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Ayra Khan, BS  
*Thomas Jefferson University*

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# Macular Holes: Diagnosis, Treatment, and Complications

By Ayra Khan, BS | Faculty Reviewer: Jason Hsu, MD

**M**acular hole is a condition that

affects the central visual field of the eye. Without prompt diagnosis and intervention, macular holes can progressively worsen, significantly impacting both vision and overall quality of life.

resulting in a blind spot over the central visual field.

The most common cause of MH is vitreous traction. With age, a gel-like fluid that fills the eye known as the vitreous retracts and detaches from the retina.

Typically, this does not cause problems. However, vitreous separation with pathologic vitreomacular adhesion can

exert force, disrupting retinal layers and leading to development of MH. Other causes of MH include trauma and a high degree of myopia. MH may also

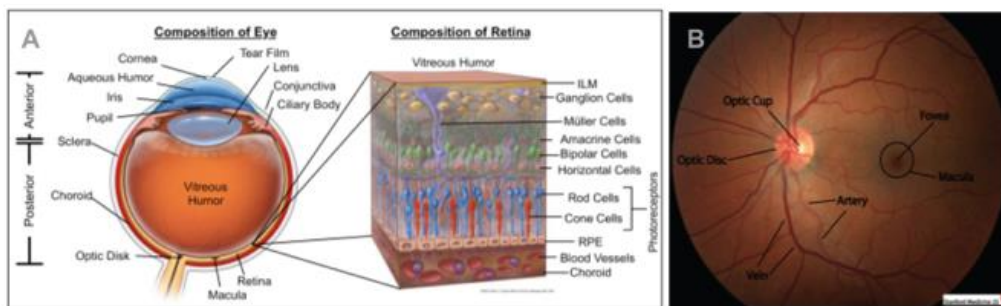


Figure 1. A) Location of the macula and layers of the retina in a schematic. B) Labeled fundoscopic photo illustrating the position of the macula in relation to other structures in the eye.<sup>3,4</sup>

## Diagnosis

A macular hole (MH) is a defect of the neurosensory retinal layer in the macula.<sup>1</sup> This defect can manifest as either a partial, lamellar macular hole (LMH) or a full-thickness macular hole (FTMH). The macula is a region of the retina responsible for central, fine-detail, and color vision. It contains the fovea, a depression in the retina where vision is the sharpest (*Figure 1*).<sup>2</sup> Untreated MH can lead to progressive deterioration of central vision, potentially

be associated with other ocular conditions such as diabetic retinopathy, hypertensive retinopathy, and epiretinal membrane.<sup>1</sup>

Diagnosis of MH involves thorough analysis of history and physical exam findings. Early symptoms include metamorphopsia (distorted, wavy vision) but with time, progression to a central scotoma, or blind spot, may occur. Important history components include duration of symptoms, ocular history, associated conditions, and medications that may cause cystoid macular edema.<sup>5</sup> An Amsler grid test can determine the presence of metamorphopsia. Examination

often includes slit-lamp biomicroscopy, indirect retinal peripheral examination, and optical coherence tomography (OCT) to visualize damage to the macula (Figure 2).<sup>5</sup>

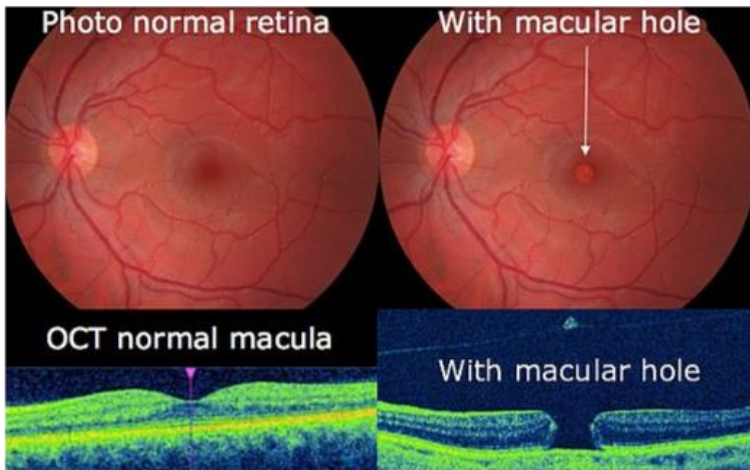


Figure 2. Funduscopy photo of normal retina versus retina with macular hole and correlation to OCT findings.<sup>6</sup>

Traditionally, staging has been used to assess prognosis and treatment options. One of the most widely used staging systems is Gass’s 1995 classification based on biomicroscopy. However, updates have since been proposed to this classification with the advent of OCT (Table 1). The International Vitreomacular Traction Study Group (IVTS) is one such group that categorized MH based on OCT findings. The main categories as proposed by the IVTS are vitreomacular adhesion (VMA), vitreomacular traction (VMT), FTMH, LMH, and macular pseudohole.

Gass 1995 Staging and Exam Findings

Stages	Biomicroscopic Findings
1-A	<ul style="list-style-type: none"> <li>• Central yellow spot</li> <li>• Loss of foveolar depression</li> <li>• No vitreofoveolar separation</li> </ul>
1-B	<ul style="list-style-type: none"> <li>• Yellow ring, bridging interface</li> <li>• No foveolar depression</li> <li>• Lack of vitreofoveolar separation</li> </ul>
2	<ul style="list-style-type: none"> <li>• Oval, horseshoe, or crescent defect in yellow ring</li> <li>• Central, round retinal defect with elevated retinal rim</li> <li>• Prefoveolar opacity</li> <li>• No prefoveolar opacity</li> </ul>
3	<ul style="list-style-type: none"> <li>• Central, round retinal defect greater than/equal to 400 microns</li> <li>• Elevated retinal rim, no Weiss’s ring</li> <li>• Prefoveolar opacity</li> <li>• No prefoveolar opacity</li> </ul>
4	<ul style="list-style-type: none"> <li>• Central, round retinal defect</li> <li>• Elevated retinal rim</li> <li>• Weiss’s ring</li> <li>• Prefoveolar opacity</li> <li>• No prefoveolar opacity</li> </ul>

Table 1. Gass 1995 classification of stages based on biomicroscopic findings.<sup>7</sup>

Within the FTMH category, there are further subdivisions based on cause, presence/absence of VMT, and size (Figure 3).<sup>8</sup> Notably impacting clinical management is the size categorization, influencing surgical approaches and the potential for spontaneous closure (Table 2).

Macular Hole Classification by Size

Size	Definition
Small	Less than or equal to 250 microns
Medium	250 - 400 microns
Large	Greater than or equal to 400 microns

Table 2. Size classification based on IVTS-proposed categorization.<sup>8</sup>

Full-Thickness Macular Hole Stages in Common Use	International Vitreomacular Traction Study Classification System
Stage 0	VMA
Stage 1: impending macular hole	VMT
Stage 2: small hole	Small or medium FTMH with VMT
Stage 3: large hole	Medium or large FTMH with VMT
Stage 4: FTMH with PVD	Small, medium, or large FTMH without VMT

FTMH= full-thickness macular hole; PVD= posterior vitreous detachment; VMA= vitreomacular adhesion; VMT= vitreomacular traction.

Figure 3: IVTS updated classification based on OCT findings and correlation to commonly used Gass staging.<sup>8</sup>

### Treatment

Smaller or earlier stage MH have a higher likelihood of spontaneous closure, making observation a reasonable option in select cases.<sup>9</sup> However, if symptoms worsen or the MH progresses, treatment is necessary to prevent vision loss.

### Ocriplasmin

Ocriplasmin is a pharmacological approach that promotes detachment of the vitreous from the macula through injection of a truncated human plasmin protein. Specifically, the proteolytic activity of this plasmin variant cleaves the protein matrix that is responsible for adhering the vitreous to the macula.<sup>10</sup> Ocriplasmin may be beneficial in small holes or earlier stages of MH when focal vitreomacular traction is present. Further MH progression and lack of closure may indicate the necessity of surgical intervention. Adverse effects attributed to ocriplasmin include photopsia and blurred vision.<sup>10</sup> Multiple cases of vision loss have been reported after ocriplasmin use, often associated with

electroretinogram abnormalities believed to be related to proteolytic activity impacting the photoreceptors. Thus, this approach has largely fallen out of favor.<sup>11,12</sup>

### Pars Plana Vitrectomy

Pars plana (posterior) vitrectomy remains the standard procedure for MH intervention. First, micro-incisions with insertion of self-retaining trans-scleral cannulas are made in the pars plana region, which is anterior to the retina and posterior to the highly vascularized ciliary body. Next, a vitrectomy probe is placed through the cannulas, and vitreous is removed to allow better access to the back of the eye (*Figure 4*).<sup>13</sup>

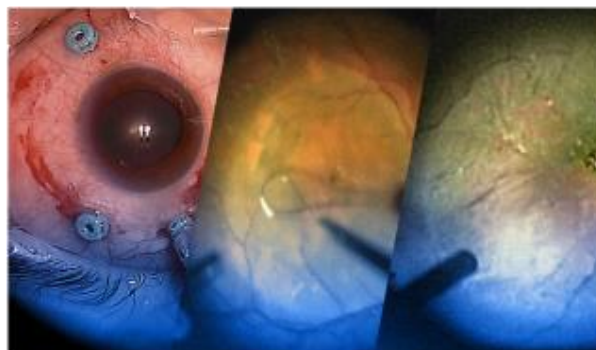


Figure 4. Insertion of vitrectomy probe into the cannulas during pars plana vitrectomy<sup>17</sup>

Often, a complete posterior vitreous detachment is not present and is surgically induced using the vitrectomy probe. After this step, internal limiting membrane (ILM) peeling may be performed (*Figure 5*). ILM peeling ensures complete release of any vitreous or tangential traction on the retina and has been shown to improve closure

rates. In cases of larger holes, a technique called inverted ILM peeling may be considered to improve closure rates. Instead of completely removing the ILM around the hole, the ILM is peeled to the edge of the hole and folded over it, thereby covering the hole with the ILM.<sup>10, 14, 15</sup>



Figure 5. Peeling of stained ILM during vitrectomy.<sup>18</sup>

After peeling, the fluid is removed from the vitreous cavity while air is infused into the eye. A tamponade agent of gas or, rarely, oil is injected into the vitreous cavity. The surface tension created by the tamponade agent prevents influx of fluid through the macular hole and may promote migration of cells into the hole, thereby helping with the healing process.<sup>13,16</sup> This process has high success rates and contributes significantly to improvements in vision and quality of life.<sup>15</sup>

Postoperative care is a crucial step of the treatment process. In the past, strict face down positioning after surgery was highly recommended, however some

physicians believe that this may not be necessary (*Figure 6*).<sup>14, 19</sup>

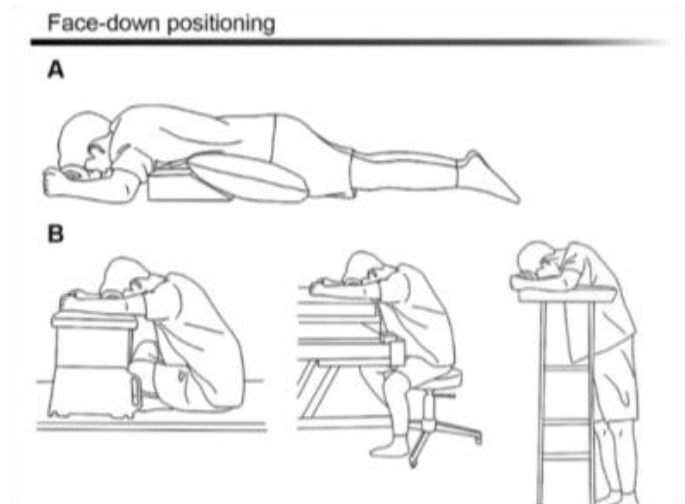


Figure 6. Illustration of recommended face down posturing post-vitrectomy.<sup>17</sup>

Studies indicate that face down positioning may still be helpful in cases with inadequate filling of gas for tamponade or with large MH.<sup>19</sup> Follow-up appointments are necessary to evaluate the healing process. This usually consists of an appointment 1-2 days after surgery, followed by another 1-2 weeks after surgery.<sup>5</sup> Patients are usually prescribed antibiotic and steroid eye drops. A patch and shield will be placed over the eye, and the patient will be instructed to avoid heavy lifting, flying (if an air or gas bubble is present), and particles entering the eye. A post-vitrectomy status bracelet may be provided for other physicians' awareness as use of certain anesthetic gasses, especially nitrous oxide, is contraindicated in the presence of an air or gas bubble. Further appointments and frequency depend on the



nature of the healing, outcome of surgery, and patient symptoms. In these appointments, the physician assesses visual acuity, the development of new symptoms, and postoperative anatomy as needed.<sup>5</sup> Typically, visual improvement manifests within 6 - 8 weeks, but it will often take months to see what the final visual outcome will be (*Figure 7*).<sup>21</sup>

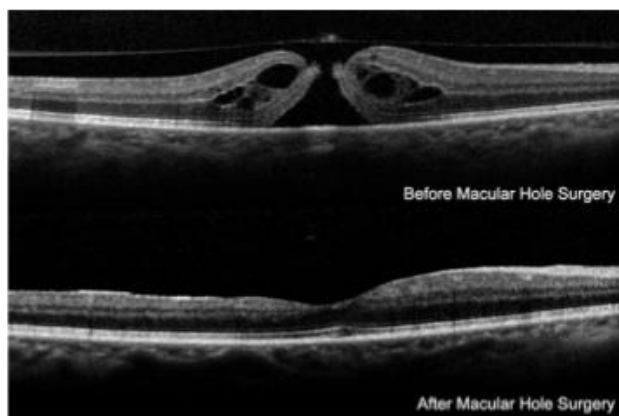


Figure 7. OCT depiction of the macula before and after pars plana vitrectomy<sup>22</sup>

### ***Vitrectomy Complications***

As with any surgery, there is an inherent risk of complications with vitrectomy. One of the most common postoperative complications is accelerated cataract formation. Cataract formation is influenced by various factors such as surgery duration, light toxicity, intraocular tamponade, and increased oxygen tension. Specifically, increased oxygen tension in the eye can lead to oxidation of lens proteins resulting in a cloudy lens. Another less common reason for cataract formation after vitrectomy is accidental rupture of the posterior capsule by trauma during surgery,

leading to hydration and thereby opacification of the lens.<sup>23</sup>

There is also potential for elevated intraocular pressure (IOP) post-operation. Transiently, this finding may be caused by the gas or oil tamponade. Chronic elevation is most likely due to inflammation caused by length of surgery, a response to the prescribed steroid eye drops, or patient medical conditions such as diabetes or glaucoma. IOP should be monitored and managed by a physician as persistent IOP elevation may cause damage to the optic nerve.<sup>24</sup>

Furthermore, manipulation during vitrectomy can cause new tears in the retina since the vitreous is being manipulated.<sup>25</sup> If the retinal tear is caught before progression to retinal detachment, photocoagulation or cryopexy may be performed to secure the retina to the wall. In the case that retinal detachment occurs, the physician may recommend different procedures based on the severity of the detachment. Symptoms of retinal detachment include photopsia, sudden floaters, and reduced peripheral vision.<sup>26</sup> This is a medical emergency where sight may be permanently lost, so medical help should be sought immediately after experiencing these symptoms.

Other complications include infection, hemorrhage, and blind spots; however, these are uncommon.<sup>21</sup> Still, it is important to recognize the possibility and probability of these complications so

educated decisions are made about undergoing vitrectomy.

The success rate for vitrectomy MH closure is approximately 90%, contingent on cause, stage, and size of MH. MH due to trauma or high myopia have slightly lower closure rates. It has been hypothesized that this is because of additional traction due to long axial length and posterior staphyloma.<sup>27,28</sup> In regards to size, holes above 500 microns in diameter have a 50% closure rate, significantly lower than MH with a diameter less than 400 microns.<sup>27</sup> MH stage has also been associated with closure success rate, as stage 2 MH have higher closure rates than stage 3 and 4 MH.<sup>27</sup> In order to increase the probability of closure, different ILM flap techniques and tamponade agents may be used during surgery, depending on the cause, stage, and size of the MH. If unsuccessful, numerous techniques have been tested to aid closure, including more extensive ILM peeling, inverted ILM flaps, free ILM flaps, hydrodissection, amniotic membrane grafts, and even autologous retinal transplants.<sup>14</sup>

### **Autologous Retinal Transplant**

A recent innovation for MH treatment is autologous retinal transplant. The peripheral retina contains neuroepithelial stem cells. Transplantation of these stem cells to the macula may improve the healing process.<sup>29,30</sup> Although early studies in cases with large refractory MH have shown some success, drawbacks include difficulty of surgery, increased

chance of multiple surgeries, and increased inflammation following surgery.

Macular hole is a condition that requires early diagnosis and treatment to retain central vision. Individuals should seek professional help if they experience symptoms or have any concerns regarding macular holes.

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