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Short Communication
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Macular Hole

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A 65-year-old man reported to the office emergently with a chief complaint of blurry vision at distance and near OU in the setting of seeing some new floating spots. He explained that he had woken up early that morning seeing well but as the day wore on he noticed the computer looked funny with his right eye primarily affected. His ocular history included cataract surgery OU 4 years ago with YAG capsulotomies, OU within the last year. His systemic history included hypertension and high cholesterol; for which he was properly medicated. He denied trauma or allergies of any kind.

His best corrected entering visual acuities measured 20/70 and 20/20 at distance and at near. There was no improvement upon pinhole testing or refraction. The retinoscopic reflex OD appeared peculiar. The external examination was normal for color, brightness and extraocular muscle motilities. Confrontation fields were normal OS but the facial Amsler grid was distorted OD. There was no afferent pupil defect. Biomicroscopy uncovered normal anterior segment structures with normal Goldman applanation pressures of 16 mmHg in both eyes. Dilated examination uncovered a stage II incomplete macular hole, OD.

Additional testing, over time, included traditional Amsler grid testing as well as 21-line raster optical coherence tomography and color photography (see photographs).

Acute idiopathic macular holes (IMH) can significantly reduce central vision [1-7]. Women over the age of 65 are most commonly affected [1, 4, 6, 8-14]. Most holes are unilateral with an incidence of bilaterality reported in up to 29% of cases, according to some reports in the literature [4].

Symptoms of impending macular holes include decreased vision and metamorphopsia. The symptoms are produced when there is vitreous shrinkage (vitreous syneresis) with or without traction from persistent macular vitreous adhesions (vitreomacular adhesion syndrome or vitreomacular traction syndrome) [15-17]. Maculopathies caused by this chronic pathophysiology include epiretinal membrane formation (ERM-retinal wrinkling) and cystoid macular edema (CME) [15-17]. Macular hole formation is induced secondary to mechanical traction and schisis formation leading to foveal tissue distortion, focal fluid accumulation with eventual subretinal tissue detachment [15-17].

The current standard for treatment of full-thickness macular holes (FTMH) is surgery, rather than observation. Surgical procedures have been shown to decrease the incidence of hole enlargement and can often result in significant restoration of pre-incident visual acuity [1,2].

The prevalence of macular holes is 3.3 per worldwide 1000 [4, 11]. Causes of macular holes include trauma, solar retinopathy and degenerative/pathologic myopia [9, 10]. The majority of macular holes result from are age-related vitreoretinal changes or are idiopathic in nature [8, 12, 19]. IMH usually affect older patients (6th-7th decade of life) [1, 9]. Women are affected more commonly than men at a ratio between 2-3:1 [8].

The etiology of IMH is related to vitreomacular stress [9]. Cystic macular edema, choroidal vascular insufficiency and anteroposterior vitreoretinal traction have also been implicated [9, 12, 14]. Tangential vitreoretinal traction has gained acceptance as a mechanism as well [1, 3, 8-10, 12, 20].

Gass devised the widely accepted method of classifying macular holes [8, 13, 21]. He separated the progression of macular holes into four stages.

Stage 1 (macular cyst) is defined by a serous detachment of the fovea. In early stage 1 holes (stage 1a) the concavity of the fovea is lost and a yellow spot representing increased visibility of the yellow xanthophyll pigment appears in the center of the macular area (lemmon drop nodule). As traction builds and this stage progresses (stage 1-B), the pigment is displaced outward, towards the boundaries of the circular foveal depression causing it to appear ring-shaped [8, 13, 21]. This change in appearance, from a nodule to a ring, is specific to macular hole development. In 50% of cases the process spontaneously aborts [21].

Stage 2 (early macular hole/lamellar macular hole) is defined by a full-thickness retinal dehiscence with intact photoreceptors

[22]. Biomicroscopically, it appears as an oval, crescent, or horseshoe shaped retinal defect on the inside edge of the foveal xanthophyll ring. They can also appear as a central, round retinal defects surrounded by a rim of elevated retina. Secondary to centrifugal movement of retinal receptors, these macular holes may enlarge. Up to 70% of stage 2 holes progress to stage 3 and they can also occasionally spontaneously abort [9, 21].

Stage 3 (full thickness macular hole [FTMH]) is defined by a 400m-600m diameter hole, usually surrounded by a rim of elevated retina. The vitreous becomes separated from the fovea and a pre-foveal opacity (operculum), representing this separation, may or may not be visible. Posterior vitreous detachment (PVD) is not present [8, 21].

Stage 4 (FTMH with PVD) are defined by the final step of a detachment of the posterior vitreous from the optic nerve. A pseudo-operculum, if present, is usually found near the temporal border of the Weis's ring.

Patients with stage 1 and 2 macular holes present with symptoms of decreased visual acuity/metamorphopsia [9-11]. Stage 3 finds visual acuity significantly more depressed. Acuities for eyes with FTMH range from 20/40-5/200 with an average of 20/200 [8]. Central scotomas corresponding to the macular hole can be demonstrated with an Amsler grid, facial Amsler grid (confrontation field testing) or by presenting a thin beam of light across the hole and asking the patient to observe if the beam is distorted, fractured or incomplete (Watske-Allen sign) [7, 8].

The prognosis for IMH holes is good. Fifty percent of stage 1 macular hole's progress to stage 3 [7-9]. Seventy percent of holes reaching stage 2 progress to stage 3 [9]. If a patient develops a macular hole in one eye, there is a 29 % risk of macular hole development in the fellow eye [4]. Risk factors for the development of macular hole in fellow eyes include pigment epithelial defects and macular retinal thinning [9]. The presence of a PVD in the unaffected eye lowers the risk to almost 0 % (release of retinal traction) [9, 23-25].

Stage 1 holes, along with vitreomacular adhesion and traction syndromes holes should be observed for progression. Patients can be advised to the signs and symptoms of progression as well as use a home Amsler grid to periodically monitor themselves for changes. Stage 2-4 holes may be treated by a retinologist experienced in vitreomacular procedures. Current modalities include 3-port pars plana vitrectomy with gas tamponade using SF6 (sulfahexafluoride) or C3F8 (perfluoropropane) with subsequent 80%-90% face-down positioning for 2 weeks. Classic complications following surgery include cataract formation, RPE alterations, other retinal breaks/detachment, hole enlargement, hole reopening, vascular occlusion, cystic macular edema, choroidal neovascularization, field loss and endophthalmitis [3, 27, 28].

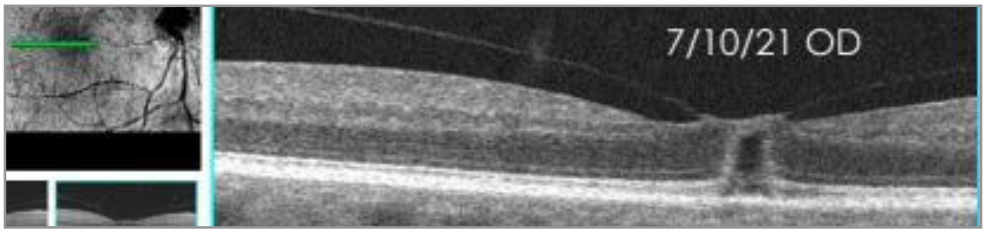
Kelly and Wendel were the first to use the technique of pars plana vitrectomy and gas tamponade followed by 1 or more weeks of face-down positioning to achieve anatomical hole closure. Acuity improvement of 2 or more lines occurred in 42% of patients [5, 6, 24].

The Vitrectomy for Macular Hole Study Group conducted a multicentered, randomized clinical trial and found that pars plana vitrectomy in patients with stage 2 macular holes decreased the incidence of progression to stages 3 and 4 with better acuities compared to observation alone. Willis and colleagues found patients who had pars plana vitrectomy performed within 2 months of the onset of symptoms faired best [2, 22].

Recent studies have investigated the use of adjunctive agents and removal of the internal limiting membrane during vitreomacular procedures to improve anatomic hole closure and acuity recovery [6, 29, 30]. Using thrombin-activated fibrinogen with selective removal of the internal limiting membrane achieved closure in up to 96% of cases [29]. The use of chemical adjuncts, while not proven conclusively, shows promise and is worthy of clinical study. More recently vitrectomy with autologous platelet plugging has shown great success in macular hole repair helping to reduce the "face-down" requirement [29, 31].

The face-down positioning following macular hole repair remains a major issue for patients considering the procedure. While the results of recent reports do not necessarily prove that "face-down" positioning is more likely to close a repaired macular hole when compared with "face-forward" positioning the "face down" position seems to make logical sense as the tamponade effect is maximized; this seemingly supports the possibility that the guideline of "face down" positioning produces superior outcomes [32].

This patient was promptly referred to the retinologist who confirmed the diagnosis. A three-port pars plana vitrectomy procedure was scheduled but then aborted when his symptoms spontaneously resolved. He was reappointed for 4-6 month follow-up examinations with repeat OCT testing and home Amsler grid monitoring. His visual acuity has improved to 20/25 and to date has not required the procedure. Even in the setting of the literature he has not elected to move forward with a procedure in the absence of symptoms with good function.



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