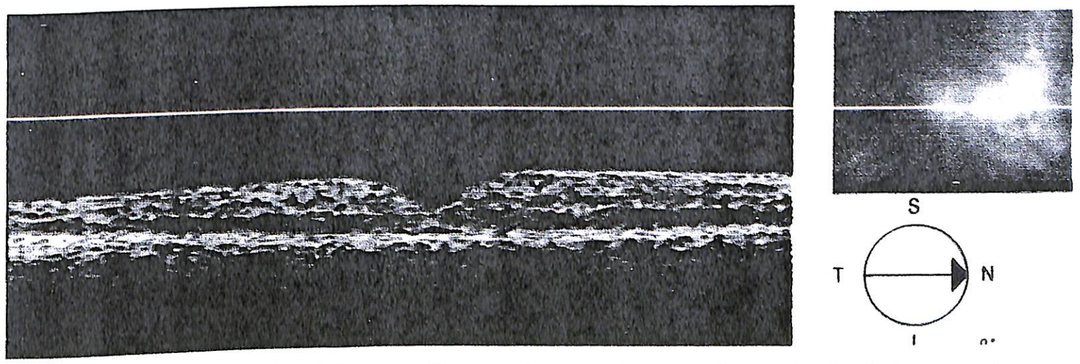

Figure 4. OCT of the right eye :after surgical repair : macula getting back to normal

Two weeks later (four weeks post operatively) patients returned to control, visuall acuity of the right eye and left eye were 0,9 LogMAR and 0.5 LogMAR respectively. Funduscopic examination revealed clear media, round and sharp border of optic disc, cup/disc ratio was 0.3, artery/vein ratio was 2/3, tigroid and flat retina, on macula was found no edema and the hole was getting better Amsler grid test revealed metamorphopsia, with no evidence of scotoma. Then the patient was diagnosed as resolving macular hole on the right eye. Patient should visit for control to hospital after two weeks.

Case 2

A 41-years old woman came to Cicendo Eye Hospital (CEH) on November 26th 2009 with chief complaint of decreased vision on his left eye since two months ago with swing vision sensation, and often headache. No history of trauma, red eyes, no history wearing spectacle or previous surgery found. History of hypertension and diabetes mellitus was denied. General examination was within normal limit. Ophthalmological examination revealed the uncorrected visual acuity (UCVA) of right and left eyes were 0.2 LogMAR and 1.0 LogMAR. Ocular motility was full to all direction. Intraocular pressure (IOP) using Schiotz tonometer were 17.3 mmHg for both eyes. Anterior segment examination of both eyes were within normal limits.

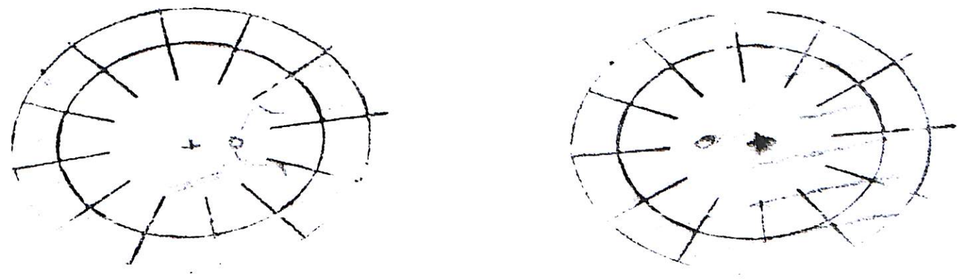

Figure 5. Fundus drawing November 26th 2009

The posterior segment of the right eye (RE) was within normal limits, and there was macular edema and decreased foveal reflex in left eye. Amsler grid examination revealed metamorphopsia in the left eye with no scotoma. Patient was advised to have ocular coherence tomography (OCT). OCT of the left eye showed full thickness hole with separation of the vitreous from the macula and not present operculum on the posterior hyaloid face. The patient

was diagnosed with macular hole stage 3 on LE. The planed management pars plana vitrectomy, peeling of the internal limiting membrane and SF6 gas tamponade.That time, the patient was not ready to be operated.

Patient came back for follow up visit on Januari 5st 2010 and ready ready to be operated. At the next day, the surgery performed. Pars plana vitrectomy, peeling of the internal limiting membrane, fluid gas exchange and SF6 gas tamponade was done under general anesthesia. Antibiotic cream was applied to the eye before it closed. The therapy were Tobramisin eye drop 6 times a day, steroid eye drop 6 times a day, cyclon eye drop 3 times a day, Ciprofloxacin 500 mg 2 times a day, and Paracetamol 500 mg 3 times a day. The patients maintaned face-down positioning post operatively for 10 days.

One day postoperatively the visual acuity of the left eye was hand movement. Digital IOP was normal, bulbar subconjunctival bleeding, and other anterior segment within normal limits. Funduscopic examination revealed slightly cloudy media, appeared hemorrhage in inferior vitreous, papil appeared round shadowy, flat retina and present SF6, foveal reflex difficult to assess. The diagnosis was macular hole on LE (post pars plana vitrectomy, peeling of the internal limiting membrane and SF6).

One week post operatively patients returned to control, visual acuity of the left eye were 3/60. Funduscopic examination revealed sligthly cloudy media, appeared still hemorrhage in inferior vitreous, round and sharp border of optic disc, cup/disc ratio and artery/ vein ratio were physiologyst, flat retina and present SF6, decreased foveal reflex and macular hole.

One week later (two weeks post operatively), patients returned to control, visual acuity of the right eye and left eye were 0.1 LogMAR and 1/60 respectively. The result of funduscopy examination still same with previous.

Two weeks later (four weeks post operatively) patients came back to visit, visual acuity of the right eye and left eye were 0.1LogMAR and 2/60 respectively. The result of funduscopy examination still the same with previous, except the media appeared clearly. Then the patien was suggested to be performed second OCT and the result was the macular hole in persistent condition.

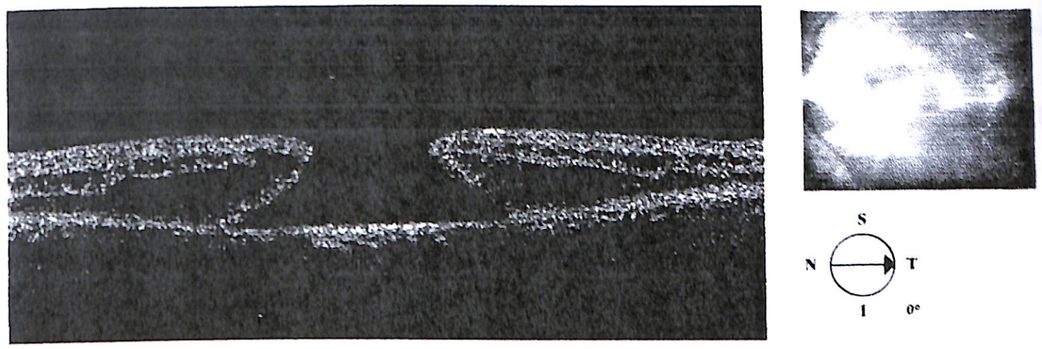

Figure 6. OCT of the left eye after surgical repair still revealed full thickness hole with separation of the vitreous from the macula and not present operculum on the posterior hyaloid face.

III. Discussion

A macular hole is a defect in the center of the macular area of the retina. The macula gives us sharp central vision and reading vision. The very center portion of the macula, called the fovea, is the thinnest portion of the entire retina. It is in this very delicate foveal area that a macular hole can develop.[3-5]

In most cases, a macular hole develops as a result of anatomical changes that occur spontaneously and not from anything that the patient has done. This type of macular hole occurs most commonly in individuals over 50 years of age and is called an idiopathic macular hole. Occasionally, severe blunt trauma can cause a macular hole. A macular hole can also be seen in a very small percentage of people with retinal detachment, or in conditions that cause severe edema (swelling) of the retina.[3-5]

A macular hole causes loss of sharp "straight-ahead" vision and reading vision. In the early stages of macular hole formation, the hole is very small and the central vision may be only slightly blurred or distorted. As the hole enlarges, the vision becomes progressively worse. The hole typically enlarges to a point at which the affected eye can only see the larger letters of an eye chart. A macular hole does not cause complete blindness and does not affect the peripheral (side) vision.[3-5]

The hallmark complaint of idiopathic macular hole formation is painless central visual distortion or blur of an acute or subacute nature. When only one eye is involved, it is not unusual for the visual loss to go undetected unless cross-covering is performed. Central visual acuity

initially may be diminished only mildly; however, as the hole progresses over weeks to months, the acuity usually deteriorates, then stabilizes around the 20/200-20/800 (6/60-6/240) level.[3-5]

In these cases, The first patient is man, 73 years old, while the second patient is female, 41 years old. Theory tells that macular hole most commonly in individual as over 50 years of age, but it is not proofed in this case series. Both patient was complained of blurred vision, without existing of pain, whereas the first patient VA was 3/60, and the second patient VA was 1.0 LogMAR. From Amsler grid examination for both patient we found metamorphopsia and from funduscopy examination there was macular hole at the macula.

A currently accepted system of stages based on biomicroscopic observations was reported initially by Gass in 1988 and then revised in 1995; it explains the clinically observed appearances of macular holes and their precursor lesions. The hallmark inciting event of idiopathic macular hole formation is hypothesized to be focal shrinkage of the vitreous cortex in the foveal area. Clinically, four stages occur in idiopathic macular hole development. Although Gass' biomicroscopic interpretations of the various stages are accepted widely, OCT studies imply that the retinal changes that occur in stage 1 holes differ slightly from the accepted classification.[3-5]

In idiopathic macular hole, impaired visual function is multifactorial. Photoreceptor loss from the central foveal area may occur in some cases, although a true retinal operculum rarely is evident. The central scotoma that results from foveal dehiscence is made significantly larger by the surrounding localized retinal detachment. Cystic changes develop in the intact perifoveal retina, as well, and some eyes develop epiretinal membranes. All the factors combine to decrease the central vision. Rhegmatogenous retinal detachment beyond the macula occurs secondary to a macular hole only if abnormal vitreous traction or high myopia with staphyloma is present concurrently.[3-6]

The following descripton of the stages of macular hole formation and what OCT reveals at each stage is useful in interpreting biomicroscopic findings and making management decisions :

- A stage 0 or premacularhole state occurs when patients develop a perivofeal vitreous detachment, and only subtle changes in macular topography, such as loss of the foveal depressions, can be seen. Patients usually have a normal acuity and most do not develop advanced stage of macular holes.

- A stage 1 or impending macular holes have visual symptomps that typically include central vision loss (with visual acuity typically measuring 20/25 to 20/60) and metamorphopsia. On biomicroscopy, there is loss of the foveal depression associated with a small yellow spot (stage 1A) or yellow ring (stage 1B) in the center of fovea. OCT examination reveals that a stage 1A hole is a foveal" pseudocyst", or horizontal splitting associatedwith a vitreous detachment from the perifoveal retina but not from the foveal center.in the stage 1B holes, therre is progression of the pseudocyst posteriorly to include a break in the outer foveal layer, the margins of which constitute the yellow ring seen clinically. As many as 50% of stage 1 holes resolve spontaneously following separation of the vitreoretinal adhesion and spontaneous relief of tractional forces.
- A stage 2 macular hole represents the progression of the fovealpseudocyst to a full thickness dehiscence, as a tractional break develops in the "roof" (inner layer) of the pseudocyst.The small opening in the inner layer (< 400 µm diameter) may be either centrally or eccentrically located. Progression to stage 2 typically occurs over several weeks or months and usually involves a further decline in visual acuity. OCT demonstrates that the posterior hyaloid typically remains attached to thefoveal center in the stage 2 holes.
- A stage 3 macular hole is afully developed hole (≥ 400 µm diameter), typically accompanied by a rim of thickened and slightly elevated retina. Visual acuity may range from 20/40 to 5/200, but generally around 20/200. The posterior hyaloid remains attached to the optic disc, but is detached from the macular region. An operculum may or may not be present, suspended on the posterior hyaloid overlying the hole.
- A stage 4 macular hole is a fully developed hole with a complete posterior vitreous detachment signified by a Weiss ring.[3,4,5,7]

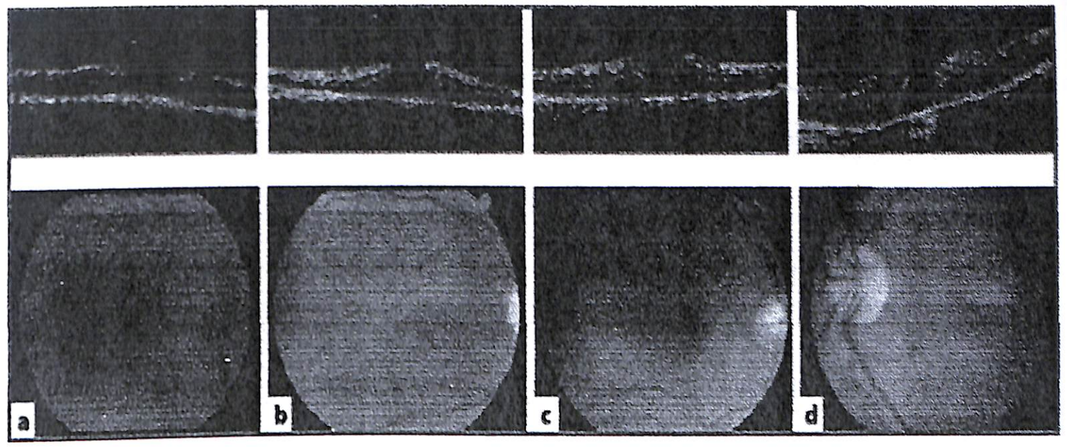

Figure 7. Fundus photographs and OCT images of stage 1–4 macular holes. **a.** Stage 1b lesion with a partial-thickness foveal defect. **b.** Stage 2 full-thickness hole associated with persistent vitreo-foveal attachment. **c.** Stage 3 full thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posteriorhyaloid face. **d.** Stage 4 full-thickness hole with complete posterior vitreous separation from the macula and optic disc.

Source :Kirchhof B, Vitreoretinal surgery. Essentials in ophthalmology (e-book).[5]

The diagnosis of idiopathic macular hole is a clinical one, made at the slit lamp with a handheld or fixed lens, used in either a contact or noncontact fashion. At times, the diagnosis can be difficult, especially when a unilateral stage 1 or stage 2 hole is present.[3]

Ancillary testing can be helpful in certain cases. Fluorescein angiography is not generally of benefit, although it can help to rule out other entities that mimic macular hole. In stage 1 macular holes, fluorescein angiography commonly is normal or reveals only a small window defect. Some stage 2 macular holes show an intense, small, central window defect, while others manifest normal angiography or a mild window defect. In a stage 3 or 4 macular hole, the window defect tends to be mild and corresponds in size and location to the retinal defect.[3,4,6,7]

The slit-beam test (Watzke-Allen sign) usually is reliable to test subjectively for a full-thickness retinal defect. In this test, a thin, vertically oriented slit beam is focused on the macula and the patient is asked to describe the line of light. In a full-thickness defect, a break or thinning

of the beam is seen centrally.The scotoma associated with a macular hole can be mapped using the scanning laser ophthalmoscope or with the aiming beam of a laser.[3,4,6,7]

Imaging tests of the retina and vitreous, such as ultrasonography, optical coherence tomography, and scanning laser biomicroscopy, are used to confirm the diagnosis and to assess the attachment of the vitreous to the fovea. OCT is the most clinically useful ancillary test.[3-7]

To confirm the diagnosis for both patient, and to assess the attachment of the vitreous to the fovea, it was performed additional examination by using OCT for imaging test of the retina and vitreous. From OCT examination, both patient was diagnosed as macular hole stage 3. For the first patient we foundfull thickness hole with separation of the vitreous from the macula and a fully detached operculum on the posterior hyaloid face. While the second patient we foundfull thickness hole with separation of the vitreous from the macula and non present operculum on the posterior hyaloid face.

Prior to 1989, idiopathic macular holes were considered untreatable. Kelly and Wendel were the first to report that vitreous surgery can improve the visual acuity in some eyes with acute, idiopathic macular holes. Since then, vitrectomy for idiopathic macular holes rapidly has become a widely performed procedure throughout the world.[3]

Vitrectomy sugery is indicated for full-thickness macular holes (stage 2,3 and 4). Because stage 1 macular holes have a high rate of spontaneous resolution and reported studies have failed to demonstrate a benefit from vitrectomy, surgery is not recommended for this earliest stage. If a stage 1 hole has significantly decreased visual acuity that persists for months, surgery should be considered. Macular hole surgery typically is performed under local anesthesia, unless the patient requests general anesthesia. Early intervention in the other-stage holes is considered an important prognostic factor, however, leading to improved functional and anatomical outcomes. Surgery for full-thickness macular holes consists of a standard pars planavitrectomy, removal of the posterior cortical vitreous, a variable degree of preretinal tissue dissection, and use of an intraocular gas or, less frequently, silicone oil with face-down positioning.A standard three-port core vitrectomy is completed using either 20-gauge, 23-gauge, or 25-gauge incisions. Following this, the intact posterior cortical vitreous is engaged, typically using the vitrectomy instrument with the cutting function disabled. Alternatively, an extrusion needle, a bent needle, or a pick can be used to engage the cortical vitreous. The posterior cortical vitreous is usually invisible until it is elevated off the retina. Elevation of the cortical vitreous and posterior hyaloid is carried out

until complete separation of the vitreous is achieved. The remainder of the posterior vitreous is removed with the vitrectomy cutting instrument. At this juncture, epiretinal membrane, if present, is peeled using a bent, sharp needle or fine (ILM) forceps. If internal limiting membrane (ILM) peeling is felt to be beneficial, it is performed at this stage. Internal limiting membrane (ILM) peeling has been demostrated in numerous studies to improve the rate of hole closure, particularly for large stage 3 or 4 holes. The internal limiting membrane can be stained with indocyanine green (ICG) dye, triamcinolone, or trypan blue to make its surgical removal technically simpler. An air-fluid exchange follows and air, dilute sulfur hexafluoride (SF_6) gas, or dilute perfluoropropane (C_3F_8) gas is placed in the vitreous cavity prior to closure of the sclerotomy sites. The surgery can be performed safely in an outpatient setting. Patients usually are instructed to maintain strict face-down positioning for at least 7 days postoperatively, although intervals from 3 days to 3 weeks have been suggested. [3-12]

Peeling of the internal limiting membrane (ILM) from the macula is a technique advocated in an attempt to further improve anatomical and visual outcomes of surgery for macular holes.The rationale for peeling the ILM is to relieve tangential traction from the edges of the hole and to promoteclosure by stimulation of wound healing. Peeling the ILM ensures thorough removal of anytangential tractional components implicated inthe development of macular holes. The removalof a potential scaffold for re-proliferation of myofibroblasts may reduce the possibility of late reopeningof surgically closed holes. Furthermore, peeling of the ILM is also believed to stimulatewound healing at the macula, possibly by inducing local expression of undefined growth factorsthat promote glial repair.[5,6]

Inner limiting membrane peeling appears to improve the rate of anatomical closure, but itseffect on visual outcome is less predictable andexcessive unsuccessful attempts to peel the ILM are associated with poor visual outcome. WhileILM peelingmay be performed for full-thickness macular holes of any stage, it is more commonly reserved for stage 3 or 4 holes, long-standing holes, those that have failed to close, or those that have re-opened following conventional surgery.[5,6]

As in any invasive surgical procedure, intraoperative and post operative complications can occur; some are unique to macular hole surgery, while others may develop during any vitreoretinal procedure. The most common complication after the surgery is cataract formation. It has long been known that vitrectomy in a phakic eye leads to accelerated nuclear sclerosis; the

addition of intravitreal gas probably speeds the process. Over 50% of phakic eyes that undergo vitrectomy for a macular hole suffer significant nuclear sclerosis during the subsequent 2-year follow-up. In successful cases, eventual removal of the cataract by standard techniques can be performed without undue risk. Some surgeons advocate removal of clear lenses or minimal cataracts at the time of macular hole surgery to eliminate the need for a second procedure.[3-12]

Retinal tears or retinal detachment or both may be seen in up to 10% of eyes that undergo vitreous surgery for an idiopathic macular hole. This incidence is relatively high, because an intraoperative and, therefore, traumatic creation of a posterior vitreous detachment is a critical step in the procedure. Intraoperative recognition of retinal tears with prompt treatment using either laser or cryotherapy can keep the occurrence of retinal detachment low. Some retinal tears develop during the immediate post operative period, so vigilant post operative fundus observation is necessary. Most retinal detachments can be repaired with standard scleral buckling, vitrectomy, or pneumatic retinopexy techniques.[3-12]

Other, less common complications include intraoperative light or mechanical toxicity to the macular retinal pigment epithelium, intraoperative enlargement of the macular hole, and late reopening of successfully closed holes. Late reopening occurs in 5% of once successfully treated holes and can occur years after the initial surgery. Second operations to close the reopened holes may be successful in some cases. Complete peeling of the ILM may decrease the rate of late reopening. Some investigators have observed dense, temporal visual field defects in eyes that have undergone vitrectomy for macular holes. Intraoperative damage to the nerve fiber layer by either excessive drying secondary to air infusion and/or toxicity of ICG have been implicated.[3-12]

Without surgery, macular holes tend to stabilize with a visual acuity of 20/200-20/800 (6/60-6/240) and a diameter of about 500 μm. With surgery, visual improvement is possible. Anatomical success can be determined 2–4 weeks after surgery, when the gas bubble has resorbed enough to be no longer in contact with the macular hole in the upright position. Anatomical success usually is defined clinically as complete disappearance of the cuff of subretinal fluid that surrounds the macular hole. It is defined on OCT as the reapproximation of retinal tissue covering the full-thickness defect. In most instances, when this occurs the edges of the macular hole are opposed firmly to the retinal pigment epithelium, which renders identification of the macular hole difficult. Visual success is defined as an improvement in postoperative visual acuity of at least two or more Snellen lines over preoperative acuity.[3-12]

Pars plana vitrectomy, internal limiting membrane (ILM) peeling, fluid gas exchange and SF6 gas tamponade was performed to both patient because from OCT examination we found a macular hole stage 3, therefore it was indicated for, aimed to close the hole, because if stage III macular hole develops, most central and detailed vision can be lost. If left untreated, a macular hole can lead to a detached retina, a sight-threatening condition. For the first patient, fourweeks after pars plana vitrectomy, internal limiting membrane (ILM) peeling, fluid gas exchange and SF6 gas tamponade operation, we found improving vision and by OCT re-examination we found the macula was getting back to normal. Whereas the second patient after pars plana vitrectomy, internal limiting membrane (ILM) peeling, fluid gas exchange and SF6 gas tamponade operation was performed, resulted in still deteriorating vision. After the second OCT was performed, we found macular hole still persistent because the macula was not attach after operation performed. Persistent macular hole in the patient number two probably because the size of the hole which cause it was difficult to enclose. It may probably because the patient did not comply to maintain postoperative face down positioning. Postoperative face down positioning, to maintain gas-bubble tamponade of the hole, was thought to be vital to achieving successful closure.

The vital prognosis of these cases are good. The functional prognosis is dubia ad bonam for patient number one and ad malam for patient number two because there was no improvement of visus after the treatment and the persistent of macular hole.

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