Retinal detachment

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Literature review current through: **May 2024.**
This topic last updated: **Dec 08, 2023.**

INTRODUCTION

Retinal detachment refers to the separation of the retina from the underlying retinal pigment epithelium and choroid. Without treatment, many peripheral retinal detachments progress to involve the central retina and lead to loss of vision.

This topic will address the pathophysiology, clinical presentation, treatment, and prevention of retinal detachment, primarily the most common type, rhegmatogenous retinal detachment (RRD). Understanding the basic pathophysiology involved in the process of retinal tear formation and retinal detachment, and the symptoms and signs of the early stages of this process, are important in identifying high-risk patients and preventing loss of vision.

NORMAL ANATOMY

The retina is the innermost layer of the eye, composed of 10 different layers including photoreceptors, which are the light-sensitive cells in the eye. These photoreceptors are interconnected with neurons that combine to form the optic nerve [1]. The photoreceptors, also called the neurosensory retina, take light information that enters the eye and convert it via a chemical reaction into electric signals that are transmitted to the brain through the optic nerve [2]. Underneath the retina is the retinal pigment epithelium, which is a pigmented cell layer that plays a role in the visual cycle and nourishes the overlying retina.

In the center of the retina just temporal to the optic nerve is a region called the macula, an avascular zone containing the highest density of cone photoreceptors and responsible for the

sharpest visual acuity. This is the area of the retina that is responsible for central and reading vision. The peripheral retina contains more rod photoreceptors, which are used for dark adaptation and peripheral vision [3].

PATHOPHYSIOLOGY OF DETACHMENT

Retinal detachment occurs when the retina separates from the underlying retinal pigment epithelium and choroid, resulting in retinal ischemia and photoreceptor degeneration. Separation can occur passively, as when a retinal hole or tear allows for an accumulation of fluid between the layers, or actively, as in the case of either vitreous traction or an exudative process (figure 1).

Embryologically, the neurosensory retina (ie, photoreceptor layer) and the underlying retinal pigment epithelium layer are derived from the inner layer and outer layer, respectively, of the optic cup. These two different layers have no anatomic junction cells forming strong adhesions, so there is a potential space between the retina and the underlying retinal pigment epithelium layer. Consequently, a retinal detachment can form when fluid enters the subretinal space between the neurosensory retina and retinal pigment epithelium, reestablishing the potential space between the two layers [4].

There are two main categories of retinal detachments: rhegmatogenous and nonrhegmatogenous retinal detachments.

Rhegmatogenous retinal detachments — A rhegmatogenous retinal detachment (RRD) is the most common type of retinal detachment. It results from a full-thickness break in the retina that allows liquefied vitreous, which is the jelly-like fluid inside the eye, to enter the subretinal space. Most cases of RRD are preceded by a posterior vitreous detachment (PVD), an age-related process where the vitreous fluid separates from the innermost retinal surface layer (figure 2). This process can pull on the retina with the resulting tractional forces creating a retinal break. If the vitreous separates cleanly from the area of the retinal break or the patient is young and their vitreous has not liquefied, a retinal detachment may not occur. Typically, tractional forces and liquefied vitreous increase the chance that a retinal break will result in RRD [5].

The main anatomical classification of detachments is based on whether they involve the central portion of the retina (the macula) or not. Macula-on retinal detachments spare the central retina, and, consequently, vision at presentation is usually better and visual outcomes after repair are also more favorable. Macula-off retinal detachments involve the macula.

Nonrhegmatogenous retinal detachment — Nonrhegmatogenous retinal detachments include exudative or serous retinal detachments, as well as tractional retinal detachments. In both cases, fluid enters the subretinal space without a retinal break.

- **Exudative retinal detachment** – In the case of an exudative or serous retinal detachment, fluid can enter the subretinal space from either retina or choroidal blood vessels. Due to an imbalance of hydrostatic and osmotic pressures, the adhesive forces between the neurosensory retina and retinal pigment epithelium are overwhelmed. This can occur due to choroidal tumors, uveitis, hypertension, ocular vascular disorders, medications, other systemic inflammatory conditions, collagen vascular diseases, or infectious diseases [6].

- **Tractional retinal detachment** – Tractional retinal detachments occur when there is a separation of the neurosensory retina and retinal pigment epithelium due to pulling on the inner surface of the retina but without the creation of a retinal break. These can occur from proliferative vitreoretinal conditions such as diabetic retinopathy, retinopathy of prematurity, sickle cell retinopathy, proliferative vitreoretinopathy, and traumatic retinopathy. In most cases, contractile fibrous bands form in the vitreous cavity that grow on the inner surface of the retina [7].

Combined traction RRD can also occur where tractional forces from fibrous or fibrovascular proliferation induce a full-thickness retinal break, as occurs in proliferative diabetic retinopathy [8].

PRECURSOR CONDITIONS

Several risk factors for rhegmatogenous retinal detachment (RRD) exist, with the most common ones being posterior vitreous detachment (PVD), lattice degeneration, peripheral retinal breaks, pathologic myopia, previous intraocular surgery, trauma, previous retinal detachment, family history [9,10], and tractional retinal detachment such as proliferative diabetic retinopathy [8,11].

Posterior vitreous detachment — PVD is typically an age-related process that occurs between ages 45 to 65 among the general population, with an earlier onset in men, or when due to trauma or myopia [12]. PVD occurs when the vitreous gel liquefies and the usual vitreous retina adhesions begin to weaken. This results in a separation of the vitreous gel from the inner-surface of the retina that can be preceded by traction, particularly in the peripheral retina, causing a full-thickness retinal break. The vitreous gel is also tightly adherent to the macula, optic nerve, and retinal blood vessels. PVDs can also cause vitreous hemorrhage bleeding in the vitreous cavity if the traction on a retinal blood vessel is strong enough to create a break in the

blood vessel wall [4]. The prevalence of PVD increases with age but also occurs in younger patients who are myopic or who have a history of ocular trauma or inflammation [12].

During the initial examination, approximately 8 to 22 percent of patients experiencing acute PVD symptoms are found to have a retinal tear [13]. However, retinal tears may also develop in the ensuing weeks [13-15].

Retinal tears — Retinal tears, also called horseshoe tears due to their U-shaped appearance, are full-thickness breaks in the retina. They occur from traction applied by the vitreous gel on the inner surface of the retina, and most commonly occur in the setting of a developing PVD. Because PVDs typically occur in late middle age, retinal tears also occur most commonly in this age group. Retinal tears can be divided into asymptomatic and symptomatic. Symptomatic retinal tears refer to a tear that occurs due to vitreoretinal traction in a patient who has recently experienced a PVD along with the sudden onset of flashes and/or floaters. More than 50 percent of untreated symptomatic retinal tears will progress into retinal detachment [16,17]. On the other hand, asymptomatic retinal tears have around 5 to 8 percent risk of progression into retinal detachment [18].

Retinal hole — Retinal holes refer to a round full-thickness opening in the neurosensory retina. They can occur either in the setting of a retinal tear with persistent traction that subsequently progresses such that the flap avulses from the rest of the retina or in the setting of progressive thinning of the retina resulting in atrophic retinal holes. The latter occurs more commonly in young myopic patients and is usually asymptomatic. Retinal holes may result in RRDs but are less of a risk factor than retinal tears because the latter have persistent vitreoretinal traction. In fact, there are case series studies over 11-year periods that have reported no progression of asymptomatic retinal holes to become RRDs [18].

EPIDEMIOLOGY AND RISK FACTORS

The incidence of rhegmatogenous retinal detachments (RRDs) ranges from 6.3 to 17.9 per 100,000 population [19].

Risk factors for the development of RRDs include posterior vitreous detachment (PVD), older age, prior intraocular surgery, myopia, lattice degeneration, family history of retinal detachment, history of retinal detachment in the other eye, ocular trauma, and congenital connective tissue disorders (such as Stickler syndrome, Marfan syndrome, or Ehlers-Danlos syndrome) [20]. They can be delayed complications of cataract surgery. (See "Cataract in adults", section on 'Outcomes'.)

Tractional retinal detachments typically arise in patients with diabetes and exudative retinal detachments in patients with inflammatory conditions of the eye [21].

CLINICAL PRESENTATION

Patients with retinal detachments present with painless loss of vision in the affected eye. The earliest symptoms of a retinal tear or rhegmatogenous retinal detachment (RRD) may also include a sudden increase (over minutes to hours) in floaters (small, shadowy shapes that appear to float in front of the eye) and/or flashes of light (photopsias). Once a retinal detachment has occurred, patients may notice a dark curtain over part of their visual field. When the retinal detachment involves only the peripheral retina (macula-on), the central visual acuity remains sharp.

Progression of symptoms — In RRDs, greater visual field loss occurs as liquefied vitreous continues to enter the subretinal space and further retinal separation occurs. Symptoms typically progress over hours to a few days. Once the macula becomes involved, patients lose central vision. If not treated, a retinal detachment can progress to involve the entire retina, resulting in complete loss of vision. Nonrhegmatogenous detachments tend to progress over weeks to months.

DIAGNOSIS AND EVALUATION

The diagnosis of retinal detachment is suspected based on history (eg, sudden onset of floaters, flashes of light [photopsias], and/or loss of vision) and confirmed by ophthalmologic examination.

Primary care evaluation — The primary care evaluation should center on which patients need prompt referral to an ophthalmologist for a dilated eye examination as this is the most accurate method for determining whether a patient has a retinal detachment.

Patients with new onset of floaters, flashes, and painless vision loss (whether partial or complete) should be referred promptly to an ophthalmologist and evaluated within 24 hours. Whether the patient has a retinal tear or retinal detachment, both require urgent treatment. If a patient's vision loss is partial and central vision remains spared, this does not decrease the urgency of referral compared with a patient with complete vision loss. Treatment of a retinal tear before it progresses to a rhegmatogenous retinal detachment (RRD) or a macula-on RRD before it progresses to macula-off result in better long-term outcomes and prevent permanent vision loss.

The patient's history and family history should be reviewed. If possible, check the visual acuity in the office, perform a pupil light reflex test, and evaluate visual fields via confrontation.

All patients, especially those with high-risk features (visual field loss, subjective or objective decreased vision, at least 10 new floaters, or evidence of vitreous hemorrhage on funduscopic examination [if this was performed]) should be instructed to decrease eye movement (such as not reading, watching television, or exercising) and sent urgently to an ophthalmologist or retinal surgeon within one day [22].

Ophthalmologist evaluation — At the ophthalmologist's office, the patient will undergo testing to check their visual acuity and intraocular pressures, an anterior segment examination to determine lens status (phakic, pseudophakic, or aphakic), a detailed dilated fundus examination using special condensing lenses to visualize the retina, and, possibly, an ultrasound if there is concurrent vitreous hemorrhage that may prevent adequate visualization of the retina (image 1).

Patients should also undergo examination of the fellow eye to determine if there is an asymptomatic tear or detachment. There is a relatively high rate of bilateral retinal tears or detachments (8 to 40 percent) [23].

If a retinal tear or retinal detachment is present, a general ophthalmologist will usually refer the patient to a retina specialist for treatment.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of retinal detachment includes the differential diagnosis of acute painless vision loss or flashes of light and floaters (see "Approach to the adult with acute persistent visual loss"):

- Migraine with or without aura (flashes of light/photopsia)
- Posterior vitreous detachment (PVD) (flashes of light and floaters)
- Retinal artery occlusion (painless vision loss
- Retinal vein occlusion (painless vision loss)
- Vitreous hemorrhage (floaters)
- Ischemic optic neuropathy (painless vision loss)
- Occipital lobe stroke (painless vision loss)
- Intraocular inflammation (floaters with photophobia)
- Vitreous amyloidosis (floaters)
- Intraocular lymphoma (floaters)

- Vitreous syneresis (benign floaters)

MANAGEMENT OF PRECURSOR CONDITIONS

Examination and management of posterior vitreous detachment — Indirect ophthalmoscopy with scleral depression or slit-lamp biomicroscopy are used to clinically diagnose posterior vitreous detachment (PVD) and rule out retinal tear or retinal detachment. In the absence of a retinal tear or retinal detachment, no treatment is needed, but close monitoring within four to six weeks is required as the evolution of a PVD may result in new retinal breaks. Most importantly, patients diagnosed with a PVD should be given specific instructions for return if they experience any increase in floaters or flashes or develop visual field loss.

Retinal breaks

Symptomatic retinal breaks — Retinal breaks can occur in patients with PVD, as well as other patients. Approximately 7 to 18 percent of eyes with a symptomatic PVD will have one or more retinal tears [13,24]. The presence of vitreous or retinal hemorrhage also significantly increases the risk of a concomitant retinal tear [25]. Symptomatic retinal tears have a high risk of progressing to a rhegmatogenous retinal detachment (RRD), and prompt treatment within one to two days is indicated with either laser retinopexy or cryoretinopexy. Both types of treatment create a chorioretinal adhesion around the retinal tear, thus sealing the subretinal space from the vitreous fluid and preventing further enlargement and progression of the retinal tear. This adhesion is created using a thermal laser in the setting of laser retinopexy and using a freezing probe in the setting of cryoretinopexy. Both procedures can be performed in the office.

Treatment is initially successful in approximately 80 percent of patients, with the remaining patients requiring further treatment due to inadequate closure of the break, new break formation, or subsequent retinal detachment [26,27]. In a systematic review of studies reporting outcomes among patients with symptomatic retinal breaks, the cumulated incidence of subsequent retinal detachment among those treated with laser photocoagulation or cryoretinopexy treatment was between 2.1 and 8.8 percent, compared with 35 to 47 percent among untreated patients [28].

Data on outcomes after cryoretinopexy treatment are limited. In one study from the 1970s, 231 eyes with retinal breaks were treated successfully without subsequent retinal detachment or new break formation [29].

Asymptomatic retinal breaks — Asymptomatic retinal tears progress to retinal detachment in approximately 5 percent of cases [18]. Due to the lower risk of RRD, not all retina specialists treat asymptomatic retinal tears, and treatment is typically guided by risk factors for progression. The presence of lattice degeneration, myopia, aphakia, pseudophakia, or history of retinal detachment in the fellow eye increases the risk of progression to RRD, and these patients may warrant treatment [30]. If a retinal tear is asymptomatic and appears chronic based on examination findings and the patient lacks the risk factors described above, we tend to observe, as opposed to treat, these tears. Patients who are not treated are seen in follow-up, typically in one to three months initially and then yearly if the retina examination is deemed to be stable.

Asymptomatic operculated and atrophic retinal holes rarely progress to RRD, and these are typically followed with routine eye examinations on a yearly basis without prophylactic laser retinopexy or cryoretinopexy.

Lattice degeneration — Lattice degeneration refers to thinning of the peripheral retina, typically seen in patients who are myopic. The presence of lattice degeneration alone without retinal tears has a low risk of progression to RRD. Thus, lattice degeneration is usually observed on a yearly basis unless there are strong risk factors for RRD such as a history of RRD in the fellow eye. If a symptomatic retinal tear occurs in an eye with lattice degeneration, prophylactic laser retinopexy or cryoretinopexy may be used to treat both the retinal tear and areas with lattice degeneration.

Fellow eye in patients with retinal detachment — In patients with an RRD, the fellow-eye risk of detachment is approximately 10 percent for phakic retinal detachment and as high as 20 to 36 percent for aphakic or pseudophakic detachment [31,32]. Consequently, prophylactic treatment of at-risk pathology in the fellow eye such as symptomatic or asymptomatic retinal tears, retinal holes, and lattice degeneration may be considered.

RETINAL DETACHMENT MANAGEMENT

Indications for urgent surgery — An acutely symptomatic retinal detachment should be surgically repaired as soon as possible (preferably within one to two days), particularly if the macula is not involved (macula-on retinal detachment). The reason that macula-on retinal detachments are repaired sooner is to preserve vision.

Patients should be advised to minimize activity and reading and, if possible, maintain bed rest with their head turned such that the area of detached retina remains in the most dependent position to reduce the progression of retinal detachment until surgery is performed [19].

There are three main methods of repairing retinal detachments: pneumatic retinopexy, scleral buckle, and pars plana vitrectomy (PPV).

Repair of large rhegmatogenous retinal detachments — Large rhegmatogenous retinal detachments (RRDs) are treated with either pneumatic retinopexy (along with either laser retinopexy or cryoretinopexy), scleral buckling, or PPV. Choice of procedure is based on clinical judgment, patient preferences, available resources, and the surgeon's skill and experience with each procedure.

The rate of vision improvement depends on whether gas tamponade is used and, if so, the size of the tamponade. Procedures without gas tamponade or ones using a small gas tamponade (ie, scleral buckling and pneumatic retinopexy) will result in faster visual improvement than those using a larger tamponade of either a short-acting gas (eg, SF6), which lasts for two to three weeks, or a long-acting gas (eg, C3F8), which lasts for six to eight weeks. Vision improvement will typically occur once the gas is 50 percent gone [33].

Pneumatic retinopexy — Pneumatic retinopexy is performed in the office setting. The procedure involves injecting a partial gas bubble into the vitreous cavity of the eye with concurrent or subsequent application of laser retinopexy or cryoretinopexy to seal the retinal breaks. After injection of the gas bubble, the patient is instructed to position in a specific way such that the gas bubble will tamponade and seal the retinal break, thus allowing the retina to reattach. Positioning in this manner is typically for a one-week period; the patient can be in a standing or seated position or in bed as long as they are able to position their head at the appropriate angle. This positioning should be maintained as close to 100 percent of the time as possible.

Pneumatic retinopexy leads to successful retinal attachment in approximately 70 to 80 percent of patients after one procedure [34-36]. In a trial comparing pneumatic retinopexy with scleral buckling among patients with RRD, the retina was successfully reattached with one or more procedures in 90 percent of those in the retinopexy group and in all of those in the scleral buckling group [37].

An important benefit of pneumatic retinopexy is that this procedure can be performed in the office and is relatively quick compared with the other two surgical procedures. In addition, pneumatic retinopexy has fewer postoperative complications than does scleral buckle [38].

Patient selection is crucial to maximize success, and this procedure is best used for uncomplicated retinal detachments involving the superior one-half of the retina [37].

Scleral buckling — Scleral buckling is the oldest method of repairing retinal detachments. Instead of an internal approach (as used in PPV), scleral buckling is focused on the outside of the eye where a surgical explant is sutured to the wall of the sclera. The inward indentation helps close retinal breaks by pushing the underlying retinal pigment epithelium closer to the retinal break, thus relieving the vitreoretinal traction that allows vitreous fluid to enter the subretinal space. This technique should always be used for patients with a detachment of the retina secondary to a post-traumatic dialysis [39] (figure 3).

Explants used in this procedure are typically made from soft or hard silicone, and their configuration around the eye may be radial, segmental, circumferential, or encircling depending on the size, configuration, and number of breaks. During the procedure to implant a scleral buckle, cryoretinopexy is often performed concurrently to help seal the retinal breaks, and drainage of subretinal fluid via an external approach may be used to help facilitate reattachment of the retina.

The anatomical success rate of reattaching the retina with scleral buckling is as high as 91.7 percent [40]. Complications after scleral buckle surgery may include diplopia, cystoid macular edema, epiretinal membrane, anterior segment ischemia, buckle extrusion, and buckle infection [41]. However, one large benefit of scleral buckling surgery is that it does not accelerate cataract formation compared with PPV; thus, this procedure is often considered in younger patients.

Pars plana vitrectomy — While scleral buckling is the oldest technique to repair retinal detachments, PPV has become the preferred treatment modality for most RRDs today. PPV is an internal, intraocular surgery, with three primary goals: (1) remove the traction around the retinal breaks, (2) tamponade the retinal breaks, and (3) seal the retinal breaks with laser retinopexy or cryoretinopexy.

For complex cases with significant scar tissue called proliferative vitreoretinopathy, PPV with or without scleral buckling may be the procedure of choice. PPV is also preferred if there is significant media opacity (eg, cataract, vitreous hemorrhage) that results in poor visualization of retinal breaks during scleral buckling surgery as these media opacities can be addressed during PPV surgery.

Once the traction is relieved from the retinal surface, a tamponade agent is used to close the retinal breaks via surface tension. Subsequently, the laser retinopexy or cryoretinopexy that is applied can create a permanent chorioretinal adhesion to prevent the retinal breaks from opening once the tamponade agent is dissipated or removed. The most common tamponade agents are gas including sulfur hexafluoride (SF6), perfluoropropane (C3F8), or silicone oil (SO). SF6 lasts for two to three weeks, C3F8 lasts for seven to eight weeks, and SO remains in the eye

until it is removed (usually six months after surgery). Those patients with an air or gas bubble are unable to travel on a plane until the bubble has been completely absorbed.

Vitrectomy leads to successful retinal reattachment in approximately 80 to 90 percent of patients with RRD [42]. However, virtually all patients over age 50 treated with vitrectomy will develop a visually significant cataract within 6 to 18 months.

Complications from PPV include formation of proliferative vitreoretinopathy and subsequent retinal redetachments in 10 to 15 percent of cases. High or low intraocular pressure can occur transiently in up to one-third of cases. Endophthalmitis and choroidal hemorrhages are rare but potentially devastating complications that occur much less frequently than 1 percent of the time [41,42].

Small, localized rhegmatogenous retinal detachments — For small, localized RRDs, laser retinopexy or cryoretinopexy may be used. In contrast with treating either retinal tears or retinal holes where treatment is applied around the edges of the break, when there is a small, localized RRD, there is some subretinal fluid present. Thus, laser retinopexy and cryoretinopexy must be applied both around the edges of the break where possible and around the subretinal fluid to prevent further expansion of the retinal detachment. If the subretinal fluid does expand beyond the treated area, then surgery with one of the above techniques will be needed.

Postoperative care — Postoperative care following retinal detachment surgery is crucial for successful recovery and to minimize complication. Specific postoperative instruction depends on the surgical procedure performed.

Patients are required to use eye drops during the postoperative period and these include antibiotics, steroid, atropine, and in some occasions intraocular pressure lowering drops.

Following pars plana vitrectomy or pneumatic retinopexy, (when gas is placed in the eye), the patient is required to be in a specific position usually for the first week (such as facedown position) with the goal of sealing the retina break and decreasing the chance of cataract progression. Most of the time, positioning is not required for a primary scleral buckle procedure.

Patients requiring gas tamponade must refrain from traveling to high altitudes (the patient should follow specific surgeon's instructions as to what altitude is safe) or flying due to the risk of gas expansion, which can lead to retinal artery occlusion and permanent vision impairment. Patients are also required to avoid any scuba diving until the gas bubble is resorbed. In situations where variation in altitude is inevitable, or when a patient needs to fly after surgery,

using silicone oil as tamponade is helpful, but this will require a second surgery to remove the silicone oil.

The use of nitrous oxide for general anesthesia is contraindicated in patients with intraocular gas following retina surgery as this can result in gas expansion and retina artery occlusion with permanent vision loss [43].

Management of nonrhegmatogenous retinal detachment

Tractional retinal detachment — Treatment of tractional retinal detachments usually requires vitrectomy with or without scleral buckling to relieve all retinal traction. An encircling scleral buckle may be used to relieve residual anterior traction and provide support for peripheral retinal breaks. A tamponade agent is often used as well.

The most common form of tractional retinal detachments arise from proliferative diabetic retinopathy. Because this type of retinal detachment is typically slow progressing, tractional retinal detachments not involving the macula may be observed as long as the proliferative diabetic retinopathy has been adequately treated with panretinal photocoagulation and/or antivascular endothelial growth factor intravitreal injections and the tractional retinal detachment remains stable [21].

Tractional retinal detachments that threaten or involve the macula should be treated promptly, similar to RRDs.

Exudative retinal detachment — Treatment is directed at the underlying cause. If the cause is from uveitis or systemic conditions, systemic corticosteroids may be used. If medications are the offending agent, then cessation of these medications may be recommended if safe to do so. If the exudative retinal detachment is related to a choroidal tumor, treatment of the tumor is recommended first, which may lead to resolution of the retinal detachment.

If treatment of the underlying cause does not lead to resolution of the subretinal fluid and retinal detachment, PPV may be warranted.

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more

sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Detached retina (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Pathophysiology** – Retinal detachment occurs when the retina separates from the underlying retinal pigment epithelium and choroid, resulting in retinal ischemia and photoreceptor degeneration. Without treatment, many symptomatic retinal detachments progress to involve the entire retina and lead to loss of vision.
  - There are two main categories of retinal detachments: rhegmatogenous (more common) and nonrhegmatogenous retinal detachments. Rhegmatogenous retinal detachments (RRDs) result from a break in the retina that allows liquefied vitreous to enter the subretinal space (figure 2). Nonrhegmatogenous detachments are due to exudative processes or tractional forces (figure 1). (See 'Pathophysiology of detachment' above.)
- **Risk factors** – Risk factors for RRDs include posterior vitreous detachment (PVD), older age, prior intraocular surgery, myopia, lattice degeneration, family history of retinal detachment, history of retinal detachment in the other eye, ocular trauma, and congenital connective tissue disorders. Tractional retinal detachments typically arise in patients with diabetes and exudative retinal detachments in patients with inflammatory conditions of the eye. (See 'Epidemiology and risk factors' above.)
- **Clinical presentation** – Patients with retinal detachments present with painless loss of vision in the affected eye. They may also note an increase in floaters or flashes of light. (See 'Clinical presentation' above.)
- **Diagnosis** – The diagnosis of retinal detachment is suspected based on history (eg, sudden onset of floaters, flashes of light [photopsias], and/or loss of vision) and confirmed by ophthalmologic examination. (See 'Diagnosis and evaluation' above.)

- **Primary care evaluation** – Patients with high-risk features (visual field loss, subjective or objective decreased vision, or evidence of vitreous hemorrhage on funduscopic examination [if performed]) should be instructed to decrease eye movement (such as not reading or exercising) and sent urgently to an ophthalmologist or retinal surgeon within one day. (See 'Primary care evaluation' above.)

- **Management of precursor conditions**

  - **PVD** – In the absence of a retinal tear or retinal detachment, no treatment is needed, but close monitoring within four to six weeks is required as the evolution of a PVD may result in new retinal breaks. (See 'Examination and management of posterior vitreous detachment' above.)

  - **Symptomatic retinal breaks** – For patients with a symptomatic retinal break, we suggest treatment with either laser photocoagulation or cryoretinopexy rather than monitoring (**Grade 2C**) to prevent subsequent retinal detachment. (See 'Symptomatic retinal breaks' above.)

  - **Asymptomatic retinal breaks** – In patients with asymptomatic retina breaks, close follow-up is usually observed. However, the presence of lattice degeneration, myopia, aphakia, pseudophakia, or history of retinal detachment in the fellow eye increases the risk of progression to RRD, and these patients may warrant treatment. (See 'Asymptomatic retinal breaks' above.)

- **Management of retinal detachment** – Patients with a symptomatic retinal detachment require treatment as soon as possible (preferably within one to two days), with one or more of the following procedures: pneumatic retinopexy (with either laser or cryoretinopexy), scleral buckle, or vitrectomy. Without treatment, there is a significant risk of permanent vision loss. (See 'Retinal detachment management' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Jorge Arroyo, MD, MPH, who contributed to earlier versions of this topic review.

REFERENCES

1. Berrocal MH, Acaba-Berrocal L. Early pars plana vitrectomy for proliferative diabetic retinopathy: update and review of current literature. Curr Opin Ophthalmol 2021; 32:203.
2. Chapman-Dames A. Shafer's sign and associated retinal tear. Clin Exp Ophthalmol 1993; 76:174.
3. Erie JC, Raecker ME, Baratz KH, et al. Risk of retinal detachment after cataract extraction, 1980-2004: a population-based study. Trans Am Ophthalmol Soc 2006; 104:167.
4. Garweg JG, Tappeiner C, Halberstadt M. Pathophysiology of proliferative vitreoretinopathy in retinal detachment. Surv Ophthalmol 2013; 58:321.
5. Ghazi NG, Green WR. Pathology and pathogenesis of retinal detachment. Eye (Lond) 2002; 16:411.
6. Wolfensberger TJ, Tufail A. Systemic disorders associated with detachment of the neurosensory retina and retinal pigment epithelium. Curr Opin Ophthalmol 2000; 11:455.
7. Gupta MP, Herzlich AA, Sauer T, Chan CC. Retinal Anatomy and Pathology. Dev Ophthalmol 2016; 55:7.
8. Yang CM, Su PY, Yeh PT, Chen MS. Combined rhegmatogenous and traction retinal detachment in proliferative diabetic retinopathy: clinical manifestations and surgical outcome. Can J Ophthalmol 2008; 43:192.
9. Flaxel CJ, Adelman RA, Bailey ST, et al. Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration Preferred Practice Pattern®. Ophthalmology 2020; 127:P146.
10. Mitry D, Charteris DG, Fleck BW, et al. The epidemiology of rhegmatogenous retinal detachment: geographical variation and clinical associations. Br J Ophthalmol 2010; 94:678.
11. Risk factors for idiopathic rhegmatogenous retinal detachment. The Eye Disease Case-Control Study Group. Am J Epidemiol 1993; 137:749.
12. Snead MP, Snead DR, James S, Richards AJ. Clinicopathological changes at the vitreoretinal junction: posterior vitreous detachment. Eye (Lond) 2008; 22:1257.
13. Coffee RE, Westfall AC, Davis GH, et al. Symptomatic posterior vitreous detachment and the incidence of delayed retinal breaks: case series and meta-analysis. Am J Ophthalmol 2007; 144:409.
14. Jindachomthong KK, Cabral H, Subramanian ML, et al. Incidence and Risk Factors for Delayed Retinal Tears after an Acute, Symptomatic Posterior Vitreous Detachment. Ophthalmol Retina 2023; 7:318.
15. Uparkar M, Natarajan S. Late detection of retinal breaks after acute posterior vitreous detachment. Am J Ophthalmol 2008; 145:183; author reply 183.

16. Davis MD. The natural history of retinal breaks without detachment. Trans Am Ophthalmol Soc 1973; 71:343.
17. Shea M, Davis MD, Kamel I. Retinal breaks without detachment, treated and untreated. Mod Probl Ophthalmol 1974; 12:97.
18. Byer NE. What happens to untreated asymptomatic retinal breaks, and are they affected by posterior vitreous detachment? Ophthalmology 1998; 105:1045.
19. Kuhn F, Aylward B. Rhegmatogenous retinal detachment: a reappraisal of its pathophysiology and treatment. Ophthalmic Res 2014; 51:15.
20. Mahabadi N, Al Khalili Y. Neuroanatomy, retina. In: StatPearls [Internet], StatPearls Publishing, 2021.
21. Prasad S, Galetta SL. Approach to the patient with acute monocular visual loss. Neurol Clin Pract 2012; 2:14.
22. Hollands H, Johnson D, Brox AC, et al. Acute-onset floaters and flashes: is this patient at risk for retinal detachment? JAMA 2009; 302:2243.
23. Chauhan DS, Downie JA, Eckstein M, Aylward GW. Failure of prophylactic retinopexy in fellow eyes without a posterior vitreous detachment. Arch Ophthalmol 2006; 124:968.
24. Hildebrand GD, Fielder AR. Anatomy and physiology of the retina. In: Pediatric Retina, Reynolds J, Olitsky S (Eds), Springer, 2011. p.39.
25. Bond-Taylor M, Jakobsson G, Zetterberg M. Posterior vitreous detachment - prevalence of and risk factors for retinal tears. Clin Ophthalmol 2017; 11:1689.
26. Smiddy WE, Flynn HW Jr, Nicholson DH, et al. Results and complications in treated retinal breaks. Am J Ophthalmol 1991; 112:623.
27. Garoon RB, Smiddy WE, Flynn HW Jr. Treated retinal breaks: clinical course and outcomes. Graefes Arch Clin Exp Ophthalmol 2018; 256:1053.
28. Blindbaek S, Grauslund J. Prophylactic treatment of retinal breaks--a systematic review. Acta Ophthalmol 2015; 93:3.
29. Morse PH, Scheie HG. Prophylactic cryoretinopexy of retinal breaks. Arch Ophthalmol 1974; 92:204.
30. Mitry D, Singh J, Yorston D, et al. The fellow eye in retinal detachment: findings from the Scottish Retinal Detachment Study. Br J Ophthalmol 2012; 96:110.
31. Sharma MC, Chan P, Kim RU, Benson WE. Rhegmatogenous retinal detachment in the fellow phakic eyes of patients with pseudophakic rhegmatogenous retinal detachment. Retina 2003; 23:37.

32. Benson WE, Grand MG, Okun E. Aphakic retinal detachment. Management of the fellow eye. Arch Ophthalmol 1975; 93:245.
33. Neffendorf JE, Gupta B, Williamson TH. THE ROLE OF INTRAOCULAR GAS TAMPONADE IN RHEGMATOGENOUS RETINAL DETACHMENT: A Synthesis of the Literature. Retina 2018; 38 Suppl 1:S65.
34. Yannuzzi NA, Li C, Fujino D, et al. Clinical Outcomes of Rhegmatogenous Retinal Detachment Treated With Pneumatic Retinopexy. JAMA Ophthalmol 2021; 139:848.
35. Hilton GF, Tornambe PE. Pneumatic retinopexy. An analysis of intraoperative and postoperative complications. The Retinal Detachment Study Group. Retina 1991; 11:285.
36. Tornambe PE, Hilton GF. Pneumatic retinopexy. A multicenter randomized controlled clinical trial comparing pneumatic retinopexy with scleral buckling. The Retinal Detachment Study Group. Ophthalmology 1989; 96:772.
37. Mulvihill A, Fulcher T, Datta V, Acheson R. Pneumatic retinopexy versus scleral buckling: a randomised controlled trial. Ir J Med Sci 1996; 165:274.
38. Sena DF, Kilian R, Liu SH, et al. Pneumatic retinopexy versus scleral buckle for repairing simple rhegmatogenous retinal detachments. Cochrane Database Syst Rev 2021; 11:CD008350.
39. Ozsaygili C, Duru N. COMPARISON OF INTRAVITREAL DEXAMETHASONE IMPLANT AND AFLIBERCEPT IN PATIENTS WITH TREATMENT-NAIVE DIABETIC MACULAR EDEMA WITH SEROUS RETINAL DETACHMENT. Retina 2020; 40:1044.
40. Ryan EH, Ryan CM, Forbes NJ, et al. Primary Retinal Detachment Outcomes Study Report Number 2: Phakic Retinal Detachment Outcomes. Ophthalmology 2020; 127:1077.
41. Dhoot AS, Popovic MM, Nichani PAH, et al. Pars plana vitrectomy versus scleral buckle: A comprehensive meta-analysis of 15,947 eyes. Surv Ophthalmol 2022; 67:932.
42. Znaor L, Medic A, Binder S, et al. Pars plana vitrectomy versus scleral buckling for repairing simple rhegmatogenous retinal detachments. Cochrane Database Syst Rev 2019; 3:CD009562.
43. Hart RH, Vote BJ, Borthwick JH, et al. Loss of vision caused by expansion of intraocular perfluoropropane (C(3)F(8)) gas during nitrous oxide anesthesia. Am J Ophthalmol 2002; 134:761.

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