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Idiopathic macular hole – literature review

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HIGHLIGHTS

Full-thickness idiopathic macular hole is a prevalent cause of visual impairment, predominantly affecting elderly females, with metamorphopsia being a hallmark symptom; diagnosis relies on optical coherence tomography, and treatment typically involves posterior pars plana vitrectomy with internal limiting membrane peeling.

ABSTRACT

Full-thickness idiopathic macular hole (MH) is a common cause of visual loss, affecting 3 per 1000 people. It usually affects females in their sixth to eighth decades of life. Best Visual Acuity decreases to less than 0.5. A typical MH's symptom is the pincushion metamorphopsia, where the shape of an observed object is enrolled inwards and lines are bowed towards the center, in the direction of the fixation point. Over the years many theories of MH formation have been revealed. A recently approved theory states that vitreomacular adhesion with focal contraction of the perifoveal cheloid and pathologic traction can lead to a loss of full-thickness central retina causing a MH. The most valuable diagnostic procedure for MH is optical coherence tomography. Among other useful examinations are fluorescein angiography and angio-OCT. Classification of the stages of development of MH is done with an anatomical Gass classification and the recent The International Vitreomacular Traction Study Group Classification of Vitreomacular Adhesion, Traction, and MH. Natural history of the MH differs in the early and advanced stages. Pharmacological vitreolysis with ocriplasmin is used in the treatment of the disease. The most effective therapy is the posterior pars plana vitrectomy. Dye and peeling of the internal limiting membrane helps to achieve the closure of the MH and the good visual acuity.

Key words: full-thickness MH, natural history, genesis theory, classification, treatment

INTRODUCTION

Regardless of age, the comfort of a person's life is inextricably linked to good visual acuity. Both diseases that develop over a spectrum of symptoms and those with a sudden onset can negatively affect the quality of daily functioning. They seem to be all the more troublesome for the patient if, in a short period of time, they lead to damage to central vision involving the macular area. The macula is the space of the posterior pole of the eyeball, 2 mm in diameter, the central fovea forming it consists of a fovea (0.35 mm in diameter) and the surrounding slope. Surrounding the fovea is the retinal nonvascular zone (0.6 mm in diameter), nourished by the choroidal capillary network. The retina in the fovea is thin. Its central half is filled by an inverted cone-shaped zone made up of Muller cells (Muller cell cone). The base of the cone forms the bottom of the well and extending into the cone area forms the inner boundary membrane, while its truncated peak in the umbo forms the outer boundary membrane, under which there are no receptor cell nuclei.
The inner boundary membrane is only 10–20 nm long at this point. Through the cytoplasm of the Muller cells, located at the top of the cone, long nerve fibers radiate out, projecting above the outer boundary layer, towards the photoreceptors. The cone cells are a reservoir of the yellow pigment (xanthophyll) and provide a specific reinforcement connecting the receptor cells in the fovea [1, 2]. The loss of all retinal layers within the anatomical fovea results in the formation of a full-thickness macular hole (MH).

ETIOLOGY, PATHOPHYSIOLOGY AND CLASSIFICATION

Full-thickness idiopathic MH is a relatively common cause of visual impairment with an average incidence of 3 per 1000 people [3]. It mainly affects women between the sixth and eighth decades of life. At younger ages, it is mainly associated with myopia [4]. Elevated blood fibrinogen levels and a history of glaucoma increase the incidence [5]. The presence of the disorder is not influenced by cardiovascular disease, hypertension or status post ovary removal. The use of estrogen is considered a factor reducing the risk of MH, but it has been observed that higher levels of estradiol in the vitreous body occur in those with the condition. It has also been noted that the MH occurs more frequently in women who experience menopause with greater hormonal fluctuations, where hot flashes and sweats are not uncommon [5, 6]. In the majority of cases, MH occurs spontaneously; however, a number of conditions that may accompany it have been described in the literature, including proliferative diabetic retinopathy, congenital defects of the nerve II disc, choroidal neovascularisation, Best's disease or adult macular degeneration and retinal arteriovenous junction [7].
In the first half of the XIX century, holes in the central retina were mainly associated with cases of ocular trauma and were not treated as a separate disease entity. The earliest report making the diagnosis of MH dates back to 1869, when Knapp described the case of a patient who was initially diagnosed with post-traumatic haemorrhage in the macula and later found to have a full-thickness hole [7]. These early observations underpinned the theory of the traumatic origin of the hole. It proved that the loss of the central retina occurs under the influence of the energy transmitted by the impact to the vitreous body and by a contrecoup mechanism. Two years later, in 1871, Noyes gave a detailed description of the ophthalmoscopic examination of MH [8]. Examining a 13-year-old girl after blunt trauma to the eyeball, he noted the difference in the depth of the focus from the surface to the depth of the lesion, finding a full-thickness retinal defect in the macula. In his observations, he stated that the retina appeared to have been "cut out with scissors or a punch" [9]. The authorship of the first histopathological description of MH is attributed to the Fuchs and Coats. They too proposed the theory of cystic and vascular degeneration as the cause of the formation of a full-thickness retinal defect in the hole. Coats, observing cysts in the retina adjacent to the walls of the hole, drew the suspicion that swelling of the central retina, which he found not only in trauma patients, leads to MH formation [9, 10]. The theory of cystic degeneration was also addressed by Zeeman, Vogt and Samuel, among others. The last author stated that a special type of cavity develops in the fossa because it lacks the Muller cell support spicules that limit swelling, resulting in the formation of round cystic lesions [11]. Researchers differed in their opinion as to where the first retinal cysts form: whether in the nerve fibre layer or the outer plexiform layer, but the process of full-thickness MH development they recognised looked similar. Swelling was recognised in the centre of the posterior pole, followed by cystic degeneration with numerous spaces, in which cystic rupture then occurred and finally a full-thickness hole was formed [11, 12]. Advocates of a non-traumatic pathogenesis of MH formation have also leaned towards a vascular theory. Among others, Coats and Kuhnt argued that age-related changes in the retinal vessels precede macular cystic degeneration [10]. This theory is often referred to as ocular vasospasm (angiospasm) [7, 10]. The foundations of the modern vitreous theory of MH formation were laid in 1924 by Lister. He considered the involvement of anterior-posterior vitreoretinal traction, the pull and release of which could lead to the formation of a full-thickness macular defect [10, 13]. The pull originated from the base of the vitreous and was transmitted by shrinking trans vitreous fibres of the vitreous body [7]. In the 1980s, Morgan and Schatz combined the vascular, cystic degeneration and vitreous theories defining it as involutional macular attrition. According to the authors, vitreous forces pulling the involutivity altered retina of the posterior pole of the eyeball are responsible for the formation of the MH. Underlying the involutional changes

are disorders of the subplasma choroidal vasculature leading to an adverse change in perfusion and the formation of focal lesions within the fovea and pigment epithelium. Underlying these pathologies, cystic degeneration develops resulting in macular thinning, which becomes more susceptible to the damaging forces exerted by the vitreous [14, 15]. Morgan and Schatz, however, did not quibble over the direction of the forces acting. They did not divide vitreous traction into anterior-posterior and tangential-tangential. The nature of vitreous traction was explored further by Gass and Johnson when they announced in 1988 the hypothesis that the formation of a full-thickness MH occurs as a result of focal contraction of the pre-foveal cortex of the vitreous body and the action of tangential vitreous traction. These authors believed that adhesion of the vitreous body in the macula was necessary for the formation of MH, and in the observations they made, the vitreous body moved freely without visible anterior-posterior traction [7]. In 1995, Gass, based on the above theory, gave an updated classification of the successive stages of MH formation visible in the slit lamp [16]. Initially, tangential traction of the pre-foveal part of the vitreous cortex detaches the retina in the fovea giving an image of a yellow disc about 100–200 μm in diameter. The foveal depression disappears. This is stage 1A of the MH. The yellow colouration of the ring is due to the presence of xanthophyll. As a result of further thickening and shrinking of the vitreous cortex, the foveal retina is raised to the level of the surrounding periocular retina, emphasising the foveal retina around the centre. On the fundus of the eye, a yellow ring (donut-shaped) is visible, which is a serous foveal retinal detachment with lateral displacement of the xanthophyll – stage 1B threatening hole. Eventually, the receptor layer in the centre ruptures and there is centrifugal displacement of the cones, xanthophyll and radial nerve fibres, while the overlying inner limiting membrane, the horizontal protuberances of the Muller cells and the thickened vitreous may remain intact forming stage 1B – concealed hole. In the light of the biomicroscope, it is not possible to detect the transition from the threatening stage to the hidden hole. In stage 1A and 1B, complete loss of the neurosensory retina in the fovea is not observed. At this stage, stage 2 may develop – the full-thickness hole is less than 400 μm in diameter and the vitreous body is still adjacent to the optic disc and macula. As a result of spontaneous vitreous-hole detachment, a pre-hole haze may form – a pseudovessel, consisting of a thickened vitreous and proliferating glia, larger than the underlying hole. The fundus of the eye then shows a central, circular cavity with a seam of raised retina and pre-foveal opacity. If a hole develops in the pre-capillary cortex of the vitreous body at its junction with the edge of the hole, the haze is not visible and in the light of the biomicroscope an eccentric, oval, horseshoe-shaped retinal defect inside the yellow ring is observed. Stage 2 provides the first biomicroscopic opportunity to identify a full-thickness hole in the macula. Further enlargement of the hole beyond 400 μm leads to stage 3, and, when accompanied by detachment of the vitreous body from the macula and nerve disc II to stage 4 [16]. At this stage of full-thickness MH, the ophthalmoscope may show a lid, radial retinal striations and, at the bottom of the defect, drusen, yellow-white deposits or atrophy of the pigment epithelium arguing for a long duration of the disease. Epiretinal membranes, cystic degeneration and an envelope of subretinal fluid are often found [7]. In 2004, a diagnosis of additional stage 0 MH was proposed. By examining the healthy eye in patients with MH, it has been found that, despite the absence of clinical symptoms and a normal fundus appearance, an oblique traction reaching the fovea can be found on optical coherence tomography (OCT). It often does not affect the morphology or thickness of the macula but poses a risk of developing a second full-thickness hole [17]. A 2014 study identified two types of traction: V-shaped and U-shaped. V-shaped tractions lead to changes (formation of fissures, cysts) in the outer layers of the retina, while U-shaped tractions lead to changes in the inner layers [18]. With the development of OCT studies, it has been shown that the condition preceding the formation of a full-thickness fovea is most often intraretinal cysts, and the theory deprecated by Gass of the involvement of anterior-posterior traction in the formation of MH began to be appreciated again in the early XX century by authors such as Azzolini, Haouchine, Johnson and Tanner. It was shown that centrifugal traction pulling at the retinal fovea disrupts the connections between Muller cell cone cells and photoreceptors, leading to the formation of a cystic lesion, which can then propagate in its vault and spread deep into the photoreceptor layer to form a full -thickness central retinal defect, while tangential traction contributes to the enlargement of the hole. The retinal loss is not accompanied by loss of the cones. In the course of hole formation, they are displaced lateral to the centre of the hole [19]. The latest classification of full-thickness MHs has been proposed in *The International Vitreomacular Traction Study group Classification of Vitreomacular Adhesion, Traction, and MH*. According to the new systematics, determining the stage of a hole should take into account: the size of the hole measured at the narrowest point with the OCT camera, the presence or absence of traction, and the cause of the hole – primary and secondary. Holes below 250 μm are small holes, 250–400 μm are medium holes and above 400 μm are large holes. Primary holes – idiopathic, vitreoretinal traction is involved in their formation. Secondary holes – resulting from an additional factor e.g. trauma, lightning strike, high myopia, type 2 telangiectasia, age-related macular degeneration treated with anti-VEGF [20]. The previous classification has been replaced with the following equivalents: Stage 0 corresponds to vitreous body adhesion

(VMA – vitreomacular adhesion) in the macula. A stage 1, threatening hole is vitreomacular traction (VMT – vitreomacular traction). Both VMA and VMT can be subdivided into focal (up to 1500 μm), wide (above 1500 μm) and isolated and associated with other eye diseases such as age-related macular degeneration, retinal vascular occlusion, etc. Subsequently, stadium 2 and 3, small and large hole correspond to small, medium or large MH with associated VMT. The final stadium 4 MH with vitreous detachment is a small, medium or large full-thickness MH without VMT [20].

NATURAL COURSE OF THE DISEASE

The course of the disease varies greatly depending on the stage of the hole. A threatening MH may regress spontaneously, remain stable or progress to a full-thickness hole. De Bustros, during a two-year follow-up of 35 patients included in the *Vitrectomy for Prevention of Macular Hole Trial*, found progression to a full-thickness MH in 40% of patients from stage 1. The progression from stage 1A to 1B usually occurred within weeks or months, while the formation of a full-thickness retinal defect was observed after an average of 4.1 months [21]. Together, Kokame and De Bustros noted that visual acuity may serve as a prognostic factor for progression to a more advanced stage of the disease. The risk of progression from a stage 1 MH to a full-thickness hole is higher in those with a best corrected visual acuity (BCVA) of 0.4 and worse. In the patients observed, with a BCVA between 0.4 and 0.25, 60% of 15 eyes progressed to subsequent stages of the disease, while with a BCVA of 0.8–0.5, 30% of 20 eyes progressed to a more advanced stage [22].

Kakehashi et al. observing a group of 23 patients diagnosed with stage 1 MH, reported disease regression in 22% [23]. In contrast, Hikichi et al. during a one-year study involving 40 patients with threatening MH, found regression in 48%, stabilisation in 29% and progression to full-thickness hole in 23%. Stopping disease progression and regression was most often associated with vitreous detachment in the macula [24].

The majority of stage 2 holes progress further, while less than 10% decrease in stage [23]. Hikichi, during a mean follow-up of 4 years (2–8 years) of 48 eyes with stage 2 disease, found 96% progression and 4% arrest of lesion progression. In addition, 85% of the orifices enlarged beyond 400 μm [25]. Casuso et al. focusing on 15 individuals with stage 2 MH, showed 100% progression to more advanced stages of the disease within 5 years [26]. In contrast, Guyer et al. presented results where 33% of the lesions regressed and 67% progressed to stage 3 and 4 disease [27].

The holes in stages 3 and 4 rarely regress. Most often they lead to deterioration of central vision, increase in diameter, cystic changes and photoreceptor atrophy. In the *Vitrectomy for Macular Hole Trial*, Freeman et al. reported regression of the disease in 4% of patients during a 6-month follow-up. However, closure of the hole was understood not as a return to normal macular morphology, but as a flattening of the edges of the hole [28]. Casuso et al. observing 48 eyes for 5 years reported no cases of hole closure in stadium 3 and 4. The lesioned eyes remained stable or underwent further progression [26].

The results regarding the occurrence of MH in the companion eye are also very divergent. Otsuji et al. observed that with a detached vitreous body in the macula, no healthy eye developed a full-thickness hole. In contrast, in a group of 40 eyes with vitreous detachment over a 3-year follow-up period – 11 (28%) developed a full-thickness hole [29]. Ezra et al. put the risk of developing a full-thickness retinal defect in the fellow eye at 15.9% over a 5-year follow-up [30]. Akiba et al. note that the formation of a second eye hole is influenced by the inherent vitreous body and macular changes. In their 39-month follow-up, 8 out of 15 (53%) eyes with cysts in the macula developed a full-thickness hole, while progression occurred in 6 out of 23 (26%) eyes with an initially identified central yellow disc [31]. Other sources report that the 5-year risk of hole formation with an undisconnected vitreous ranges from 10% to 20%, where it falls below 1% after vitreous detachment in the macula. In addition, in patients with MH, the companion eye in which a full-thickness hole forms in the future shows a reduced response on focal electroretinography (FERG) [32].

SIGNS

Patients rarely present to an ophthalmologist at the onset of the disease, as stage 1 MH progresses asymptomatically or with mild visual disturbances. Often more advanced stages are discovered incidentally when the patient covers the healthy eye or when metamorphopsia is more severe and central vision is impaired. Visual acuity usually falls below 0.5. Typically, the disease is accompanied by cushion deformities (pincushion metamorphopsia). The shape of the object the patient is looking at is pulled inwards and the lines curve inwards towards the fixation point. This is because the foveal photoreceptors become centrifugally displaced [19]. Occasionally, micropsies may also occur.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

In order to establish the diagnosis of MH, a thorough fundus examination must be performed. The best known is the Watzke–Allen test, where a vertical and horizontal beam of light is passed through the altered macula using a Goldmann lens. Patients with stage 3 or more holes will report a break in the light gap (positive test) while those with a smaller hole or other macular pathology will report line thinning (negative test) [3, 32]. Also, when projecting

a 50 μm beam of targeted laser light onto the MH, patients with a full-thickness central retinal defect will not find it. In contrast, they will report the appearance of lines drawn to the centre of the chart in the Amsler test [19]. Among imaging studies, the gold standard for the diagnosis of MH is Optical Coherence Tomography (OCT). It allows both detection and assessment of the stage of the MH, as well as differentiation from other disorders of the posterior pole of the eye. It provides a near histological view of the appearance of the lesion and also measures its size [10, 32]. Spectral Domain OCT (SD OCT) allows a more accurate assessment of the morphology of the MH and, in particular, the identification of changes at the level of the junction of the outer and inner photoreceptor segments. The examination provides a wealth of numerical data, allows 3D imaging of the orifice and also predicts the quality of vision after surgical intervention [33]. Fundus autophotofluorescence, which is a non-invasive test by mapping changes at the level of the pigment epithelium (RPE), allows observation of pinpoint fundus fluorescence at the site of the hole at stage 2 and its larger area at higher stages [3]. Fluorescein angiography, while not essential for the diagnosis of MH, can be used to confirm the diagnosis and assess the loss of RPE and the extent of oedema. In the early stages of the hole, the lesions may not be visible on the angiogram and stage 1 and 2 may be indistinguishable. The examination may show a central window defect in the RPE, the hyperfluorescence being more strongly expressed the higher the stage of the disease. In addition, the cuff of fluid surrounding the hole is visible as a hypo- or hyperfluorescent ring [32] . One of the most recent tests to investigate flow changes in the choriocapillaries of the macular area is angio-OCT. Analysis of the data suggests that the vascularisation area and density of choriocapillaries in the macula are significantly smaller in the MH than in the healthy eye [34]. Static perimetry, particularly microperimetry, is also a useful test for determining the severity of macular changes. Determination of the sensitivity of the visual field using markers projected onto the macular area makes it possible to demonstrate the area of absolute macula and its change after vitrectomy. The fixation observed during the procedure gives information about the actual location of the centre of sharpest vision. The disadvantage of microperimetry is the long duration of the examination, which may result in poorer patient co-operation. Dynamic B-projection ultrasonography, which allows the assessment of vitreous adhesion in the macula and the determination of the presence of a lid, has now lost its importance.

Making a diagnosis of full-thickness MH requires a thorough fundus examination and differentiation with other ophthalmic conditions. Stage 1 can be misdiagnosed as a number of other disorders including solar retinopathy, laser index retinopathy, macular yolk degeneration, central serous retinopathy, vitreoretinal pull syndrome or cystoid macular oedema. The diagnosis of solar retinopathy is usually exaggerated by a history of sun exposure. Central serous retinopathy occurs in younger individuals than MH. In contrast to patients with MH, in vitreoretinal pull syndrome the retina in the fovea is elevated above the level of the surrounding retina, and subretinal fluid is observed in age-related macular degeneration or serous retinopathy [3, 7]. More advanced stages of MH should be differentiated from pseudotumours. Pseudoaneurysms are centrally located depressions of the epiretinal membrane (ERM). In contrast to full-thickness holes, there is no cuffing of subretinal fluid and drusen-like fundus changes, and visual acuity is better [7].

TREATMENT

The management of MH varies according to stage and the implementation of surgical treatment is debatable.

Initially, full-thickness MHs were attempted to be treated pharmacologically with anxiolytics and vasodilators. Surgical methods of MH supply for many years concerned holes with coexisting retinal detachment. Cryotherapy, photocoagulation of the macular area, gas or oil injection into the vitreous chamber without vitrectomy were performed [7, 35]. In 1961, Meyer-Schwickerath used banding with drainage of subretinal fluid, cryotherapy and scleral intrusion to flatten the hole [7]. In contrast, in 1982 Gonvers and Machemer were the first to perform vitrectomies with gas injection into the vitreous chamber and subsequent face-down maintenance. By omitting the use of photocoagulation of the macular area, they avoided destruction of macular function [36]. Kelly and Wendel, extending the above method to include removal of the vitreous cortex and epiretinal membranes, obtained in 1991 a significant improvement in vision and flattening of the macular area in patients operated on for independent MH [37]. In 2010, the technique was proposed to be supplemented by the application of an inverted flap dissected temporally from the macula of the internal limiting membrane to close especially large holes [38]. As it is associated with intra- and postoperative difficulties in terms of displacement or loss of the flap, followed by poorer visual acuity and reopening of the hole, attempts are being made to take it from the upper macular area with equally good anatomical and visual results [39, 40].

Over the years, research has also been conducted into the use of various adjuvants that, when administered intravitreally during surgery, would improve the prognosis of hole closure and improve postoperative visual acuity. When used intraoperatively, these substances may have a positive effect on hole closure, especially in traumatised eyes with high myopia, in chronic holes and in children [35]. Platelets applied in advanced stages of MH accelerate its healing

process [41], and also enable closure of MH in eyes with high myopia [42]. Also, intraoperative administration of the plasmin-thrombin complex results in good visual acuity and does not increase the incidence of perioperative complications [43]. Autologous platelet concentrate as well as whole blood are helpful in achieving positive results in persistent MHs that have not closed after vitrectomy with ILM (*internal limiting membrane*) peeling and gas administration [44]. In contrast, no additional benefit is found with the use of transforming growth factor beta (TGF-β) [45, 46] and serum [46–48].

MHs occurring in eyes with high myopia are successfully treated by surgery to penetrate the macular area using an exoimplant of silicone sponge and a titanium pin [49]. Vitrectomy with the application of human amniotic membrane over the MH area also has an encouraging effect in closing the hole and improving vision [50].

The current surgical treatment of full-thickness MH is posterior vitrectomy with removal of the internal limiting membrane and endotamponade with gas. Thorough posterior detachment of the vitreous body allows removal of the pulling vitreous and macular tractions, and the use of dye facilitates thorough scrubbing of the inner limiting membrane of the macular area [3]. For those with MH greater than or equal to 400 μm diameter, postoperative head positioning is recommended [51]. In 2013, the EVRS (European VitreoRetinal Society) published the results of a year-long international study on MH (*The EVRS MH Study*). In 85.7% of cases undergoing MH vitrectomy, closure of the hole was achieved. Other reports even indicate a 91–98% success rate [52]. The most important factors at the time of surgery affecting hole closure are the stage of progression, the duration of the hole and the use of dye to stain the ILM. Surgery at a less advanced stage is more likely to result in closure of the MH. The use of dye improves the chances of success of the surgery and does not affect post-operative visual acuity. Once the hole is closed, vision improves and the effect increases over time. The most common postoperative complications include cataracts, retinal detachment and failure to close the hole [53]. Full-thickness stage 1 MHs are usually left for observation. Spontaneous closure is more common in small holes, and sometimes vitrectomy with ERM peeling is recommended in such cases. As an alternative in stage 2 MH to posterior vitrectomy, pneumatic vitreolysis or pharmacovitreolysis may be considered [52]. Pneumatic vitreolysis is a procedure that seeks to induce posterior vitreous detachment with release of traction in the macula by injecting a gas bubble, usually perfluoropropane (C_3F_8), into the vitreous chamber [54]. A new treatment for early, small MHs is pharmacological vitreolysis using ocriplasmin, a recombinant form of human plasmin. Its injection into the vitreous body results in the release of vitreoretinal traction [3]. Women, before 65 years of age, with a VMA <1500 μm in diameter, an aperture size <250 μm, with their own lens and the absence of ERM have a better prognosis for successful treatment [55]. Posterior vitrectomy remains the most effective treatment option [3], especially if surgical measures were undertaken within 6 months of the onset of symptoms. The average visual acuity is in the range of 0.5, but patients may report central vision disturbances despite hole closure [52].

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