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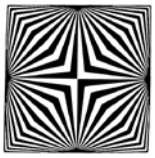

Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration

AMERICAN ACADEMY OF OPHTHALMOLOGY
The Eye M.D. Association

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As a service to its members and the public, the American Academy of Ophthalmology has developed a series of guidelines called Preferred Practice Patterns that **identify characteristics and components of quality eye care.** (See Appendix 1.)

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TABLE OF CONTENTS

INTRODUCTION 2
ORIENTATION 3
Entity 3
Disease Definition 3
Patient Population 3
Activity 3
Purpose 3
Goals 3
BACKGROUND 4
Natural History of Precursors to Rhegmatogenous Retinal Detachment 4
Posterior Vitreous Detachment 4
Symptomatic Retinal Breaks 4
Asymptomatic Retinal Breaks 4
Lattice Degeneration 5
Epidemiology of Rhegmatogenous Retinal Detachment 5
Risk Factors for Rhegmatogenous Retinal Detachment 5
Myopia 5
Lattice Degeneration 5
Cataract Surgery 5
Trauma 6
Rhegmatogenous Retinal Detachment in the Fellow Eye 6
Other Risk Factors 6
PREVENTION AND EARLY DETECTION 6
CARE PROCESS 6
Patient Outcome Criteria 6
Diagnosis 7
History 7
Examination 7
Diagnostic Tests 7
Treatment 7
Complications of Treatment 8
Follow-up 8
History 9
Examination 9
Provider 9
Counseling/Referral 10
APPENDIX 1. QUALITY OF OPHTHALMIC CARE CORE CRITERIA 10
APPENDIX 2. SUMMARY OF MAJOR RECOMMENDATIONS FOR CARE 12
APPENDIX 3. VITREOUS HEMORRHAGE 14
GLOSSARY 14
RELATED ACADEMY MATERIALS 16
REFERENCES 16

INTRODUCTION

The Preferred Practice Pattern® (PPP) guidelines have been written on the basis of three principles.

- Each Preferred Practice Pattern should be clinically relevant and specific enough to provide useful information to practitioners.
- Each recommendation that is made should be given an explicit rating that shows its importance to the care process.
- Each recommendation should also be given an explicit rating that shows the strength of evidence that supports the recommendation and reflects the best evidence available.

In the process of revising this document, a detailed literature search of articles in the English language was conducted on the subject of posterior vitreous detachment, retinal breaks, and lattice degeneration for the years 2002 to 2007. The results were reviewed by the Retina Panel and used to prepare the recommendations, which they rated in two ways. The panel first rated each recommendation according to its importance to the care process. This "importance to the care process" rating represents care that the panel thought would improve the quality of the patient's care in a meaningful way. The ratings of importance are divided into three levels.

- Level A, defined as most important
- Level B, defined as moderately important
- Level C, defined as relevant but not critical

The panel also rated each recommendation on the strength of evidence in the available literature to support the recommendation made. The "ratings of strength of evidence" also are divided into three levels.

- Level I includes evidence obtained from at least one properly conducted, well-designed, randomized controlled trial. It could include meta-analyses of randomized controlled trials.
- Level II includes evidence obtained from the following:
  - Well-designed controlled trials without randomization
  - Well-designed cohort or case-control analytic studies, preferably from more than one center
  - Multiple-time series with or without the intervention
- Level III includes evidence obtained from one of the following:
  - Descriptive studies
  - Case reports
  - Reports of expert committees/organizations (e.g., PPP panel consensus with external peer review)

The evidence cited is that which supports the value of the recommendation as something that should be performed to improve the quality of care. The panel believes that it is important to make available the strength of the evidence underlying the recommendation. In this way, readers can appreciate the degree of importance the committee attached to each recommendation and they can understand what type of evidence supports the recommendation.

The ratings of importance and the ratings of strength of evidence are given in bracketed superscripts after each recommendation. For instance, "[A:II]" indicates a recommendation with high importance to clinical care [A], supported by sufficiently rigorous published evidence, though not by a randomized controlled trial [II].

The sections entitled Orientation and Background do not include recommendations; rather they are designed to educate and provide summary background information and rationale for the recommendations that are presented in the Care Process section. A summary of the major recommendations for care is included in Appendix 2.

ORIENTATION

ENTITY

Precursors to rhegmatogenous retinal detachment (ICD-9 #361.00) and related entities with the following ICD-9 classifications:

- Posterior vitreous detachment (379.21)
- Retinal break without detachment (361.30)
- Multiple retinal breaks without detachment (361.33)
- Horseshoe tear without detachment (361.32)
- Operculated break without detachment (361.32)
- Round hole without detachment (361.31)
- Retinal dialysis (361.04)
- Lattice degeneration of the retina (362.63)

DISEASE DEFINITION

Posterior vitreous detachment (PVD) is a separation of the posterior vitreous cortex from the internal limiting lamina of the retina. It often precedes rhegmatogenous retinal detachment (RRD) (see Glossary). Vitreous traction at sites of significant vitreoretinal adhesion is responsible for most retinal breaks that lead to retinal detachment.

Lattice degeneration is a vitreoretinal degenerative process with visible lesions that predispose to retinal tears and detachment (see Glossary).

PATIENT POPULATION

The patient population consists of individuals who present with symptoms or signs suggestive of PVD, retinal breaks, vitreous hemorrhage, or retinal detachment and asymptomatic patients who have an increased risk of retinal detachment.

ACTIVITY

Evaluation and management of patients with disorders associated with an increased risk of retinal detachment.

PURPOSE

The purpose of evaluating, diagnosing, and managing patients with disorders predisposing to RRD is to prevent visual loss and functional impairment related to retinal detachment and to maintain quality of life.

GOALS

- Identify patients at risk of RRD
- Examine patients with symptoms of acute PVD to detect and treat significant retinal breaks
- Manage patients at high risk of developing retinal detachment
- Educate high-risk patients about symptoms of PVD, retinal breaks, and retinal detachments and about the need for periodic follow-up

BACKGROUND

NATURAL HISTORY OF PRECURSORS TO RHEGMATOGENOUS RETINAL DETACHMENT

Precursors to retinal detachments are PVD, symptomatic retinal breaks, asymptomatic retinal breaks, lattice degeneration, and cystic and zonular traction retinal tufts. Because spontaneous reattachment is exceedingly rare, nearly all patients with a symptomatic RRD will progressively lose vision unless the detachment is repaired. Currently, more than 95% of RRDs can be successfully repaired, although more than one procedure may be required. The treatment of breaks before a significant detachment has occurred usually prevents progression, is uncomplicated and results in excellent vision. The early diagnosis of a retinal detachment is also important because the rate of successful reattachment is higher and the visual results are better if detachment spares the macula.[1,2] Successful treatment allows patients to maintain their abilities to read, work, drive, care for themselves, and enjoy a better quality of life.[3]

Posterior Vitreous Detachment

Posterior vitreous detachment is the cause of many retinal breaks that may then lead to retinal detachments. The symptoms of PVD include light flashes and floaters, and patients with such symptoms are at high risk of retinal detachment.[4-8] The light flashes are typically best seen in the dark and are caused by vitreous traction on the peripheral retina. The floaters may be due to blood, condensations of vitreous collagen, or epipapillary glial tissue torn from the optic nerve head or the area adjacent to the optic nerve head. Approximately 20% of patients with acute symptoms of PVD have a retinal tear at the time of the initial examination,[1-3,6,9] and there is a direct correlation between the amount of vitreous hemorrhage and the likelihood of a retinal tear.[10] (See Appendix 3.) Patients with acute PVD who have no retinal breaks on presentation have a 2% to 5% chance of developing them in the weeks that follow.[7,9,11] Approximately 80% of patients who have no breaks on presentation, but then develop breaks later, have at least one of the following characteristics: pigmented cells or hemorrhage in the vitreous or retina at the initial evaluation, or new symptoms that prompted a return visit to the ophthalmologist.[9]

Symptomatic Retinal Breaks

A symptomatic retinal break is defined as one caused by vitreoretinal traction in a patient with a new PVD or a break associated with a significant increase in flashes and floaters. At least one-half of untreated symptomatic retinal breaks with persistent vitreoretinal traction (horseshoe or flap tears) will cause a clinical retinal detachment unless treatment is applied.[12-14] Prompt creation of a chorioretinal adhesion around these symptomatic tears reduces the chances of retinal detachment to less than 5%.[12,13,15-18] Traumatic dialyses and tears along the vitreous base are managed similarly to symptomatic tears. Symptomatic operculated tears have not been reported to progress to retinal detachment unless the vitreous remains adherent to blood vessels in the area of the break.[13,14] Traumatic dialyses and tears along the vitreous base are managed similarly to symptomatic tears.

Asymptomatic Retinal Breaks

Asymptomatic operculated holes and atrophic round holes very rarely lead to retinal detachment. Byer followed 46 eyes with asymptomatic operculated tears over an average of 11 years.[19] Davis followed 28 eyes for up to 5 years, and 80% of the eyes had a retinal detachment in the fellow eye.[14] None of the combined 74 eyes progressed to retinal detachment over the follow-up period.

Eyes with signs and symptoms of acute PVD may have atrophic retinal breaks whose characteristics suggest that they are unrelated to vitreoretinal traction. These breaks are considered to be pre-existing and not symptomatic. Many ophthalmologists treat these breaks, although the literature provides no guidance.[20] Randomized clinical trials are not available for guidance, so no evidence of a benefit of prophylactic therapy has been proven.[20]

About 5% of eyes with asymptomatic horseshoe tears progress to retinal detachment.[19,21,22] Horseshoe tears discovered in asymptomatic fellow eyes are less likely than symptomatic horseshoe tears to lead to retinal detachment.

Lattice Degeneration

Lattice degeneration can cause RRD by two mechanisms, including either round holes without PVD or tractional tears associated with PVD. Younger myopic patients who have lattice degeneration with round holes need regular follow-up visits, because they can develop small, localized retinal detachments, which occasionally slowly enlarge to become clinical retinal detachments. Treatment should be considered if the detachments are documented to increase in size.[21,23]

Generally, however, atrophic round holes within lattice lesions and minimal subretinal fluid do not require treatment. Byer studied 423 eyes with lattice degeneration in 276 patients over a period averaging almost 11 years.[23] Of these, 150 eyes (35%) had atrophic holes in the lattice, and 10 of these 150 had subretinal fluid extending more than one disc diameter from the break. Six other eyes developed new subclinical retinal detachments during follow-up. Clinical retinal detachments developed in three of the 423 eyes.[23] Two were due to round retinal holes in lattice lesions of patients in their mid-twenties and one was due to a symptomatic tractional tear. These data indicate that patients with lattice degeneration with or without round holes are not at significant risk of subsequent retinal detachment without a previous retinal detachment in either eye.

Retinal detachment usually occurs in eyes with lattice degeneration when a PVD causes a horseshoe tear, and such tears should be treated.[21,23]

Folk et al retrospectively studied 388 consecutive patients with lattice degeneration in both eyes who had phakic retinal detachment as a result of the lattice degeneration in one eye.[24] In the second eye all areas of lattice degeneration were prophylactically treated by laser photocoagulation or cryotherapy in 237 eyes and 151 eyes received no treatment. During a mean follow-up period of 7.9 years, retinal detachment occurred in three treated eyes (1.8%) and in nine untreated eyes (5.1%). The clinical significance of this retrospective study of variable follow-up is uncertain.

EPIDEMIOLOGY OF RHEGMATOGENOUS RETINAL DETACHMENT

The annual incidence of RRD is approximately 10 to 15 per 100,000 persons.[25,26] Of these cases, approximately 20% have had cataract surgery and 10% have had ocular trauma.[27-29] In one study conducted from 1976 to 1995, the 10-year risk of RRD after cataract surgery was about six times higher than would be expected in persons who have not had cataract surgery.[29]

RISK FACTORS FOR RHEGMATOGENOUS RETINAL DETACHMENT

Aside from retinal breaks, risk factors for RRD include myopia, lattice degeneration, cataract surgery, trauma, a history of RRD in the other eye, or a strong family history of retinal detachments. Combinations of these factors in a single eye increase the risk.

Myopia

More than half of nontraumatic RRD occurs in myopic eyes.[30] As axial length increases, so does the risk of RRD. Individuals with low myopia (1 to 3 diopters) have a fourfold risk of RRD and higher myopes (>3 diopters) have a tenfold risk compared with nonmyopic individuals.[30]

Lattice Degeneration

Lattice degeneration is present in 6% to 8% of the population and increases the risk of retinal detachment.[23,31] Approximately 20% to 30% of patients with RRD have lattice degeneration.[23]

Cataract Surgery

The overall risk of RRD after cataract surgery is approximately 1%.[32-35] The following conditions have been reported to increase the risk of retinal detachment after cataract surgery: axial myopia, pre-existing vitreoretinal disease, male gender, younger age, vitreous prolapse into the anterior

chamber, vitreous loss, and spontaneous extension of the capsulotomy at the time of surgery.[35-37] A case-control study found that, in the absence of a posterior capsular tear at the time of cataract surgery, subsequent neodymium: yttrium-aluminum-garnet laser (Nd:YAG) capsulotomy did not increase the risk of retinal detachment.[38] However, other studies do suggest an association between Nd:YAG capsulotomy and RRD, especially in myopic patients.[39-42]

Trauma

Patients with blunt or penetrating ocular injuries that have altered the structure of the vitreous or retina are at increased risk of RRD.[43] Vitreoretinal interface changes caused by trauma may be detected at the time of injury or years later.

Rhegmatogenous Retinal Detachment in the Fellow Eye

Patients with a history of nontraumatic detachment in one eye have about a 10% increased risk of developing RRD in the fellow eye, because pathologic vitreoretinal changes are frequently bilateral.[21,24,26,29,44] The fellow eye in a patient with pseudophakic retinal detachment is also at higher risk of developing a retinal detachment, whether the fellow eye is phakic or pseudophakic. Phakic fellow eyes in patients with pseudophakic retinal detachment have about a 7% risk of RRD, indicating that the entire risk of developing RRD cannot be attributed to cataract surgery alone.[45]

Other Risk Factors

Other risk factors that have been reported include prior retinopathy of prematurity[46] and Stickler syndrome.[47,48]

Despite case reports of retinal detachment in patients who have had keratorefractive surgery, large studies have not shown an increased risk in patients with similar refractive errors.[49,50] Retinal detachment following refractive lens exchange in patients with high myopia has been described to occur in 2% to 8% of patients.[51] Phakic intraocular lenses have not been associated with increased risk of retinal detachment compared with other intraocular interventions in highly myopic patients.[50,52,53]

PREVENTION AND EARLY DETECTION

There are no effective methods of preventing the vitreous changes that lead to RRD. If factors associated with an increased risk of retinal detachment are discovered during a routine eye examination in an asymptomatic patient, a peripheral fundus examination is advisable.[A:III] Patients at high risk should also be educated about the symptoms of PVD and retinal detachment as well as about the value of periodic follow-up examinations.[8] [A:II]

CARE PROCESS

PATIENT OUTCOME CRITERIA

In general, outcome criteria include the following:

- Identification of patients at risk
- Prevention of visual loss and functional impairment
- Maintenance of quality of life

DIAGNOSIS

The initial evaluation of a patient with risk factors or symptoms includes all features of the comprehensive adult medical eye evaluation,[54] with particular attention to those aspects relevant to PVD, retinal breaks, and lattice degeneration.

History

A patient history should include the following elements:

- Symptoms of PVD[4-8 [A:I]]
- Family history[47,48 [A:II]]
- Prior eye trauma[43 [A:III]]
- Myopia[30,55 [A:II]]
- History of ocular surgery, including refractive lens exchange and cataract surgery[27-29,56-58 [A:II]]

Examination

The eye examination should include the following elements:

- Examination of the vitreous for hemorrhage, detachment, and pigmented cells[4-9,59 [A:II]]
- Peripheral fundus examination with scleral depression[60 [A:III]]

There are no symptoms that can reliably distinguish a PVD with an associated retinal break from a PVD without an associated retinal break; therefore, a peripheral retinal examination is required.[60 [A:III]] The preferred method of evaluating peripheral vitreoretinal pathology is with indirect ophthalmoscopy using scleral depression.[61 [A:III]] Many patients with retinal tears have blood and pigmented cells in the anterior vitreous. Slit-lamp biomicroscopy with a mirrored contact lens or a small indirect condensing lens may complement the examination.

Diagnostic Tests

If it is impossible to evaluate the peripheral retina, B-scan ultrasonography should be performed to search for retinal tears or detachment and for other causes of vitreous hemorrhage.[62 [A:II]] Other experts have advocated the rarely used procedure of bilateral patching to attempt to clear the vitreous hemorrhage enough to detect retinal tears.[63] If no abnormalities are found, frequent follow-up examinations are recommended.[A:III]

TREATMENT

The goal of treatment is to create a firm chorioretinal adhesion with cryotherapy or laser photocoagulation in the attached retina immediately adjacent to and surrounding the retinal tear and/or the focal accumulation of subretinal fluid associated with the break.

Treatment of peripheral horseshoe tears should be extended to the ora serrata.[15,64,65 [A:II]] The most common cause of failure in treating horseshoe tears is failure to treat completely to the ora serrata. Continued vitreous traction may extend the tear beyond the treated area, thus allowing fluid to leak into the subretinal space resulting in a clinical retinal detachment.[15,64,65] Treatment of dialyses must extend over the entire length of the tear, reaching the ora serrata beyond each horn or end of the dialysis.

Sufficient information to make evidence-based recommendations for treatment of these conditions exists only for acute, symptomatic horseshoe tears. There is insufficient information to make evidence-based recommendations for management for other vitreoretinal abnormalities. In making the decision to treat other vitreoretinal abnormalities, including lattice degeneration and asymptomatic retinal breaks, the risks that treatment will be unnecessary, ineffective, or harmful must be weighed against the possible benefit of reducing the rate of subsequent retinal detachment. Table 1 summarizes recommendations for management.

The surgeon should inform the patient of the relative risks, benefits, and alternatives to surgery.[66,67 [A:III]] The surgeon is responsible for formulating a postoperative care plan and should inform the patient of these arrangements.[66,67 [A:III]]

Retinal detachments may occur in spite of appropriate therapy. Traction may pull the tear off the treated area, especially when breaks are large or have bridging retinal blood vessels, because the

treatment adhesion is not complete for up to 1 month.[15,17,64] Furthermore, 10% to 16% of patients will develop additional breaks during long-term follow-up.[17,68,69] Pseudophakic patients are more likely to require retreatment or to develop new breaks.[17]

TABLE 1 MANAGEMENT OPTIONS

Type of Lesion	Treatment*
Acute symptomatic horseshoe tears	Treat promptly[12,13,15-18 [A:II]]
Acute symptomatic operculated tears	Treatment may not be necessary[A:III]
Traumatic retinal breaks	Usually treated[A:III]
Asymptomatic horseshoe tears	Usually can be followed without treatment[A:III]
Asymptomatic operculated tears	Treatment is rarely recommended[A:III]
Asymptomatic atrophic round holes	Treatment is rarely recommended[A:III]
Asymptomatic lattice degeneration without holes	Not treated unless PVD causes a horseshoe tear[A:III]
Asymptomatic lattice degeneration with holes	Usually does not require treatment[A:III]
Asymptomatic dialyses	No consensus on treatment and insufficient evidence to guide management
Eyes with atrophic holes, lattice degeneration, or asymptomatic horseshoe tears where the fellow eye has had a retinal detachment	No consensus on treatment and insufficient evidence to guide management

PVD = posterior vitreous detachment

* There is insufficient evidence to recommend prophylaxis of asymptomatic retinal breaks for patients undergoing cataract surgery.

Complications of Treatment

Epiretinal membrane proliferation (macular pucker) has been observed after treatment for PVD, but it remains unknown whether treatment increases the risk of epiretinal membrane formation. In one long-term follow-up study, the percentage of eyes that developed macular pucker after treatment of retinal breaks was no greater than the percentage of eyes observed to have macular pucker before treatment.[15] The method of creating a chorioretinal adhesion appears to be unrelated to the incidence of postoperative macular pucker.[70]

FOLLOW-UP

The guidelines in Table 2 are for routine follow-up in the absence of additional symptoms. Patients with new symptoms or a change in symptoms may require more frequent evaluation. Patients with no positive findings at the initial examination should be seen at the intervals recommended in the Comprehensive Adult Medical Eye Evaluation PPP.[1 [A:III]] All patients with risk factors should be advised to contact their ophthalmologist promptly if new symptoms such as flashes, floaters, peripheral visual field loss, or decreased visual acuity develop.[27,28,56,71 [A:II]]

Younger myopic patients who have lattice degeneration with holes need regular follow-up visits, because they can develop small, localized retinal detachments (subclinical retinal detachments) that may slowly enlarge to become clinical retinal detachments. Treatment should be considered if the detachments progress in size.[21,23 [A:III]]

Patients presenting with an acute PVD and no retinal breaks have a small chance of developing retinal breaks in the weeks that follow.[7] Thus, selected patients, particularly those with any degree of vitreous pigment, hemorrhage in the vitreous or on the retina, or visible vitreoretinal traction, should be asked to return for a second examination within 6 weeks following the onset of symptoms.[7,69 [A:III]]

TABLE 2 RECOMMENDED GUIDELINES FOR FOLLOW-UP[A:III]

Type of Lesion	Follow-up Interval
Symptomatic PVD with no retinal break	Depending on symptoms, risk factors, and clinical findings, patients may be followed in 1 to 6 weeks, then 6 months to 1 year
Acute symptomatic horseshoe tears	1 to 2 weeks after treatment, then 4 to 6 weeks, then 3 to 6 months, then annually
Acute symptomatic operculated tears	2 to 4 weeks, then 1 to 3 months, then 6 to 12 months, then annually
Traumatic retinal breaks	1 to 2 weeks after treatment, then 4 to 6 weeks, then 3 to 6 months, then annually
Asymptomatic horseshoe tears	1 to 4 weeks, then 2 to 4 months, then 6 to 12 months, then annually
Asymptomatic operculated tears	2 to 4 weeks, then 1 to 3 months, then 6 to 12 months, then annually
Asymptomatic atrophic round holes	1 to 2 years
Asymptomatic lattice degeneration without holes	Annually
Asymptomatic lattice degeneration with holes	Annually
Asymptomatic dialyses	If untreated, 1 month, then 3 months, then 6 months, then every 6 months If treated, 1 to 2 weeks after treatment, then 4 to 6 weeks, then 3 to 6 months, then annually
Eyes with atrophic holes, lattice degeneration, or asymptomatic horseshoe tears in patients in whom the fellow eye has had a retinal detachment	Every 6 to 12 months

PVD = posterior vitreous detachment

History

A patient history should identify changes in the following:

- Visual symptoms[4-8,59 [A:I]]
- Interval history of eye trauma or intraocular surgery[28,43,72 [A:II]]

Examination

The eye examination should emphasize the following elements:

- Measurement of visual acuity[[A:III]]
- Evaluation of the status of the vitreous, with attention to the presence of pigment, hemorrhage, or syneresis[4-9,59 [A:II]]
- Examination of the peripheral fundus using scleral depression[60,73 [A:II]]
- B-scan ultrasonography if the media are opaque[62 [A:II]]

For treated patients, if the treatment appears satisfactory at the first follow-up visit, indirect ophthalmoscopy and scleral depression at 3 or more weeks will determine the adequacy of the chorioretinal scar, especially around the anterior boundary of the tear. If the tear and the accompanying subretinal fluid are not completely surrounded by the chorioretinal scar, additional treatment should be administered.[[A:III]] At any postoperative visit, if subretinal fluid has accumulated beyond the edge of treatment, additional treatment should be considered.[15,17 [A:III]]

Even if a patient has had adequate treatment, additional examinations are important. Between 5% and 14% of patients found to have an initial retinal break will develop additional breaks during long-term follow-up, and these percentages appear to be similar regardless of how the initial breaks were treated.[17,68] New breaks may be particularly likely in eyes that have had cataract surgery.[17]

PROVIDER

It is essential that ancillary clinical personnel be familiar with the symptoms of PVD and retinal detachment so that symptomatic patients can gain prompt access to the health care system.[8 [A:II]] Patients with symptoms of possible or suspected PVD or retinal detachment and related disorders

should be examined promptly by an ophthalmologist skilled in binocular indirect ophthalmoscopy and supplementary techniques.[A:III] Patients with retinal breaks or detachments should be treated by an ophthalmologist with experience in the management of these conditions.[A:III]

COUNSELING/REFERRAL

All patients at increased risk of retinal detachment should be instructed to notify their ophthalmologist promptly if they have a substantial change in symptoms, such as an increase in floaters, loss of visual field, or decrease in visual acuity.[27,28,56,71] [A:II] If patients are familiar with the symptoms of retinal tears or detachment, they may be more likely to report promptly after their onset, increasing the chances for successful surgical and visual results.[2] Patients who undergo refractive surgery to reduce myopia should be informed that they remain at risk of RRD despite reduction of their refractive error.[A:III]

APPENDIX 1. QUALITY OF OPHTHALMIC CARE CORE CRITERIA

Providing quality care
is the physician's foremost ethical obligation, and is
the basis of public trust in physicians.
AMA Board of Trustees, 1986

Quality ophthalmic care is provided in a manner and with the skill that is consistent with the best interests of the patient. The discussion that follows characterizes the core elements of such care.

The ophthalmologist is first and foremost a physician. As such, the ophthalmologist demonstrates compassion and concern for the individual, and utilizes the science and art of medicine to help alleviate patient fear and suffering. The ophthalmologist strives to develop and maintain clinical skills at the highest feasible level, consistent with the needs of patients, through training and continuing education. The ophthalmologist evaluates those skills and medical knowledge in relation to the needs of the patient and responds accordingly. The ophthalmologist also ensures that needy patients receive necessary care directly or through referral to appropriate persons and facilities that will provide such care, and he or she supports activities that promote health and prevent disease and disability.

The ophthalmologist recognizes that disease places patients in a disadvantaged, dependent state. The ophthalmologist respects the dignity and integrity of his or her patients, and does not exploit their vulnerability.

Quality ophthalmic care has the following optimal attributes, among others.

- The essence of quality care is a meaningful partnership relationship between patient and physician. The ophthalmologist strives to communicate effectively with his or her patients, listening carefully to their needs and concerns. In turn, the ophthalmologist educates his or her patients about the nature and prognosis of their condition and about proper and appropriate therapeutic modalities. This is to ensure their meaningful participation (appropriate to their unique physical, intellectual and emotional state) in decisions affecting their management and care, to improve their motivation and compliance with the agreed plan of treatment, and to help alleviate their fears and concerns.
- The ophthalmologist uses his or her best judgment in choosing and timing appropriate diagnostic and therapeutic modalities as well as the frequency of evaluation and follow-up, with due regard to the urgency and nature of the patient's condition and unique needs and desires.
- The ophthalmologist carries out only those procedures for which he or she is adequately trained, experienced and competent, or, when necessary, is assisted by someone who is, depending on the urgency of the problem and availability and accessibility of alternative providers.

- Patients are assured access to, and continuity of, needed and appropriate ophthalmic care, which can be described as follows.
  - The ophthalmologist treats patients with due regard to timeliness, appropriateness and his or her own ability to provide such care.
  - The operating ophthalmologist makes adequate provision for appropriate pre- and postoperative patient care.
  - When the ophthalmologist is unavailable for his or her patient, he or she provides appropriate alternate ophthalmic care, with adequate mechanisms for informing patients of the existence of such care and procedures for obtaining it.
  - The ophthalmologist refers patients to other ophthalmologists and eye care providers based on the timeliness and appropriateness of such referral, the patient's needs, the competence and qualifications of the person to whom the referral is made, and access and availability.
  - The ophthalmologist seeks appropriate consultation with due regard to the nature of the ocular or other medical or surgical problem. Consultants are suggested for their skill, competence and accessibility. They receive as complete and accurate an accounting of the problem as necessary to provide efficient and effective advice or intervention, and in turn respond in an adequate and timely manner.
  - The ophthalmologist maintains complete and accurate medical records.
  - On appropriate request, the ophthalmologist provides and full and accurate rendering of the patient's records in his or her possession.
  - The ophthalmologist reviews the results of consultations and laboratory tests in a timely and effective manner and takes appropriate actions.
  - The ophthalmologist and those who assist in providing care identify themselves and their profession.
  - For patients whose conditions fail to respond to treatment and for whom further treatment is unavailable, the ophthalmologist provides proper professional support, counseling, rehabilitative and social services, and referral as appropriate and accessible.
- Prior to therapeutic or invasive diagnostic procedures, the ophthalmologist becomes appropriately conversant with the patient's condition by collecting pertinent historical information and performing relevant preoperative examinations. Additionally, he or she enables the patient to reach a fully informed decision by providing an accurate and truthful explanation of the diagnosis; the nature, purpose, risks, benefits, and probability of success of the proposed treatment and of alternative treatment; and the risks and benefits of no treatment.
- The ophthalmologist adopts new technology (e.g., drugs, devices, surgical techniques) in judicious fashion, appropriate to the cost and potential benefit relative to existing alternatives and to its demonstrated safety and efficacy.
- The ophthalmologist enhances the quality of care he or she provides by periodically reviewing and assessing his or her personal performance in relation to established standards, and by revising or altering his or her practices and techniques appropriately.
- The ophthalmologist improves ophthalmic care by communicating to colleagues, through appropriate professional channels, knowledge gained through clinical research and practice. This includes alerting colleagues of instances of unusual or unexpected rates of complications and problems related to new drugs, devices or procedures.
- The ophthalmologist provides care in suitably staffed and equipped facilities adequate to deal with potential ocular and systemic complications requiring immediate attention.
- The ophthalmologist also provides ophthalmic care in a manner that is cost effective without unacceptably compromising accepted standards of quality.

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APPENDIX 2. SUMMARY OF MAJOR RECOMMENDATIONS FOR CARE

DIAGNOSIS

The initial evaluation of a patient with risk factors or symptoms includes all features of the comprehensive adult medical eye evaluation,[54] with particular attention to those aspects relevant to PVD, retinal breaks, and lattice degeneration.

History

A patient history should include the following elements:

- Symptoms of PVD[4-8] [A:I]
- Family history[47,48] [A:II]
- Prior eye trauma[43] [A:III]
- Myopia[30,55] [A:II]
- History of ocular surgery, including refractive lens exchange and cataract surgery[27-29,56-58] [A:II]

Examination

The eye examination should include the following elements:

- Examination of the vitreous for hemorrhage, detachment, and pigmented cells[4-9,59] [A:II]
- Peripheral fundus examination with scleral depression[60] [A:III]

There are no symptoms that can reliably distinguish a PVD with an associated retinal break from a PVD without an associated retinal break; therefore, a peripheral retinal examination is required.[60] [A:III] The preferred method of evaluating peripheral vitreoretinal pathology is with indirect ophthalmoscopy combined with scleral depression.[61] [A:III]

Diagnostic Tests

If it is impossible to evaluate the peripheral retina, B-scan ultrasonography should be performed to search for retinal tears or detachment and for other causes of vitreous hemorrhage.[62] [A:II]

TREATMENT

Table A2 summarizes recommendations for management.

TABLE A2 MANAGEMENT OPTIONS

Type of Lesion	Treatment*
Acute symptomatic horseshoe tears	Treat promptly[12,13,15-18] [A:II]
Acute symptomatic operculated tears	Treatment may not be necessary[A:III]
Traumatic retinal breaks	Usually treated[A:III]
Asymptomatic horseshoe tears	Usually can be followed without treatment[A:III]
Asymptomatic operculated tears	Treatment is rarely recommended[A:III]
Asymptomatic atrophic round holes	Treatment is rarely recommended[A:III]
Asymptomatic lattice degeneration without holes	Not treated unless PVD causes a horseshoe tear[A:III]
Asymptomatic lattice degeneration with holes	Usually does not require treatment[A:III]
Asymptomatic dialyses	No consensus on treatment and insufficient evidence to guide management
Eyes with atrophic holes, lattice degeneration, or asymptomatic horseshoe tears where the fellow eye has had a retinal detachment	No consensus on treatment and insufficient evidence to guide management

PVD = posterior vitreous detachment

* There is insufficient evidence to recommend prophylaxis of asymptomatic retinal breaks for patients undergoing cataract surgery.

The surgeon should inform the patient of the relative risks, benefits, and alternatives to surgery.[66,67 [A:III]] The surgeon is responsible for formulating a postoperative care plan and should inform the patient of these arrangements.[66,67 [A:III]]

FOLLOW-UP

The guidelines in Table 2 in the main body of the text are for routine follow-up in the absence of additional symptoms. Patients with no positive findings at the initial examination should be seen at the intervals recommended in the Comprehensive Adult Medical Eye Evaluation PPP.[54 [A:III]] All patients with risk factors should be advised to contact their ophthalmologist promptly if new symptoms such as flashes, floaters, peripheral visual field loss, or decreased visual acuity develop.[27,28,56,71 [A:II]]

History

A patient history should identify changes in the following:

- Visual symptoms[4-8,59 [A:I]]
- Interval history of eye trauma or intraocular surgery[28,43,72 [A:II]]

Examination

The eye examination should emphasize the following elements:

- Measurement of visual acuity[A:III]
- Evaluation of the status of the vitreous, with attention to the presence of pigment, hemorrhage, or syneresis[4-9,59 [A:II]]
- Examination of the peripheral fundus with scleral depression[60,73 [A:II]]
- B-scan ultrasonography if the media are opaque[62 [A:II]]

PROVIDER

It is essential that ancillary clinical personnel be familiar with the symptoms of PVD and retinal detachment so that symptomatic patients can gain prompt access to the health care system.[8 [A:II]] Patients with symptoms of possible or suspected PVD or retinal detachment and related disorders should be examined promptly by an ophthalmologist skilled in binocular indirect ophthalmoscopy and supplementary techniques.[A:III] Patients with retinal breaks or detachments should be treated by an ophthalmologist with experience in the management of these conditions.[A:III]

COUNSELING/REFERRAL

All patients at increased risk of retinal detachment should be instructed to notify their ophthalmologist promptly if they have a substantial change in symptoms, such as an increase in floaters, loss of visual field, or decrease in visual acuity.[27,28,56,71 [A:II]] Patients who undergo refractive surgery to reduce myopia should be informed that they remain at risk of RRD despite reduction of their refractive error.[A:III]

APPENDIX 3. VITREOUS HEMORRHAGE

Vitreous hemorrhage can be the presenting sign of PVD or it may present during the evolution of the PVD. Approximately 80% of patients who have no breaks on presentation, but then develop breaks later, have at least one of the following characteristics: pigmented cells or hemorrhage in the vitreous or retina at the initial evaluation, or new symptoms that prompted a return visit to the ophthalmologist.[9] Sixty-seven percent of patients who present with associated vitreous hemorrhage were found to have at least one break, with 31% having more than one break and 88% of the breaks occurring in the superior quadrants.[74]

On examination, even if the presence of vitreous hemorrhage is sufficiently dense to obscure the posterior pole, the peripheral retina frequently can be seen with indirect ophthalmoscopy and scleral depression. Patients who present with vitreous hemorrhage sufficient to obscure retinal details and a negative B-scan ultrasonographic evaluation should be followed periodically. For eyes in which a retinal tear is suspected, a repeat ultrasonographic study should be performed within approximately 4 weeks of the initial evaluation.

GLOSSARY

Atrophic retinal breaks or holes: Full-thickness retinal defects, unrelated to vitreoretinal traction. These can occur within lattice lesions or in areas of the retina that appear otherwise normal.

Cystic retinal tufts: Small congenital lesions of the peripheral retina. They are slightly elevated and usually whitish in color with variable surrounding pigmentation. They are firmly attached to the overlying vitreous cortex and are sometimes a cause of retinal tears following PVD.

Epiretinal membrane: See Macular pucker.

Flap retinal tear: A horseshoe tear.

Horseshoe tear: A retinal tear caused by vitreoretinal traction on the retina. The tear is horseshoe shaped due to a flap of torn tissue that remains attached to the detached vitreous gel.

ICD-9: International Statistical Classification of Diseases and Related Health Problems, Ninth Edition.

Lattice degeneration: A peripheral vitreoretinal lesion characterized by retinal thinning, overlying vitreous liquefaction, and firm vitreoretinal adhesions at its margins. Most lesions are ovoid with long axes parallel to the ora serrata. Round holes frequently occur within the lattice lesion unassociated with PVD. If horseshoe tears are present, they are seen at the development of PVD and usually are observed at the margins of lattice lesions.

Macular pucker: Distortion of the retina in the macular region due to proliferation and contraction of a fibrocellular membrane on the inner surface of the retina.

Operculated retinal tear or break: A defect in the retina caused by vitreoretinal traction at the site of the lesion. The traction pulls a circular or oval piece of retinal tissue (the operculum) free from the retinal surface. If this occurs during PVD, all traction in the vicinity of the retinal break is usually eliminated.

Posterior vitreous detachment (PVD): A separation of the posterior vitreous cortex from the internal surface of the retina. This usually occurs as an acute event after substantial age-related liquefaction in the vitreous gel; the separation usually extends rapidly to the posterior margin of the vitreous base in all quadrants. Adhesions between the vitreous cortex and retina or retinal blood vessels may cause retinal breaks and/or vessel rupture. Vitreous hemorrhage and/or localized intraretinal hemorrhage may accompany this event. Posterior vitreous detachment is

diagnosed by slit-lamp biomicroscopy, which will usually show a prominent plane defining the posterior vitreous face. The presence of a glial annulus in the vitreous cavity (Weiss ring) is strong evidence of PVD.

PVD: See Posterior vitreous detachment.

Retinal breaks: Full-thickness defects in the retina. Those caused by vitreoretinal traction are usually called tears. Those that are round and unassociated with vitreoretinal traction are usually called holes.

Retinal dialysis: A specific type of crescentic peripheral retinal break at the ora serrata, usually associated with trauma.

Rhegmatogenous retinal detachment (RRD): A separation of the retina from the retinal pigment epithelium caused by fluid passing from the vitreous cavity into the subretinal space through a break in the retina (from Greek *rhegma,* "rent").

Round retinal hole: A round, full-thickness defect or break in the retina, unassociated with vitreoretinal traction.

RRD: See Rhegmatogenous retinal detachment.

Stickler syndrome: The most common inherited vitreoretinal and systemic disorder associated with RRD. Ocular features include (1) high myopia; (2) retrolental, transvitreal, and epiretinal membranes and strands; (3) chorioretinal pigment alterations; (4) lattice degeneration, often with a perivascular component that extends posteriorly; and (5) various other abnormalities including glaucoma and cataract. Systemic features include a generalized skeletal dysplasia, often with a marfanoid habitus, flattened facies, high arched or cleft palate, hearing loss, and other extracranial skeletal anomalies, many of which can be very subtle. The inheritance pattern is autosomal dominant, and a gene defect has been related to COL2A1.

Vitreoretinal adhesion: A firm attachment between the cortical vitreous and the inner surface of the retina. Condensed vitreous strands adhering to the retina may sometimes be visualized with biomicroscopy or indirect ophthalmoscopy and scleral depression. Traction of the vitreous on the retina during PVD may cause retinal breaks to occur at these sites.

Zonular traction retinal tufts: Small congenital lesions of the peripheral retina caused by thickened zonules that have been displaced posteriorly to the anterior retina.

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