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Causes of Diplopia in Patients with Epiretinal Membranes

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Abstract

Purpose—To describe the causes of diplopia in patients with an epiretinal membrane (ERM) and presenting diplopia.

Design—Retrospective observational case series

Methods—We reviewed patients diagnosed with an ERM, who had been seen by both retinal and strabismus specialists in a tertiary medical center. Data recorded: orthoptic evaluation, retinal misregistration (optotype-frame test, and synoptophore central peripheral superimposition slides at 5 and 10 degrees), and cause of any diplopia (retinal misregistration vs strabismus vs optical/refractive error). We defined central-peripheral rivalry-type diplopia as presenting symptomatic diplopia with evidence of retinal misregistration, and where other causes did not fully explain diplopia. The frequency of each cause of diplopia in patients with ERM was determined.

Results—Of 50 patients with ERM, 25 had symptomatic diplopia and 25 had no diplopia. 11 (44%) of 25 diplopic patients had retinal misregistration as the sole cause (central-peripheral rivalry-type diplopia), 7 (28%) strabismus (1 of 7 initially appeared to have central-peripheral rivalry-type diplopia), 1 (4%) optical/refractive error (monocular diplopia), 2 (8%) mixed retinal misregistration (central-peripheral rivalry-type diplopia) and strabismus and for 4 (16%) diplopia cause was indeterminate. Unexpectedly, 15 (60%) of 25 patients without diplopia had evidence of retinal misregistration.

Conclusions—Patients with ERM and presenting diplopia may have one of several causes of diplopia, most commonly retinal misregistration (central-peripheral rivalry-type diplopia). Nevertheless, diplopic patients with retinal misregistration may also have treatable strabismus or optical/refractive error as the primary barrier to single vision and therefore many potential barriers to single vision should be considered.

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Introduction

An epiretinal membrane (ERM) is a thin gliotic tissue proliferation overlying the retina that may contract to cause dragging and distortion of the macula. An ERM may cause abnormalities of the retinal mosaic leading to aniseikonia,¹⁻⁶ metamorphopsia,^{3,6-11} and decreased visual acuity.^{1,12} In addition, binocular misregistration of the retinal mosaics may cause binocular diplopia,^{1,5,9} which we term central-peripheral rivalry-type diplopia, also known as dragged-fovea diplopia,³ or macular diplopia¹³ and may manifest as foveal-peripheral rivalry on synoptophore testing.⁷ Nevertheless, there are many potential causes of diplopia in adults¹⁴ such that in the patient with an ERM, diplopia may, or may not, be specifically caused by the ERM. The purpose of this study was to describe the specific causes of diplopia in patients presenting with both an ERM and symptomatic diplopia.

Methods

This study was designed as a retrospective observational case series. Institutional Review Board approval was obtained from the Institutional Review Board at the Mayo Clinic, Rochester, Minnesota for data collection and analysis. All procedures and data collection were conducted in a manner compliant with the Health Insurance Portability and Accountability Act. All research procedures adhered to the tenets of the Declaration of Helsinki.

Patients

We retrospectively reviewed the medical records of ERM patients seen by both retinal (RI) and strabismus (JMH) specialists. Retina and strabismus examinations were required to be within 6 months of each other. Optical coherence tomography (OCT) was performed by the Heidelberg Spectralis SD-OCT (Heidelberg Engineering, Inc., Franklin, Massachusetts) at the time of the retina examination, and OCT images reviewed by a retina specialist (RI, masked to the clinical findings), to confirm the presence of a visually significant ERM in either or both eyes. Patients with coexisting ocular conditions, including other retinal diseases, were not excluded if OCT review confirmed the ERM to be visually significant, but we did exclude patients with other retinal conditions, if they were felt to be the primary cause of visual symptoms. Patients who had undergone prior ERM peeling procedure were also excluded.

Clinical testing

Evaluation of diplopia

A standardized diplopia questionnaire¹⁵ was used allowing each patient to rate how frequently they experienced diplopia over the previous week. If diplopia was recorded as “rarely,” “sometimes,” “often,” or “always,” for straight ahead distance or reading positions, or the patient was wearing prism correction for diplopia, or diplopia was recorded by history, the patient was classified as having diplopia. If diplopia was recorded as “never” for straight ahead distance and reading positions by diplopia questionnaire, and there was no diplopia by history, then the patient was classified as having no diplopia.

Orthoptic evaluation

Ocular alignment was evaluated using the simultaneous prism cover test (SPCT) and prism and alternate cover test (PACT) at distance (3 meters) and near (1/3 meter) in habitual refractive correction. We evaluated each patient for evidence of binocular retinal misregistration. We were unable to perform the previously described “lights on – lights off” test for retinal misregistration, because we could not achieve sufficient room darkness.³ Instead we devised an optotype-frame test to determine whether a patient had retinal misregistration.

Optotype-frame test

For the optotype-frame test the patient was asked to fixate an isolated (uncrowded) Snellenoptotype on an illuminated monitor at 3 meters (Figure 1 Top left) and report first whether the letter itself was single or double, and then, while maintaining fixation on the letter, whether the frame of the monitor was single or double. The distance from the top of a 20/40 optotype to the inside edge of the 22-inch screen (Figure 1 Top left) subtended 2.9 degrees at 3 meters. The presence of retinal misregistration was defined as the patient reporting a double letter while seeing a single frame (Figure 1 Top right) or a single letter with a double frame (Figure 1 Bottom left). A double optotype with a double frame was considered consistent with binocular diplopia of strabismic type (Figure 1 Bottom right). For patients with 20/40 or better visual acuity in their worse-seeing eye, a 20/40-sized optotype was used for testing. If visual acuity was worse than 20/40 in the worse-seeing eye the optotype size used ranged from 20/40 (because resolution of the letter is not necessary to recognize diplopia) to 20/80. Optotype-frame testing was required for inclusion in this study.

Synoptophore superimposition test

We also assessed retinal misregistration using customized synoptophore slides. First described by Burgess et al,⁷ one eye views five boxes (Figure 2 Top) and the other eye views five X's positioned to superimpose the boxes (Figure 2 Middle). The patient was asked to align the central X into the central box, and then report whether the peripheral X's were simultaneously aligned. This testing was performed with targets subtending 5 degrees and 10 degrees. Retinal misregistration was recorded as present if one or more of the peripheral X's did not align in the corresponding peripheral box while the central X remained aligned (Figure 2 Bottom), using either the 5 or 10 degree targets.

Analysis

Specific causes of diplopia in patients with epiretinal membrane

The primary clinical examination and subsequent examinations were reviewed by a strabismologist (JMH) and orthoptist (SRH), to determine the causes(s) of diplopia for each patient. The following definitions were applied:

Retinal misregistration alone (central-peripheral rivalry-type diplopia)—

Symptomatic diplopia with evidence of retinal misregistration, either by optotype-frame test or by synoptophore, and no other barriers to single vision, i.e., no strabismus or monocular diplopia due to optical/refractive causes. Small angle vertical deviations (≤ 5 PD

bysimultaneous prism and cover test [SPCT]) were allowed without assigning strabismus as a cause, because such deviations are commonly associated with central-peripheral rivalry-type diplopia.^{3,7,16}

Strabismus alone—Symptomatic diplopia associated with a horizontal deviation greater than 0 by SPCT at distance or near, decompensating horizontal strabismus, or vertical deviation greater than 5 PD by SPCT at distance or near. If tests for retinal misregistration were negative, small vertical deviations (5 PD or less by SPCT at distance or near) were also considered causative of strabismic diplopia. If tests for retinal misregistration were positive, a diagnosis of strabismic diplopia alone as the barrier to single vision was confirmed only if the diplopia resolved with ground-in prism or surgery, because previous studies report poor success with prism correction for diplopia associated with maculopathies.^{1,7,16,17} We did not consider improvement with a temporary Fresnel prism as confirmed strabismic diplopia, since Fresnel prisms may enable suppression by degrading image quality.

Optical / refractive error alone (monocular diplopia)—Clinical documentation of monocular diplopia caused by optical abnormalities or refractive error.

Mixed retinal misregistration (central-peripheral rivalry-type diplopia) and strabismus—Evidence of both true strabismus and retinal misregistration on clinical testing with only partial improvement of diplopia, (not complete resolution), following treatment with ground-in or Fresnel prism, or with surgery. We allowed Fresnel prism treatment when defining mixed-type diplopia, because such patients were experiencing persistent diplopia (indicating no occlusive effect of the Fresnel).

Indeterminate—True strabismus (horizontal SPCT >0 PD, or decompensating latent horizontal or vertical strabismus >5 PD on PACT) or monocular diplopia present, and evidence of retinal misregistration, but insufficient post-treatment follow-up (ground-in prism, strabismus surgery, improved refractive correction) and therefore no opportunity to determine whether diplopia resolved (true strabismus or optical/refractive alone), or persisted (combination central-peripheral rivalry-type diplopia). A trial of Fresnel prism, with complete resolution of diplopia, was not sufficient to assign diplopia cause as strabismic (and not “indeterminate”) because of the potential occlusion effect.

Results

Patient demographics

Of 50 patients with ERM, 25 had symptomatic diplopia and 25 had no diplopia. Twenty-nine (58%) were male and 50 (100%) reported their race as white. Median age was 70 (range 51-94) years. Twenty-six (52%) of 50 patients had another retinal condition or history of a retinal condition (Table 1), 12 (46%) of 25 with diplopia and 14 (56%) of 25 without diplopia.

Causes of diplopia

Twenty-five (50%) patients had an ERM and symptomatic diplopia: 11 (44%) of 25 had diplopia caused by retinal misregistration alone (central-peripheral rivalry-type diplopia). On the optotype-frame test 10 (91%) of 11 had a double optotype, single frame (Figure 1 Top right) and one (9%) had a single optotype with a double frame (Figure 1 Bottom left). On the synoptophore, 5 of 7 (71%) had evidence of retinal misregistration on the 5-degree targets and 6 of 6 (100%) on the 10-degree targets. Seven (28%) of 25 patients had diplopia caused by strabismus alone, 1 (4%) had diplopia caused by optical / refractive error alone (monocular diplopia), 2 (8%) had mixed retinal misregistration (central-peripheral rivalry-type) and strabismus, and for 4 (16%) the cause of diplopia was indeterminate (Table 2). Of the seven patients classified as having strabismus alone as the cause of diplopia, six (86%) clearly had true strabismus from the outset, and one (14%) was initially thought to have retinal misregistration causing central-peripheral rivalry-type diplopia, but was subsequently determined to have true strabismus alone (See case example 2, below).

Findings in patients with diplopia

Three case examples help clarify differences between various causes.

Case 1 – Retinal misregistration alone (central-peripheral rivalry-type diplopia)—69-year-old male with diplopia, blurred vision, distortion, and a diagnosis of right ERM. Corrected visual acuity was 20/25 right eye and 20/20 left eye. There was no manifest or latent strabismus at distance, and 6 PD exophoria at near. Double Maddox rod testing showed 2 degrees of excyclotorsion, and the optotype-frame test showed retinal misregistration. Single vision could be recovered with a blink but then the double would recur. Trials of vertical prism in office achieved improvement lasting for only a few seconds. There was 3% aniseikonia vertically and horizontally using the Awaya New Aniseikonia Test.¹⁸ Based on refractive error and spectacle correction, there was no clinically significant anisometropia. The presence of retinal misregistration findings combined with the absence of other barriers to single vision led to classification as retinal misregistration (central-peripheral rivalry-type diplopia) alone.

Case 2 - Strabismus (originally thought to be central-peripheral rivalry-type diplopia)—73-year-old male with a history of diplopia and distortion. Diagnoses (made by previous providers) were fourth nerve palsy and left ERM, and previous trials of prism had been unsuccessful. On clinical examination corrected visual acuity was 20/20 right eye and 20/30 left eye. There was no manifest strabismus by SPCT, and PACT showed 2 PD right hyperphoria at distance and 6 PD exophoria, 1 PD right hyperphoria at near. Double Maddox rod testing revealed 8 degrees of excyclotorsion, and the optotype-frame test showed evidence of retinal misregistration with a double optotype, single frame (Figure 1 Top right). There was also 10% aniseikonia using the Awaya New Aniseikonia Test¹⁸ and metamorphopsia using the Amsler grid. There was no clinically significant anisometropia. When fusion potential was assessed on the synoptophore, the patient needed 5 degrees of excyclotorsion correction to fuse (using house targets). At this point, potential barriers to single vision were identified as: (1) vertical strabismus (that would be amenable to optimal prism correction), (2) torsional strabismus (that would require strabismus surgery), (3)

retinal misregistration (that would likely not be amenable to either prism or surgery), and (4) aniseikonia. A further trial of prism correction initially appeared successful but then diplopia recurred, suggesting vertical strabismus was not the primary cause of diplopia. However, synoptophore testing confirmed excyclotorsion as a barrier to fusion, and surgical correction was undertaken by performing a Harada-Ito procedure. Two months postoperatively the patient reported a 99% improvement in diplopia. Of note, findings of retinal misregistration, aniseikonia and metamorphopsia continued to be present on subsequent clinical examinations, but given the significant improvement in diplopia following strabismus surgery the cause was classified as strabismus alone.

Case 3 – Mixed retinal misregistration (central-peripheral rivalry type) and strabismus—67-year-old female with an ERM and a 3-year history of horizontal and vertical diplopia, corrected with base-out prism in her current glasses. Additional base-out Fresnel prism had been added which worked initially but then diplopia recurred. She had a negative workup for myasthenia gravis. On examination corrected visual acuity was 20/20 in both eyes with 4 PD esotropia by SPCT, 8 PD by PACT at distance, and 2 PD exophoria at near. Double Maddox rod testing showed 1 degree excyclotorsion, and both the optotype-frame test and synoptophore test were positive for retinal misregistration. The Awaya New Aniseikonia Test¹⁸ showed 10% aniseikonia but there was no metamorphopsia evident on Amsler grid testing. There was no clinically significant anisometropia. Although the patient's diplopia was improved with base-out prism (versus without), subsequent trials of additional prism and Bangerter filter over the next three examinations failed to resolve residual, primarily vertical diplopia, and therefore the cause was classified as mixed retinal misregistration (central-peripheral rivalry-type diplopia) and strabismus with the strabismic component being divergence insufficiency type esotropia.

Findings in patients without diplopia

Interestingly, 15 (60%) of 25 patients without diplopia had evidence of retinal misregistration: 6 (24%) of 25 on optotype-frame testing, 11 (85%) of 13 on synoptophore 5-degree targets and 10 (91%) of 11 on synoptophore 10-degree targets (for example Figure 2 Bottom left). Unfortunately in this retrospective study, not all tests were performed in all patients. The unexpected finding of retinal misregistration in non-diplopic patients with ERM was discovered because we decided early in our assessment of patients with ERM to perform optotype-frame testing regardless of diplopia status.

Discussion

We found retinal misregistration was the most common cause of diplopia (central-peripheral rivalry-type) in patients with ERM. Nevertheless, approximately one-third had other ocular conditions (strabismus or optical / refractive anomalies) as a cause of their diplopia. Of interest, we found that approximately half of patients with an ERM but without any symptoms of diplopia, demonstrated retinal misregistration on clinical testing.

We are unaware of previous studies describing specific causes of diplopia in patients with ERM. Our findings demonstrate that some patients may also have other, more treatable causes of diplopia (strabismus or optical / refractive error) coexistent with an ERM and

retinal misregistration. In previous studies of diplopia in the context of an ERM (or other maculopathies), treatment with prism has been concluded to be ineffective^{3,7,13,19} and partial occlusion is usually recommended.^{3,16,20} While we agree that for many patients occlusion may be the only satisfactory treatment, it is important to recognize that a small sub-group of patients may in fact have true strabismus and respond well to prism and/or strabismus surgery. Burgess et al⁷ found that one of 11 patients responded well to prism therapy, and Bixenman & Joffe¹⁹ found one of 5 patients experienced a dramatic improvement with prism. We suggest that patients with clinical evidence of an ERM and retinal misregistration should not be automatically prescribed partial occlusion treatment (for example Bangerter filter or Scotch Satin tape³), but be carefully evaluated for other barriers to single vision, such as true strabismus.

Several of our cases illustrated the need for extensive clinical testing, before the exact cause(s) of diplopia could be determined. Unlike most diplopic patients where the barrier to single vision is clearly strabismus alone, patients with ERM often present a less clear clinical picture. For these patients, possible barriers to single vision are retinal misregistration due to macular disease, true strabismus, or monocular diplopia due to optical distortion, or a combination. We found that it was often not until optimal prism correction had been tried, or strabismus surgery performed (case 2 above), and significant improvement in diplopia demonstrated, that strabismus could be confirmed as a cause of diplopia.

A surprising finding in the present study was that half of the patients without symptomatic diplopia had retinal misregistration on clinical testing (optotype-frame test or synoptophore superimposition slides). Previous studies of retinal misregistration and diplopia in the context of retinal disease, have focused on patients presenting with symptomatic diplopia,³ and therefore have not addressed whether or not retinal misregistration may occur in the absence of symptomatic diplopia. Data from our present study suggest not only that retinal misregistration is present more frequently in patients with ERM than may have been previously recognized, but importantly that not every case of such misregistration leads to symptomatic diplopia. At an anatomical level, many patients with an ERM have disruption of the retinal mosaics leading to some degree of retinal misregistration. In some of these patients this retinal misregistration is clinically detectable using the optotype-frame test and/or synoptophore. But while retinal misregistration constitutes a potential barrier to single vision by disrupting central fusion, only some patients with detectable retinal misregistration will have symptomatic central-peripheral rivalry-type diplopia. One possible explanation for a lack of symptomatic diplopia in some patients with retinal misregistration, but symptomatic diplopia in others is that there is individual variability in the amount of retinal misregistration that can be tolerated. It is also possible that a combination of factors is needed before the patient experiences symptomatic diplopia.

There are some limitations to our study. Due to the retrospective design we did not have complete data on every clinical parameter of interest, particularly synoptophore testing. Because a large proportion of patients tested on the synoptophore showed retinal misregistration, it is probable that the proportion with retinal misregistration is actually higher than we report. In addition, we used a novel test of retinal misregistration (the optotype-frame test) because we were unable to perform the previously reported lights-on,

lights-off test.³ We cannot be sure whether our optotype-frame test is directly comparable to the lights-on, lights-off test. We assessed patients referred to a strabismus practice and therefore the frequency of strabismus as a contributing cause of diplopia could be higher than would be seen in a retina practice. Nevertheless, our study highlights the importance of recognizing coexistent strabismus in patients with ERM.

Retinal misregistration was found to be the most frequent cause of diplopia (central-peripheral rivalry-type) in patients with ERM and presenting diplopia, but there are several possible causes of diplopia in patients with ERM, and determining the precise cause(s) may require a prolonged process of extensive clinical testing and several clinical examinations. Such rigorous evaluation helps identify a sub-group of patients who in fact have coexistent true strabismus that can be successfully treated with prism or even strabismus surgery. Our novel finding that retinal misregistration can be present without experiencing symptomatic diplopia suggests that the presence of retinal misregistration may be necessary but not sufficient for the development of central-peripheral rivalry-type diplopia in patients with ERM.

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Figure 1.

The Optotype-Frame Test, displaying a single, uncrowded Snellen optotype on a computer monitor. The optotype falls on central retina while the monitor frame falls on peripheral retina. Possible outcomes of the test include (Top left) single optotype with single frame, indicating no diplopia, (Top right) double optotype with a single frame, indicating retinal misregistration, (Bottom left) single optotype with a double frame, also indicating retinal misregistration, and (Bottom right) double optotype with a double screen, consistent with binocular diplopia caused by strabismus.

Figure 2.

Synoptophore slides to evaluate retinal misregistration (10-degree targets pictured). One eye views five boxes, with four boxes oriented at the ends of a plus sign (Top). The other eye views 5 x's positioned to exactly superimpose on the five boxes (Middle). When the 4 peripheral x's superimpose the 4 peripheral boxes, no retinal misregistration is present. When one of more of the peripheral x's do not align with the boxes (Bottom), retinal misregistration is present.

Table 1
Presence or History of Coexisting Retinal Conditions

Retinal Condition	N (%) ^a
Macular hole (including lamellar hole, stage 1a hole, pseudohole)	10 (38%)
Previous retinal tear	6 (23%)
Dry age-related macular degeneration	4 (15%)
Previous retinal detachment	3 (12%)
Choroidal nevus	2 (8%)
Lattice degeneration	2 (8%)
Peripheral atrophic hole	1 (4%)
Hypertensive retinal changes	1 (4%)
Irvine-Gass syndrome	1 (4%)
Proliferative diabetic retinopathy	1 (4%)
Retinal vein occlusion	1 (4%)

^aDoes not sum to 100% because some patients had more than one coexisting condition

Table 2
Causes of Diplopia in the 25 Epiretinal Membrane Patients with Diplopia

Causes of Diplopia	N (%)
Retinal misregistration (central-peripheral rivalry type diplopia)	11 (44%)
Strabismus	7 (28%)
Mixed retinal misregistration (central-peripheral rivalry type diplopia) and strabismus	2 (8%)
Optical / refractive error (monocular diplopia)	1 (4%)
Indeterminate	4 (16%)