• Review Article •

Metamorphopsia after surgery for rhegmatogenous retinal detachment

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Abstract

● Improvements in surgical techniques have led to 90% success in the surgical repair of rhegmatogenous retinal detachment (RRD). However, anatomical reattachment of the retina does not ensure complete recovery of visual function. The incidence of metamorphopsia remains the most common postoperative complaint, from 24% to 88.6%. Currently, the risk factors of metamorphopsia are categorized into macular involvement, retinal shift, outer retinal folds, subretinal fluid, secondary epiretinal membrane, outer retinal layer damage, and surgical approach. The associations of metamorphopsia with postoperative best-corrected visual acuity and postoperative vision-related quality of life were still controversial. The most popular methods for assessment of metamorphopsia remain the Amsler grid and M-Charts. Most treatments cannot progress beyond the management of negative visual sensations, through methods such as occlusion therapy and aniseikonia-correcting spectacles. The main treatment approach involves RRD prevention and the management of risk factors that can lead to postoperative metamorphopsia after RRD repair. Additional research concerning metamorphopsia treatment, further upgrades of auxiliary inspection methods, and more accurate microstructural assessments are needed to address this common complication.

● **KEYWORDS:** retinal detachment; rhegmatogenous retinal detachment; metamorphopsia; visual distortion

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INTRODUCTION

Metamorphopsia is a visual disorder in which the size, shape, and tilt of objects are distorted; this condition was first described by Foster in 1862[1]. Metamorphopsia can result in deviations that affect the perception of either vertical or horizontal lines. Retinal diseases that can cause metamorphopsia include rhegmatogenous retinal detachment (RRD), age-related macular degeneration, vitreoretinal interface disorders, central serous chorioretinopathy, diabetic and nondiabetic macular edema, and epiretinal membrane (ERM). Micropsia and macropsia are special types of metamorphopsia[2-3].

RRD is defined as the separation of neurosensory retina from retinal pigment epithelium (RPE) because of a retinal break that allows liquefied vitreous humor to infiltrate beneath the retina and then accumulate as subretinal fluid (SRF)[4]. Surgery remains the primary clinical treatment for retinal detachment; the main procedures are pars plana vitrectomy (PPV), scleral buckling (SB), and pneumatic retinopexy (PnR). Improvements in surgical techniques have led to 90% success in the surgical repair of RRD[4-8]. However, anatomical reattachment of the retina does not ensure complete recovery of visual function. Metamorphopsia is the most common postoperative complaint. This review describes the incidence of metamorphopsia after surgical repair of RRD, along with its risk facts, as well as the available clinical techniques for detection of metamorphopsia symptoms. It also explores the associations of metamorphopsia with postoperative best-corrected visual acuity (BCVA) and postoperative vision-related quality of life.

INCIDENCE OF METAMORPHOPSIA

The success rate of surgical repair of RRD can reach 90%[4-8]. However, anatomical reattachment of the retina does not guarantee full recovery of visual function. After the successful repair of retinal detachment, metamorphopsia remains the most common postoperative complaint. A study published in 1983 investigated metamorphopsia after RRD repair. In that study, all 299 patients with RRD underwent SB treatment, and 31.10% reported experiencing metamorphopsia[9]. Since then, there has been increasing research interest in postoperative metamorphopsia. The incidence of metamorphopsia after successful RRD is still at a high level, varying from 24%

to 88.6%[9-18]. Fortunately, metamorphopsia after RRD repair gradually resolves over time with the recovery of retinal structure[10,12,19-20]. During a 6-year follow-up study, postoperative metamorphopsia faded over time in all affected patients[20]. Murakami *et al*[10] showed that the M-CHARTS score significantly improved from 3mo (0.46±0.51) to 12mo (0.28±0.37) after surgery. Fukuyama *et al*[19] recently found that M-CHARTS scores significantly decreased in eyes with macula-off RRD from 1mo (0.61±0.37) to 6mo (0.43±0.37) postoperatively.

MACULAR INVOLVEMENT

Macula involvement is a strong, established risk factor for worse anatomical and functional success in surgical repair of RRD[18].

It has been reported that detachment of the macula results in shortening of the outer segments (OSs) and gradual death of the photoreceptor cells in the macula-off RRD, which may severely affect visual outcomes[21-22]. The incidence of postoperative metamorphopsia is higher in patients with macula-off RRD than in patients with macula-on RRD[9-18]. In studies that included patients with macula-on RRD and patients with macula-off RRD, the incidence of metamorphopsia was generally low (approximately 34.50% to 49%)[10-11,13,17]. In studies that only included patients with macula-off RRD, the incidence of metamorphopsia was much higher (67%-88.6%)[12,15-16]. In a study of 50 patients with macula-off RRD who underwent PPV, Schawkat *et al*[14] found that the incidence of postoperative metamorphopsia was only 24%. However, their study lacked a quantitative method to assess metamorphopsia and used a short follow-up period; moreover, the measurement period (*i.e.*, beginning after the complete disappearance of the gas bubble) was considerably different from the measurement period in other studies, which may have contributed to the low incidence observed.

Macular status not only affects the incidence but also the severity of metamorphopsia. Metamorphopsia is more severe in eyes with macula-off RRD than in eyes with macula-on RRD[9-11,13,17,23-24]. Both Zhou *et al*[17] and Saleh *et al*[13] indicated that preoperative macula-off status was an independent predictor of postoperative metamorphopsia. Notably, macular detachment occurring during surgery also causes the development of metamorphopsia[11].

RETINAL SHIFT

The retinal shift is regarded as the main cause of metamorphopsia after RRD repair[14,24-29]. Shiragami *et al*[24] first described the use of fundus autofluorescence (FAF) to detect retinal displacement. FAF is a noninvasive test that provides discrete funduscopic images based on the enhanced emission of light from lipofuscin[30]. FAF in the RPE is dependent on outer segment renewal and potentially affected by a balance between accumulation and clearance. Therefore, autofluorescence can be interpreted as a clinical sign of metabolic activity in the RPE. For a long period, RPE cells under the retinal vasculature are shielded from light irradiation; this shielding maintains a state of dark adaptation. Subsequently, displacement of the retinal vasculature enables acute exposure to the excitation light and may lead to increased FAF. Thus, a comparison of the retinal vasculature and the parallel hyperautofluorescent “RPE vessel ghost” lines suggests retinal displacement. In the study by Shiragami *et al*[24], unintentional displacement occurred in 62.8% of the operated eyes; this displacement was significantly associated with the extent of RRD and was directed downward in all affected patients. Notably, hyperautofluorescent lines after RRD repair can also occur from other origins, such as outer or inner retinal folds and outer retinal disruption[31].

In RRD, the neurosensory retina is separated from the RPE *via* retinal breaks and SRF accumulation. Therefore, the surgical procedure is intended to seal retinal breaks and remove SRF to restore the arrangement of photoreceptors and RPE. Ideally, the photoreceptors will reattach themselves in a position corresponding to the location before the detachment. However, a retinal shift occurs because of retinal mobility and elasticity, as well as the increased retinal surface area caused by the stretching force from the SRF[32]. Subsequent photoreceptor displacement and false localization of the images acquired by these displaced photoreceptors may induce postoperative metamorphopsia. Lee *et al*[27] reported that postoperative retinal displacement was evident in 72% of 32 consecutive patients with RRD, and there was a strong correlation between retinal displacement and symptoms of vision distortion in the early postoperative period. In a study that used multimodal imaging for the diagnosis of metamorphopsia (n=50), Schawkat *et al*[14] found that 24% of patients reported postoperative metamorphopsia, mainly caused by retinal shift.

Intraocular gas tamponade has been presumed to considerably influence the occurrence of retinal shift. Codenotti *et al*[25] found that retinal displacement was more common in eyes with gas tamponade (10 of 14 eyes; 71.4%) than in eyes with silicone oil (two of nine eyes; 22.2%). Consistent with those findings, dell’Omo *et al*[33] reported that retinal displacement was present in 40 eyes (41.2%) in the gas tamponade group and four eyes (14.3%) in the silicone oil group. They concluded that the type of tamponade (*i.e.*, gas) was the only significant predictor of retinal displacement[33]. Because of its higher specific gravity, lower interfacial tension, and much lower buoyancy force, silicone oil exerts a lower pressure on the retinal surface. Thus, silicone oil forms a more spherical bubble and has a lower surface area in contact with the retina, compared with gas; silicone oil may allow a portion of the detached retina to remain uncovered, thereby permitting

comparatively more gradual and slower reabsorption of the SRF. This would allow the detached and stretched retinal surface sufficient time to decrease to its original size. Conversely, in the gas group, the retinal tissue is rapidly pushed toward the RPE, which may increase the risk that the increased area of the detached retinal surface (caused by SRF-induced stretching) does not decrease to its original size[33].

The direction of retinal displacement is not entirely understood. Codenotti *et al*[25] reported that the direction of retinal displacement after vitrectomy can vary according to the tamponade used. The retina shifts downward in all eyes with gas tamponade and upward in all eyes with silicone oil tamponade. Many studies have proposed that downward retinal displacement after gas tamponade is caused by the effect of gravity on residual SRF in gas-filled eyes[24-25,28,31]. However, other studies have suggested different mechanisms. In a study of 125 eyes with macula-off RRD treated by vitrectomy, Dell'Omo *et al*[33] found that upward retinal displacement occurred in some eyes with gas tamponade (10%). However, downward displacement was more common, even in eyes with silicone oil tamponade (75%)[33]. Moreover, Lee *et al*[27] indicated that the retina had not been simply rotated around the disc or shifted downwards. More complex movements occurred, such that some regions in the macula underwent greater displacement than other regions (*i.e.*, heterogeneous shift)[27].

The early implementation of the face-down position, which may prevent the effects of gravity on residual SRF in gas-filled eyes, may further prevent retinal displacement after vitrectomy and gas injection for RRD[34]. However, this common prevention seems to be controversial. Cobos *et al*[35] suspected that the use of the face-down position did not lead to a lower incidence of retinal shift, compared with the incidences in other studies. In other studies where patients remained in the face-down position, high incidences of retinal displacement were observed[25,27].

OUTER RETINAL FOLDS

The occurrence of outer retinal folds (ORFs) is a common complication after RRD repair. ORFs were identified as hyperreflective lesions that consisted of a folded ellipsoid zone (EZ) and an external limiting membrane (ELM) band, according to spectral domain optical coherence tomography (OCT); corresponding lines of increased or decreased autofluorescence were observed *via* FAF[36-37]. The pathogenesis of ORFs involved factors such as undulations in the detached retina, residual pockets of SRF after retinal reattachment, intravitreal gas, unintentional retinal displacement, and intraoperative or perioperative hypotony[31,38]. The presence of ORFs is significantly associated with the postoperative metamorphopsia[13,36,39]. The adverse structural and functional effects caused by ORFs contribute to the development of metamorphopsia, including loss of phototransduction in photoreceptors that are separated from the RPE, apoptosis and thinning of the photoreceptor layer within the fold, excess tissue covering the adjacent retina, and the distortion from the fold and the adjacent retina[32]. Notably, both the density and the total number of folds are significantly correlated with M-CHARTS scores and decrease over time[39].

SUBRETINAL FLUID

As mentioned above, SRF has a vital role in retinal shift and the occurrence of ORFs after RRD repair. During the retinal shift, SRF causes retinal stretching and an increased surface area, especially in gas-filled eyes; the effect of gravity combined with SRF results in downward displacement[24-25,28,31-32]. There is also a strong relationship between the presence of SRF and the development of ORFs. ORFs pathogenesis mainly involves evolution from pockets of SRF to the formation of ORFs through undulations of detached retina, intraocular gas tamponade, unintentional retinal displacement, and intraoperative or perioperative hypotony[31]. Although there remains disagreement concerning the pathogeneses of retinal shift and ORFs, all current theories regard SRF as the basis. Notably, retinal shift sometimes occurs concurrently with ORFs, potentially as a less severe outcome of the same process[14,25,31]. Furthermore, persistent SRF is associated with irregularities in outer retinal bands[40-41]. Persistent SRF may cause prolonged separation of photoreceptors and RPE in the macula; this separation can disrupt metabolic function in the photoreceptor-RPE complex[42].

Zhou *et al*[17] suggested that the presence of postoperative SRF is an independent predictor of metamorphopsia. In their study, OCT showed SRF in 26 eyes (6.84%); of these 26 eyes, 20 (76.9%) had persistent metamorphopsia[17]. Rossetti *et al*[20] conducted a long-term follow-up analysis of six eyes with metamorphopsia after macula-off RRD repair. They showed that of the two eyes with EZ disruption and SRF at 6mo postoperatively, one eye had dense central scotoma and persistent metamorphopsia[20]. However, it is difficult to determine the direct effect of SRF on metamorphopsia after RRD repair because there has been minimal research concerning the relationship between metamorphopsia and SRF.

SECONDARY ERM

ERM is a fibrocellular membrane that proliferates on the inner surface of the retina, particularly in the macular area[43-44]. It has been divided into two categories, idiopathic and secondary, according to the specific causes of its formation.

Secondary ERM is a common complication after surgical repair of RRD. In the 1980s, the incidence of ERM after RRD repair varied from 3% to 8.5%[45-46]. Considering the poor

sensitivity of imaging techniques at the time, those rates of secondary ERM were presumably underreported. In recent studies, the incidence has varied from 11.5% to 70.3%[47-49]. There have been few assessments of risk factors for ERM after RRD repair. Gharbiya *et al*[47] reported that secondary ERM development was significantly correlated with older age, regardless of the surgical procedure. One study showed that en face OCT imaging had high sensitivity for detecting ERM formation, compared with B-scan imaging. That study identified risk factors for ERM after RRD repair, namely multiple retinal breaks and a maximum retinal break size of ≥2 disc diameters[48]. Hirakata *et al*[49] assessed the strength of associations between ERM formation and background clinical characteristics; they found that an increased risk of ERM formation was significantly associated with preoperative vitreous hemorrhage, multiple retinal breaks, re-detachment, and retinal detachment area, but not with the type of surgical procedure.

Internal limiting membrane peeling reportedly has a significant suppressive effect on ERM formation after RRD repair[50-54]. However, this technique is not appropriate for routine prevention because of disagreement concerning its effects on visual outcomes[50-54]. Furthermore, internal limiting membrane peeling has adverse effects such as peripheral visual field defects, optic nerve fiber layer dissociation, electroretinogram abnormalities, decreased retinal sensitivity, and a higher number of microscotomas[55-60]. Based on the current literature, we suggest that decisions regarding internal limiting membrane peeling are made after an assessment of risk factors for secondary ERM.

Patients with ERM can be completely asymptomatic when the membrane is thin and translucent; its progression to a semitranslucent, thick, and contractile state may result in macular distortion, thus inducing metamorphopsia and loss of central visual function[61-63]. Because secondary ERM mainly develops over several months after surgical repair of RRD, it is rarely a cause of metamorphopsia in the early postoperative period; however, it is responsible for long-term visual distortion. Guber *et al*[64] recently conducted a long-term study to observe changes in postoperative retinal shift. They found that retinal shift significantly decreased after 12mo; however, more patients reported metamorphopsia. Those findings suggested that ERM formation is the leading cause of long-term metamorphopsia[64].

OUTER RETINAL LAYER DAMAGE

The structural integrity of the outer retina layer, such as the ELM, EZ, and interdigitation zone (IZ), is often used to predict metamorphopsia after surgical repair of RRD[10-13,17,65-66].

EZ is anatomically related to (and named for) the ellipsoid component of the photoreceptors, which are packed with mitochondria and have the potential for high reflectivity[67-69]. It has been reported that a disrupted EZ was an independent predictor of metamorphopsia after RRD repair[12,17]. Indeed, EZ is essential to photoreceptors' structural integrity and function, and has important metabolic and light-guiding roles[69-72]. EZ reflectivity might, at least in part, represent the integrity of mitochondria in photoreceptors, which then extends to the photoreceptor function[72]. A decrease in the EZ intensity may be reflecting the reduction in healthy or functional mitochondria in the photoreceptor inner segment, which could translate to abnormal photoreceptor function. Moreover, decreases in EZ reflectivity, which may be caused by cone loss secondary to RRD, may also result in metamorphopsia[13].

IZ is a hyperreflective brand that represents the interdigitation of the apical processes of the RPE with the cone OSs[67-69]. IZ disruption has been described after RD and is significantly correlated with the postoperative metamorphopsia[10,12-13]. The recovery of IZ after retinal detachment surgery is important in relation to the visual recovery because it would indicate the reestablishment of the relationship between the OSs of the photoreceptors and the pigment epithelium when the shedding of the RPE is recovered. Notably, the gradual recovery of the EZ and IZ that, respectively, indicate that the energy metabolism of cones and the shedding by the RPE have been recovered are 2 factors that must be evaluated in the interpretation of the OCT images to perform an accurate diagnosis and good prognosis of retinal pathologic features[73].

ELM is a hyperreflective band that comprises clusters of junctional complexes and microvilli between the Müller cells and the photoreceptors[69,73]. The presence of ELM defect may therefore indicate damage or loss of both cell types, which may result in metamorphopsia. Interestingly, the restoration of IZ and EZ is also associated with the number of affected bands within the photoreceptor layers. For example, eyes with intact ELM have a greater likelihood of recovering their IZ and EZ, compared with eyes that have initial ELM defects[74]. Disruption of both the ELM and the EZ reportedly indicates that morphological changes extend towards photoreceptor cell bodies and Müller cell cones, while disruption of the EZ alone (*i.e.*, the ELM remains intact) indicates that morphological changes are restricted to the photoreceptor inner segment/outer segment level[75]. Furthermore, it was hypothesized that Müller cells provide primary structural support for the fovea, acting like a plug to bind together photoreceptor cells[76]. If Müller cells are unable to support reapproximation of the normal photoreceptors to the central fovea, growth of the normal inner and OSs may not occur.

SURGICAL APPROACH

The main surgical approaches for RRD are PPV, SB, and PnR. Although PPV is reportedly the most popular intervention

worldwide, the optimal approach for the management of RRD is unclear[6,77-78]. There have been extensive comparisons of SB and PPV for patients with RRD. A prospective randomized multicenter clinical study showed that SB provided greater BCVA improvement in phakic eyes, while no difference in BCVA was observed in pseudophakic eyes[79]. Ryan *et al*[80] reported that for phakic moderately complex primary RRD, SB had better visual outcomes than PPV. A Meta-analysis of treatments for primary RRD, published in 2019, suggested that PPV and SB had few or no differences in terms of primary success rate, visual acuity gain, and final anatomical success. However, there were some subtle differences in other aspects. For example, PPV resulted in less retinal redetachment, greater cataract progression, and the development of new iatrogenic breaks[81]. These conclusions were supported by the findings of Zhao *et al*[82]. Their study results indicated that releasable SB caused fewer intraocular complications and less cataract progression, although releasable SB and PPV procedures had similar effects on functional and anatomical success for patients with phakic primary RRD[82]. A recent nationwide study showed that SB and PPV produced equally good anatomical outcomes[7]. However, only a few studies have focused on comparing PPV with SB in terms of metamorphopsia after RRD repair. Some studies have shown no significant differences in terms of metamorphopsia between PPV and SB[11,17,23].

Despite the global popularity of PPV, the relative simplicity and elegance of PnR remain appealing. In a randomized controlled trial, PnR yielded superior BCVA, less vertical metamorphopsia, and reduced morbidity, compared with PPV. These differences likely have multiple underlying factors, including reduced invasiveness, more rapid surgical procedure, and more natural reattachment of the retina. Additionally, although the primary anatomical success of PnR is lower than the success of PPV, there are no technical difficulties in terms of secondary operations after failed primary PnR; the final overall success rate is similar between PPV and PnR[83]. Brosh *et al*[84] reported that retinal displacement may occur more frequently during PPV (44.4%) than during PnR (7.0%). Notably, displacement is more common during PPV, and the extent of macular displacement is more severe in patients who have undergone PPV. The gas bubble used in PnR provides a smaller contact angle with the retina, imparts less buoyant force to the retina and SRF, and is localized to the vicinity of the retinal break(s); thus, it may reduce the likelihood of stretching and displacement, such that metamorphopsia is less frequent and less severe. A recent randomized controlled trial of RRD repair methods showed that disruption of the EZ and ELM at 12mo postoperatively was more common after PPV than after PnR[85]. Although that trial did not demonstrate a causal relationship between metamorphopsia and disruption of the EZ and ELM, we speculate that PnR is superior to PPV in terms of postoperative metamorphopsia because disruption of the EZ and ELM is a risk factor for metamorphopsia.

METAMORPHOPSIA AND BCVA AFTER RRD REPAIR

The presence of a correlation between metamorphopsia and BCVA remains controversial. Some studies have shown that metamorphopsia is not associated with BCVA after RRD repair[10,12-14,65]. Okuda *et al*[12] showed that BCVA was not significantly correlated with horizontal or vertical metamorphopsia scores in eyes with macula-off RRD or macula-on RRD. Murakami *et al*[10] showed that the postoperative metamorphopsia score was significantly correlated with preoperative BCVA but not with postoperative BCVA. A recent study showed that both preoperative and postoperative BCVA were not correlated with the mean M-CHARTS score at any time point[65]. Although metamorphopsia can be inconvenient for patients, postoperative visual acuity appears not to be significantly affected by metamorphopsia. Improvements in visual acuity and metamorphopsia are based on the recovery of retinal structure, while the resolution of metamorphopsia appears to be associated with microstructural restoration. Notably, the perception of visual distortion is more subjective than BCVA, while the M-CHARTS score is variable among patients; it is thus challenging to determine the relationship between BCVA and metamorphopsia.

Some studies have shown that postoperative BCVA is significantly worse in eyes with metamorphopsia than in eyes without metamorphopsia[15-17,66]. van de Put *et al*[15] and Zhou *et al*[17] reported that eyes without metamorphopsia had significantly better BCVA, compared with eyes that exhibited metamorphopsia. Wang *et al*[16] found that BCVA was significantly better in the non-metamorphopsia group than in the metamorphopsia group. They speculated that metamorphopsia led to greater difficulty in reading letters on the chart. Furthermore, they speculated that macular abnormalities may lead to worse BCVA. It was also speculated that the cause of the relationship between metamorphopsia and BCVA involved EZ disruption, which may also contribute to problems with visual acuity. However, in patients with normal OCT findings, metamorphopsia may be caused by retinal shift, and visual acuity may generally remain unaffected[66].

There are some clear variations between metamorphopsia and BCVA, so we suggest concurrent use of visual acuity and metamorphopsia assessments during postoperative management of patients with RRD for more accurate conclusions concerning postoperative visual function.

CORRELATION BETWEEN METAMORPHOPSIA AND VISION-RELATED QUALITY OF LIFE AFTER RRD REPAIR

The phrase "vision-related quality of life" has been proposed to refer to a patient's emotional, social, physical, and functional well-being, all of which derive from visual improvement or deterioration[86]. The relationship between metamorphopsia and vision-related quality of life has been extensively studied. However, there is minimal literature regarding this relationship after RRD repair.

In eyes that have undergone RRD repair, vision-related quality of life is usually measured using the 25-Item National Eye Institute Visual Function Questionnaire (VFQ-25). The VFQ-25 includes questions regarding general health status and vision-related parameters, including general vision, ocular pain, near activities, distance activities, social functioning, mental health, role difficulties, dependency, driving, color vision, and peripheral vision. A higher VFQ-25 score implies a higher quality of life[87]. Lina *et al*[23] reported that metamorphopsia after RRD repair led to a decrease in the VFQ-25 composite score, while the VFQ-25 composite score was not significantly correlated with BCVA or stereopsis. Another study showed that the VFQ-25 composite score was generally lower in patients with metamorphopsia, although this difference was not statistically significant[15]. Saleh *et al*[13] conducted a questionnaire study that specifically focused on the difficulties caused by metamorphopsia in various indoor and outdoor daily activities[88]. Their study showed limited impacts of metamorphopsia on vision-related quality of life. Only three patients (15%) reported severe effects on their activities of daily living.

ASSESSMENTS OF METAMORPHOPSIA AFTER RRD REPAIR

Considering the research advances with respect to metamorphopsia, there are many different test methods, such as preferential hyperacuity perimetry, shape discrimination hyperacuity test, and new aniseikonia test[89-92]. However, the Amsler grid and M-CHARTS approaches remain the most popular methods for assessment of metamorphopsia after RRD repair because of their low cost, good compliance, simple design, convenient procedure, and lack of additional equipment or training.

Amsler Grid The Amsler grid was first described by Amsler in the mid-20th century[93]. It comprises a 10 cm ×10 cm white square on a black background. The white square is subdivided at 5-mm intervals by vertical and horizontal parallel lines. Each small 5-mm square subtends an angle of l° at 28 cm to 30 cm; the entire grid is thus 20° high and 20° wide (10° on each side of the center). When the grid is introduced into the visual field, its image occupies only a small central area of 10° around the fixation point, but this area is the most important component of the test. During the test, patients must subjectively determine whether lines crossing in the grid are straight, whether the lines are parallel to each other from beginning to end (particularly near the center), and whether the small squares are regular and perfectly equal.

Because of its low cost, ease of use, and lack of need for additional training, the Amsler grid is the method most commonly used for metamorphopsia detection and monitoring. However, it has multiple limitations. First, although a normal contralateral eye is needed to aid in fixing the central point of the grid and applying eccentric vision, some patients have only monocular vision or exhibit abnormal visual function in both eyes. Furthermore, patients with metamorphopsia may describe the lines as small angular irregularities, with larger or smaller undulations. These statements are helpful for determining the presence or absence of metamorphopsia, but they cannot indicate its severity. It would be difficult for patients to draw an image of the lines affected by metamorphopsia, and it may not be useful for home-screening in terms of quantifying the severity of metamorphopsia.

M-CHARTS

When a dotted line is used and the dot interval changes from fine to coarse, metamorphopsia decreases and finally disappears. Based on this phenomenon, the M-CHARTS assessment was developed in 1999 as a new method for evaluating the severity of metamorphopsia[94]. It consists of 19 types of dotted lines with dot intervals at visual angles of 0.2°-2.0°. There are two types of M-CHARTS assessments: type 1 with a single line (for general use), and type 2 with two dotted lines and an intervening fixation point (for patients with central scotoma). Initially, a vertical straight line (0°) is shown to the patient and the patient fixates on a fixation point in the center of the line. If the patient recognizes the straight line as straight, the test is finished and the metamorphopsia score is 0. If the patient recognizes the straight line as an irregular or curved line, then the patient is sequentially shown an array of dotted lines with intervals that change from fine to coarse. When the patient recognizes a dotted line as straight, its visual angle is recorded as the patient's metamorphopsia score. This procedure is repeated to determine the patient's horizontal M-CHARTS score.

Although the M-CHARTS assessment has 19 different lines, it remains uncomplicated. Throughout the test, the patient simply looks at the line, determines whether it is straight, and provides a "yes" or "no" answer without any additional training. In contrast to the Amsler grid, the M-CHARTS assessment does not require aid from the normal contralateral eye and can be used for a wider range of patients. The M-CHARTS assessment reportedly has greater sensitivity than the Amsler

grid in patients with age-related macular degeneration[95-96], but there have been no assessments of differences between Amsler grid and M-CHARTS assessments in patients with metamorphopsia after RRD repair. In such patients, the M-CHARTS assessment appears to be superior because it provides a simple and objective method to quantify the subjective perception of visual distortion.

CONCLUSION

Although previous studies have often used visual acuity as the main indicator of visual function after RRD repair, there is increasing research focus on postoperative metamorphopsia because of improvements in the quantification of metamorphopsia. In this review, we discussed the relationships of metamorphopsia with BCVA and vision-related quality of life. We believe that although some studies have shown a negative correlation between BCVA and metamorphopsia, these are not equivalent parameters; we recommend that BCVA and metamorphopsia are used simultaneously for the assessment of postoperative visual function.

In this review, we summarized studies regarding metamorphopsia after RRD surgery and categorized risk factors into macular involvement, retinal shift, ORFs, SRF, secondary ERM, outer retinal layer damage, and surgical approach. The current literature is very dependent on the rapid development of examination methods, such as OCT. Although some risk factors are controversial, the mechanisms that underlie their relationships with metamorphopsia remain unclear. Further upgrades of auxiliary inspection methods and more accurate microstructural assessments will soon clarify these relationships.

This review did not discuss the treatment of metamorphopsia after RRD repair because there remain no good methods to address postoperative metamorphopsia. Most treatments cannot progress beyond the management of negative visual sensations, through methods such as occlusion therapy and aniseikonia-correcting spectacles. Currently, the main treatment approach involves RRD prevention and the management of risk factors that can lead to postoperative metamorphopsia after RRD repair. Management approaches include performing vitrectomy with silicone oil tamponade instead of gas tamponade to prevent retinal shift, reducing the amount of SRF by using perfluorocarbon liquid, and performing internal limiting membrane peeling to avoid secondary ERM formation. Additionally, PnR appears to be superior for the prevention of postoperative metamorphopsia, compared with PPV or SB. Notably, these options focus specifically on metamorphopsia, while clinical judgments must be made based on a patient's actual situation, rather than their risk of metamorphopsia alone. Because the incidence of postoperative metamorphopsia is high (24%–88.6%), we expect that additional research concerning metamorphopsia treatment will be conducted to improve the quality of life in affected patients.

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