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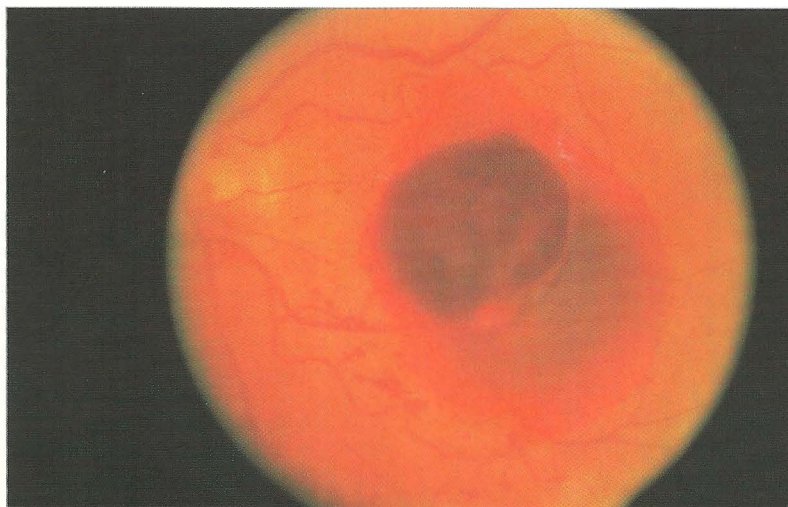
CLINICAL MODULES FOR OPHTHALMOLOGISTS

VOLUME XVIII NUMBER 10

DECEMBER 2000 (SECTION 1 OF 3)

Diagnosis and Management of Vitreous Hemorrhage

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Diagnosis and Management of Vitreous Hemorrhage

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CLINICIAN'S CORNER

Cover: Figure 4. This yellow retinal macroaneurysm has ruptured producing both a subretinal and preretinal hemorrhage.

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Focal Points (ISSN 0891-8260) is published quarterly by The Foundation of The American Academy of Ophthalmology, 655 Beach St., San Francisco, CA 94109-1336. Yearly subscriptions are \$125 for Academy members and \$175 for nonmembers. Periodicals postage paid at San Francisco, CA, and additional mailing offices. POSTMASTER: Send address changes to *Focal Points*, P.O. Box 7424, San Francisco, CA 94120-7424.

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Learning Objectives

The study of this *Focal Points* module will help the reader to:

- Describe the evolution of phacoemulsification
- Compare current techniques of incision, anesthesia, and nuclear removal

Key Words: macroaneurysm, neovascularization, retinal detachment, retinal tear, retinopathy, vitrectomy, vitreous hemorrhage

Introduction

The sudden or rapidly progressive loss of vision from a vitreous hemorrhage may be a frightening and visually disabling event for an individual. The vitreous hemorrhage, although visually compromising, actually serves as a symptom of an underlying disease process. The critical issue is the underlying condition that is responsible for the vitreous blood and decreased vision. A vitreous hemorrhage may clear spontaneously or it may be removed surgically. However, if the underlying cause of the hemorrhage is ignored, there may be irreversible blindness, such as from an untreated retinal detachment.

An appropriate evaluation including history, examination, and possibly B-scan ultrasonography should be undertaken in order to determine the underlying cause, and a management strategy should be initiated. Finally, the patient must be educated and reassured.

Pathogenesis

The retina and choroid are highly vascularized structures, and there are numerous disorders that may produce bleeding into the vitreous cavity. These various disorders can be placed in four broad categories according to the pathogenesis of the vitreous hemorrhage.

Neovascularization of Retina and Disc

As a result of ischemic damage, the retina may undergo an aberrant form of “repair” in which new vessels proliferate on the surface of the retina or optic nerve. These new vessels are fragile and are particularly likely to rupture and bleed from vitreous traction. Although diabetes mellitus is the most common cause

of retinal neovascularization, other causes include retinal vein occlusions, sickle cell disease, and inflammatory diseases that produce an occlusive vasculitis.

In this category of retinal ischemia as a precursor to vitreous hemorrhage, it is important to remember that neovascularization of the iris and angle can be seen regardless of the underlying disease. This can lead to neovascular glaucoma, which can occur very rapidly, with devastating consequences.

Rupture of a Normal Retinal Vessel

Although not as likely to bleed as vessels generated by neovascularization, normal retinal vessels are susceptible to rupture from both external and internal forces. A vigorous tug on the retina and its vasculature, either from a spontaneous posterior vitreous detachment (PVD) or from blunt trauma to the eye, may result in a vitreous hemorrhage. This hemorrhage may occur with or without a retinal tear.

A strong and sudden surge in retinal venous pressure, as seen with a subarachnoid or subdural hemorrhage, may result in retinal and vitreous bleeding. This condition, termed *Terson syndrome*, occurs in approximately 20% of patients with subarachnoid hemorrhages. Hyperviscosity syndromes and central retinal vein occlusions may also rupture normal retinal vessels, producing a vitreous hemorrhage.

Diseased Retinal Vessels

A macroaneurysm of a retinal arteriole, which is most often associated with systemic hypertension, is an example of a vascular abnormality that may result in a vitreous hemorrhage.

Extension Through the Retina

A subretinal or choroidal lesion may lead to a vitreous hemorrhage as the blood may dissect through the retina into the vitreous cavity without an associated retinal detachment. Choroidal neovascularization in age-related macular degeneration (ARMD) is usually responsible for this phenomenon, but it can also be seen with a choroidal melanoma. In cases of trauma, a subretinal hemorrhage may break through the retina and create a vitreous hemorrhage.

Clinical Manifestations

History

A carefully documented history is very important in establishing a differential diagnosis. If a patient reports

that the loss of vision was preceded by the sudden onset of floaters and flashing lights, then one should consider the possibility of a posterior vitreous detachment and look for an associated retinal tear.

Documentation of the patient's medical history may be of help in determining the underlying etiology of the vitreous hemorrhage. For example, in a patient with diabetes, one must suspect proliferative retinopathy. In a patient with hypertension, a retinal vein occlusion or a macroaneurysm should be considered as a possible etiology of vitreous bleeding. If the patient lost vision following a stroke, then Terson syndrome should be considered. A history of leukemia may indicate hyperviscosity syndrome or an associated thrombocytopenia. In appropriate patients it is important to inquire about the possibility of sickle cell disease. The patient's systemic medications should be reviewed. In ARMD or in a patient with a posterior vitreous detachment, warfarin sodium (Coumadin) may be implicated as a factor in the production of a vitreous hemorrhage.

The patient's ocular history may also be significant in establishing an etiology of a vitreous hemorrhage. Posterior vitreous detachment with a retinal tear is more common in high myopia, and the relationship between cataract surgery or YAG capsulotomy and retinal detachment is well known, particularly when there is vitreous loss at the time of cataract surgery. Patients with glaucoma are at higher risk for a retinal vein occlusion. Some uveitic syndromes such as pars planitis, Behçet's disease, and sarcoidosis may have a proliferative retinopathy secondary to retinal ischemia from an occlusive vasculitis.

The age of a patient may be useful in formulating a differential diagnosis of a vitreous hemorrhage. In infants, one must consider trauma from shaken-baby syndrome, while elderly patients are more predisposed to macular degeneration, retinal vein occlusion, and macroaneurysms. Congenital retinoschisis is a cause of vitreous hemorrhage in young males, and there is often a positive family history.

Ocular Examination

The vision should be recorded to document the degree of visual disability and to provide a reference for future follow-up exams. It is important to perform tonometry. A low intraocular pressure (IOP) may indicate a retinal detachment, while a high IOP may suggest ghost cell or neovascular glaucoma.

Slit-lamp biomicroscopy should be performed on every patient with a vitreous hemorrhage. Examine the anterior segment for signs of inflammation such as keratoprecipitates and "cells and flare." Khaki-colored cells are a feature of ghost cell glaucoma. Study the iris before dilation for signs of neovascularization and check for a Marcus Gunn pupil. The

anterior vitreous should be evaluated, not only for red blood cells, but also pigmented cells, which may indicate a retinal tear or detachment.

The fundus examination is usually performed with indirect ophthalmoscopy. Even in the case of a fairly dense vitreous hemorrhage, the far peripheral retina often can be seen with scleral depression (especially superiorly, where tears are more likely to occur). Many times, a treatable retinal break can be seen. In some cases, a preretinal blood clot and some vascular abnormalities can indicate a branch retinal vein occlusion. In cases of mild vitreous hemorrhages, the posterior pole can be studied with the slit lamp and a noncontact fundus lens. Look for a posterior vitreous detachment. If the hemorrhage is predominantly "boat shaped," or subhyaloid, consider proliferative diabetic retinopathy, a macroaneurysm, macular degeneration, Terson syndrome, or Valsalva retinopathy, which are not associated with an acute PVD.

A vitreous hemorrhage may be documented by the amount of retinal detail that can be visualized on ophthalmoscopy, and a grading system may be utilized as was developed for the Diabetic Retinopathy Vitrectomy Study. In a mild hemorrhage, the retinal vessels can be seen, while in a moderate hemorrhage, only the optic nerve can be identified. In a severe hemorrhage, no retinal details can be visualized.

Diagnostic Studies

B-Scan Ultrasound

Ultrasonography with the B-scan mode is probably the most important study performed in the evaluation of a patient with a vitreous hemorrhage (see Figure 1). This study can be used to look for a number of conditions, including a PVD, retinal detachment, giant retinal tear, choroidal tumor, and choroidal hemorrhage. Standardized echography can often be helpful in both the identification and localization of a retinal tear associated with a vitreous hemorrhage. In addition, the density of the hemorrhage can be ascertained, which may allow one to estimate the rate of spontaneous clearing.

Other Diagnostic Studies

Additional diagnostic studies may be ordered on the basis of specific clinical presentations. For example, a hemoglobin electrophoresis is indicated if sickle cell retinopathy is suspected. In the patient with possible ocular ischemic syndrome, a noninvasive carotid Doppler study should be ordered. In mild vitreous hemorrhages, fluorescein angiography or angiography

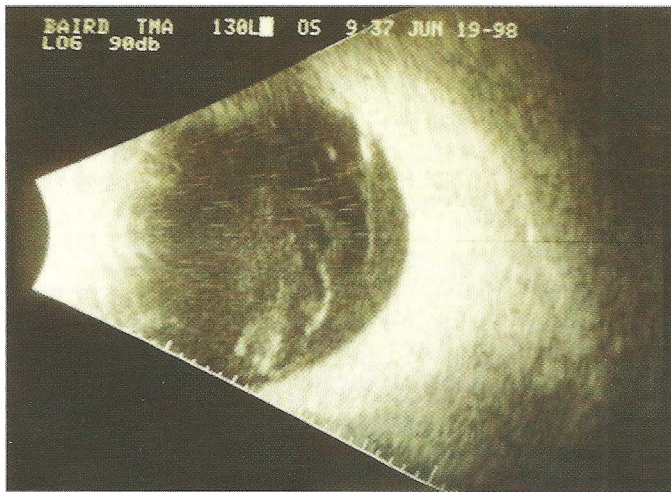


Figure 1. B-scan ultrasound shows a dense vitreous hemorrhage with a detachment of the posterior hyaloid.

may make it possible to find abnormalities in the retinal vasculature.

Differential Diagnosis

Causes of Vitreous Hemorrhages

A review of six studies indicated that five conditions account for 75–90% of all vitreous hemorrhages: proliferative diabetic retinopathy, posterior vitreous detachment with retinal tear, posterior vitreous detachment without retinal tear, proliferative sickle retinopathy, and retinal vein occlusion (see Table 1). Less frequent causes of vitreous hemorrhage are listed in Table 2.

Conditions That Simulate Vitreous Hemorrhage

There are other conditions in which vitreous opacities are present, including asteroid hyalosis, uveitis (vitritis), ocular or systemic lymphoma, and amyloidosis of the vitreous. These opacities must be distinguished from vitreous blood. This is best done while examining the anterior vitreous with the slit-lamp biomicroscope. Small cells with reddish pigmentation are characteristic of vitreous blood. In older vitreous hemorrhages, the cells lose their hemoglobin and take on a grayish-white color. Bright yellow or white bodies of various sizes are seen in asteroid hyalosis, while lymphoma cells are white and are much larger in appearance than red blood cells. Patients with a posterior uveitis will have white cells in the vitreous. These can be seen in the anterior vitreous with a slit lamp and in the posterior vitreous with a fundus lens. Look for snowbanks in the inferior periphery. The

Table 1. The Five Leading Causes of Vitreous Hemorrhage*

Proliferative diabetic retinopathy	38.6 %
Posterior vitreous detachment with retinal tear	24.6 %
Retinal vein occlusion	9.1 %
Posterior vitreous detachment without tear	7.9 %
Proliferative sickle retinopathy	2.1 %

*Compilation of four studies; see Suggested Reading 2, 5, 14, 18.

Table 2. Other Causes of Vitreous Hemorrhage

Neovascularization of Retina and Disc:

branch and central retinal artery occlusion

branch and central retinal vein occlusion

Eales disease

inflammatory diseases with occlusive vasculitis:

Behçet disease, multiple sclerosis with retinal

vasculitis, pars planitis, retinal vasculitis, sarcoid

uveitis, syphilitic retinitis, systemic lupus

erythematousus

ocular ischemic syndrome

radiation retinopathy

retinitis pigmentosa

retinopathy of prematurity

rhegmatogenous retinal detachment (chronic)

senile bullous retinoschisis

sickle cell disease

talc retinopathy

Rupture of a Normal Retinal Vessel:

anticoagulant therapy

avulsed retinal vessel syndrome

blunt or penetrating trauma

bridging retinal vessel across retinal tear

posterior vitreous detachment

hyperviscosity syndromes

Terson syndrome

Valsalva retinopathy

Diseased Retinal Vessels:

arteriovenous malformation of the retina

Coats disease

hypertensive retinopathy

juvenile retinoschisis (x-linked)

macroaneurysm

persistent hyaloid artery

retinal astrocytic hamartoma

retinoblastoma

retinal angioma

venous stasis retinopathy

Extension Through the Retina

age-related macular degeneration

choroidal melanoma

intrusion of scleral buckle

multiple hemorrhagic retinal pigment

epithelium (RPE) detachments (posterior

uveal bleeding syndrome)

presence of sheathed retinal vessels is indicative of an associated vasculitis. In amyloidosis, the vitreous contains long fibers that resemble glass wool.

Natural History of Vitreous Hemorrhage

A hemorrhage that is trapped or clotted within the vitreous gel reabsorbs extremely slowly. However, once the gel undergoes syneresis and begins to liquefy, the lysed blood can be efficiently reabsorbed. Therefore, vitreous hemorrhages clear from the eye at varying rates, depending on the extent of the blood and the status of the vitreous gel. For example, a hemorrhage occurring in a relatively young patient with no vitreous liquefaction may persist for years, while a hemorrhage in a vitrectomized eye may clear in a few weeks. The lens status may also have some bearing on the rate of spontaneous resolution of a vitreous hemorrhage. There is a greater chance of vitreous liquefaction in the aphakic eye. Furthermore, there may be some movement of the blood through the anterior chamber and trabecular meshwork.

Management: When to Consider a Vitrectomy

The crucial question is, when is it appropriate to recommend vitrectomy to surgically remove a hemorrhage? The answer requires that a number of factors be considered so that the potential risks and benefits of surgery are weighed against the risks of observation alone. If a proliferative retinopathy is the underlying cause of the hemorrhage, and the ischemic retina has not been treated with scatter laser photocoagulation, then the risk of traction retinal detachment and neovascular glaucoma is greater than the risk of surgery. On the other hand, in a patient with Terson syndrome and a unilateral vitreous hemorrhage, the risks of vitrectomy—which include retinal detachment, endophthalmitis, and cataract—are greater than the risk of observation, since these hemorrhages tend to clear spontaneously with time.

In most cases in which the underlying cause of the vitreous hemorrhage does not threaten the health of the eye, and the fellow eye has good functional vision, observation is safe and reasonable. Hemoideroidosis bulbi is the only potential risk of observation, and it is extremely rare. There are reports in the literature of a very rare toxic retinal damage occur-

ring from chronic breakdown products of red blood cells. However, patients with Terson syndrome, trauma, and other causes only rarely exhibit this toxic effect from vitreous hemorrhage.

The specific management of the patient presenting with a vitreous hemorrhage will vary with the underlying cause of the bleeding (see Table 3). In most cases, a fairly accurate diagnosis can be made after taking a careful history and performing an eye examination with ultrasonography. If an etiology of the hemorrhage cannot be established, then the patient should be followed carefully, perhaps weekly, with serial ultrasound to look for a possible retinal detachment. Elevation of the head may hasten settling of the blood (at least temporarily), allowing improved visualization of the retina. This may aid in making a diagnosis. If, after several months, the hemorrhage has failed to clear and there is good visual potential, then a vitrectomy may be considered.

Proliferative Diabetic Retinopathy

Traditionally, elevation of the head at night has been recommended to allow for settling of the blood. This can be reassuring to the patient, because the vision is usually clearer in the morning upon awakening. However, with the movement of normal activities, the hemorrhage once again diffuses and the vision is reduced. Strict enforcement of positioning with elevation of the head is somewhat controversial. Some experts find head elevation extremely useful, while others feel that its effects are only temporary, and that it does not facilitate clearing of the vitreous blood.

Many patients are cautioned to avoid exercise while the vitreous hemorrhage reabsorbs. However, reasonable exercise is unlikely to exacerbate vitreous bleeding. Therefore, one must balance the potential benefit from restricting activity with the possible side effects on blood sugar control and the long-term health of the diabetic patient.

When a vitreous hemorrhage occurs in a diabetic who has not had previous panretinal photocoagulation (PRP), there is reason for concern. This is particularly the case if the patient has type I or juvenile-onset diabetes and no evidence of a PVD. These patients are at great risk for progression of their proliferative disease and may develop a traction retinal detachment or even neovascular glaucoma. Observation of the hemorrhage for spontaneous clearing may jeopardize the potential for vision. If the patient is phakic and there has been no improvement after 2 or 3 months, I recommend vitrectomy and intraoperative laser (PRP applied via endolaser or indirect ophthalmoscopic laser delivery system). Pseudo phakic or aphakic eyes are at even higher risk of iris neovascu-

Table 3. Management of Vitreous Hemorrhage

Proliferative Diabetic Retinopathy:

- previous PRP
 - continued observation
 - or
 - PPV in 3+ months
- no previous PRP
 - phakic: PPV, laser in 2–3 months
 - aphakic or pseudophakic: PPV, laser in 1 month
- large subhyaloid hemorrhage obscuring macula
 - PRP first, if retinal periphery visible
 - PPV, laser in 1 month
- neovascularization of iris with/without glaucoma
 - immediate PPV
- traction retinal detachment with or without rhegmatogenous retinal detachment on ultrasound
 - immediate PPV
- type I diabetes mellitus with vision 5/200 or worse
 - PPV, laser at 1 month

Posterior Vitreous Detachment:

- adequate view
 - scleral depress for retinal break or detachment
- poor view
 - bilateral patch, head elevation, strict bed rest overnight
 - or
 - observe weekly with serial ultrasound

Retinal Vein Occlusion:

- CRVO
 - adequate view
 - PRP within 1 week, high risk of neovascular glaucoma
 - poor view
 - good visual potential: PPV and laser
 - poor visual potential: retinal cryoablation, cyclophotocoagulation
- BRVO
 - adequate view
 - scatter laser in ischemic sector
 - poor view
 - continued observation
 - PPV, laser after 3 months
- Sickle Cell Retinopathy:
 - adequate view
 - peripheral scatter laser
 - poor view
 - peripheral cryotherapy vs PPV

larization and glaucoma. Vitrectomy with intraoperative laser should be performed in this group if there has been no clearing of the blood after 1 month.

An eye with proliferative diabetic retinopathy and an associated artery or vein occlusion or carotid artery disease is also at very high risk for neovascular glaucoma. Vitrectomy with laser should be performed in this group if there has been no clearing after 1 month.

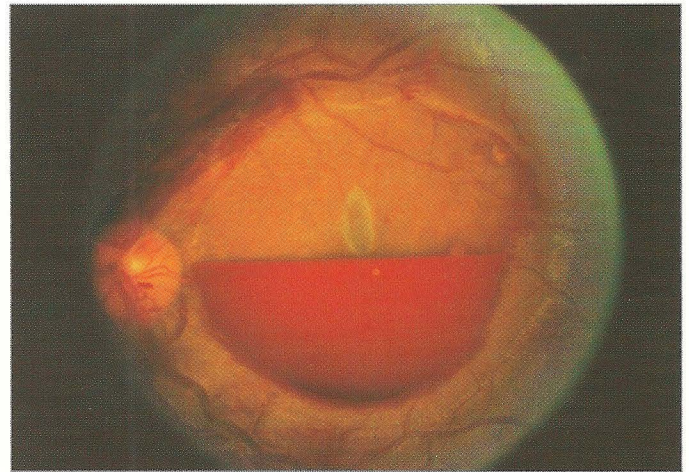


Figure 2. A subhyaloid, or boat-shaped, vitreous hemorrhage is often seen in patients with proliferative diabetic retinopathy and an attached posterior hyaloid.

If adequate PRP has already been performed prior to the vitreous hemorrhage, there is less urgency for vitrectomy. Of course, the degree of urgency depends on whether the underlying neovascularization has regressed. The proliferative process very rarely progresses further after a completed PRP. Thus, one can usually wait for spontaneous clearing of the blood without concern that the retina will undergo further damage. Alternatively, a vitrectomy can be scheduled at the patient's request to restore vision. It is reasonable to wait approximately 3 months for reabsorption of the blood before preceding to surgery.

The subhyaloid, or boat-shaped, hemorrhage deserves special mention (see Figure 2). Regardless of whether the patient has previously received a PRP, these should be surgically evacuated after a brief period of observation of perhaps one month. Untreated, these hemorrhages undergo fibrosis and become adherent to the retina. Once these clotted hemorrhages adhere to the retina, they can be very difficult to remove without traumatizing the retina and may cause permanent retinal damage.

Posterior Vitreous Detachment

One study reported that retinal tears are seen in approximately 70% of patients with an acute PVD and associated vitreous hemorrhage, as compared to only 2–4% of patients with acute PVD but without vitreous hemorrhage. It is critical to carefully search for a retinal tear with scleral depression in the face of an acute PVD with vitreous hemorrhage. If visualization of the fundus is impaired by the hazy view, then the patient can be admitted to the hospital for strict bedrest, with head elevation and bilateral patching. It is useful to provide mild sedation for the patient as this is a tedious routine. After a night of patching, the

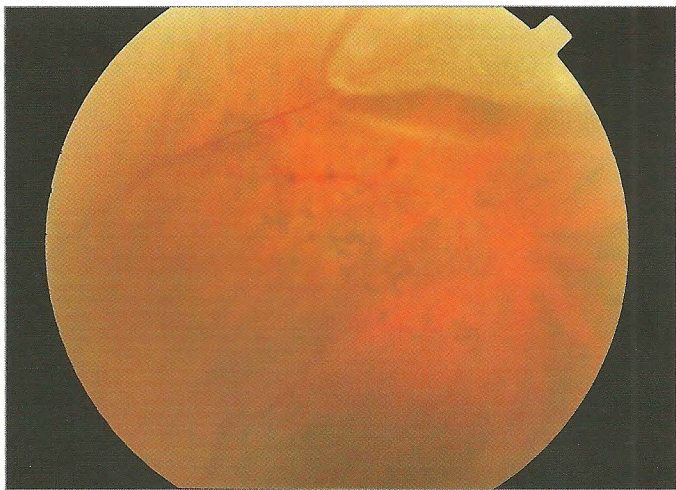


Figure 3. Persistent traction on a bridging or avulsed retinal vessel may result in a vitreous hemorrhage.

vitreous often has sufficiently cleared to permit adequate examination of the retina. Although there are some limitations to examining a patient and treating a retinal tear with cryopexy, or “cryo,” at the bedside, it may be advantageous because any patient movement can stir up the blood and once again cloud the view. Alternatively, patients can be sent home with bilateral patches to be removed by the retinal surgeon in the examination room, with cryo or laser readily available.

Another option is to follow the patient in the office, perhaps every 1–2 weeks, with serial ultrasound to rule out a retinal detachment, and wait until the hemorrhage clears. It is important to educate the patient about symptoms of a retinal detachment (flashing lights and loss of visual field). In the absence of a satisfactory view of the periphery, early vitrectomy can be considered.

Bridging Retinal Vessels

Following a PVD, vitreous adhesions to retinal vessels may result in a vitreous hemorrhage, with or without the association of a retinal tear (see Figure 3). This is also known as *avulsed retinal vessel syndrome*. As in any case of vitreous hemorrhage, the retina must be carefully evaluated for a retinal tear. In spite of appropriate cryo or laser therapy of the retinal break, persistent vitreous traction on the bridging vessel may produce recurrent vitreous hemorrhages. When this occurs, the avulsed retinal vessel may be directly treated and closed with laser photocoagulation. Occasionally, a vitrectomy is required to eliminate the traction on the avulsed vessel.

Retinal Detachment

A retinal detachment may be diagnosed by either ophthalmoscopy or ultrasonography, depending on the density of the hemorrhage. Surgical repair of the detachment is mandatory. If the retinal break(s) cannot be identified through the vitreous haze, then a vitrectomy must be included as part of the procedure.

Retinal Vein Occlusion

Central retinal vein occlusion (CRVO) does not commonly result in a vitreous hemorrhage. However, an occurrence should be considered a sign of retinal ischemia, and panretinal photocoagulation should be performed. The PRP should be done without delay because these eyes are at high risk for neovascular glaucoma. If the density of the hemorrhage does not permit visualization of the retina, a vitrectomy with laser should be considered. If the potential for vision is very poor, panretinal cryoablation can be considered, but it is rarely used today. If neovascular glaucoma is already present, many of these eyes may be treated primarily with diode cyclophotocoagulation.

Vitreous hemorrhage is more commonly associated with branch retinal vein occlusion (BRVO). Because BRVO presents much less risk of neovascular glaucoma (with the exception of hemiretinal vein occlusions, which behave similarly to CRVO and increase potential for retinal ischemia), one can wait for spontaneous clearing of the blood before performing laser photocoagulation. Of course, after a few months there may be enough patient frustration with the poor vision to warrant vitrectomy and laser. These patients must also be followed at regular intervals for evidence of iris neovascularization. Any sign of neovascularization is an indication for vitrectomy and laser photocoagulation.

Macroaneurysm

A retinal arteriolar macroaneurysm may bleed massively into the retina. Because of the compactness of the retina, the blood may be forced either into the vitreous cavity or into the subretinal space, or it may be pushed into both locations (see Figure 4, cover). When bleeding occurs in the vitreous alone, it often undergoes spontaneous resolution. If the blood fails to clear after a few months, then vitreous surgery may be advised. Surgical evacuation of a subretinal hemorrhage is controversial. Vitreous surgical techniques may be applied to evacuate submacular blood with the assistance of tissue plasminogen activator (tPA) to lyse the clot. However, vision may also recover quite nicely following spontaneous resolution of the blood. A third treatment option is the intravitre-

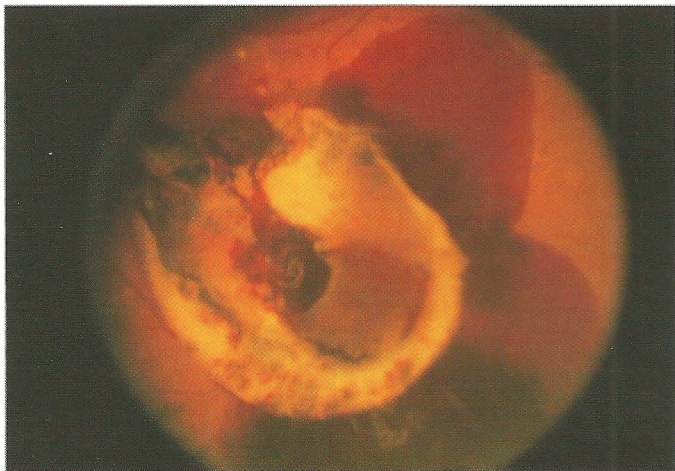


Figure 5. Age-related macular degeneration with disciform scar and subhyaloid hemorrhage.

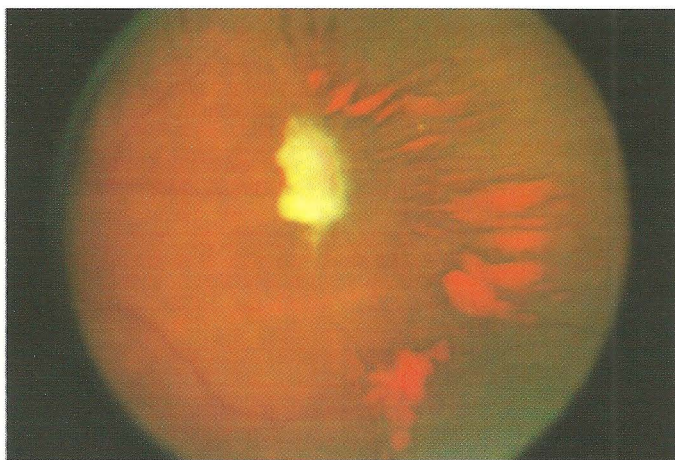


Figure 6. In this case of sickle cell retinopathy, a peripheral sea fan is bordered by preretinal or vitreous hemorrhage.

al injection of tPA and an air bubble with prone positioning to lyse and displace the blood clot.

Terson Syndrome

A subarachnoid hemorrhage may produce an acute obstruction of venous outflow from the eye, resulting in rupture of retinal capillaries. Typically, the bleeding occurs within the potential space beneath the internal limiting membrane (ILM). This produces a dome-shaped elevation of the membrane. The blood may also extend into the vitreous cavity. The hemorrhage in these cases usually resolves spontaneously, but often requires an extended amount of time. Vitreous surgery is indicated for the removal of vitreous and sub-ILM hemorrhage in the following cases: children with immature visual systems, to prevent amblyopia; bilateral cases; and unilateral cases that are persistent.

Breakthrough Vitreous Hemorrhage from Disciform Scar

Rarely, in cases of ARMD, a disciform scar may bleed into the vitreous (see Figure 5). Interestingly, the blood tracks through the retina without causing a break or detachment. When this occurs, the resulting vitreous hemorrhage may be quite dense. Prior to the introduction of improved diagnostic techniques, such as indirect ophthalmoscopy and ultrasound, large hemorrhagic disciform scars were often mistaken for choroidal melanoma. These vitreous hemorrhages usually resolve spontaneously and can be observed. However, if the hemorrhage occurs in the better seeing eye, a vitrectomy to restore peripheral vision may be indicated.

Peripheral Retinal Neovascularization

Peripheral retinal neovascularization can have a number of causes, including sickle cell retinopathy (see Figure 6). All of these causative conditions can produce a vitreous hemorrhage. Fortunately, the hemorrhage is usually mild in these cases, and vitrectomy is not usually necessary. However, it is imperative that the ischemic peripheral retina be treated with scatter laser photocoagulation. Because of the far peripheral location of the ischemic retina, use of the laser indirect ophthalmoscope facilitates treatment. Alternatively, if the vitreous blood does not permit visualization of the retina, cryotherapy can be applied externally. In general, it is best to avoid surgery in patients with sickle cell disease; they are at higher risk for medical and ocular complications during surgery. Nevertheless, on occasion, vitreous surgery may be necessary to remove a non-clearing hemorrhage in these patients.

Ghost Cell Glaucoma

A chronic or old vitreous hemorrhage has a white, milky appearance because of the loss of hemoglobin from the red blood cells. The remaining cell membranes are white or khaki colored and are called *ghost cells*. When these ghost cells gain access to the anterior chamber, they clog the trabecular meshwork, and may result in elevation of the intraocular pressure. Ghost cell glaucoma is more of a concern in patients who have had previous ocular surgery, where the anterior hyaloid is no longer intact, thus allowing the ghost cells to travel more freely into the anterior chamber. In most cases, ghost cell glaucoma can be treated medically with topical hypotensive agents until the vitreous hemorrhage has totally dissipated. If the intraocular pressure remains uncontrolled, then

a vitrectomy must be performed to remove the old hemorrhage and the source of the ghost cells.

Other Therapies

Laser Posterior Hyaloidotomy

There has been some interest in treating subhyaloid (boat-shaped) hemorrhages and subinternal limiting membrane hemorrhages by using the Nd:YAG laser to create a hole in the overlying membrane. This allows the lysed blood to flow into the vitreous cavity, where it can be reabsorbed, thus obviating the need for a vitrectomy. However, the downside is that the blood may remain in the vitreous for a while before it reabsorbs. In addition, the posterior hyaloid remains attached to the retina. There is the potential that the hyaloid will contract, resulting in traction or even a rhegmatogenous retinal detachment. Because, with time, the hemorrhage may clear spontaneously without intervention, the precise role for YAG hyaloidotomy has yet to be established.

Cryotherapy of the Peripheral Retina

In patients with vitreous hemorrhage and iris neovascularization, a peripheral cryoablation of the retina can be performed. This has been called a “blind” cryo treatment, because the hemorrhage precludes direct visualization of retina whitening. The probe is applied sequentially around the globe, allowing an iceball to form on the sclera, before it is moved to the next location. Usually three rows of treatment are placed in each meridian, with 3–4 meridians treated in each quadrant. This treatment usually successfully induces a regression of the iris neovascularization. If the vitreous hemorrhage does not clear spontaneously, then a vitrectomy can be performed at a later date when there is limited risk of developing neovascular glaucoma.

This particular treatment strategy is most useful in eyes that have no or very little visual potential. In eyes with a better visual prognosis, I prefer a vitrectomy with panretinal photocoagulation to induce regression of the iris neovascularization. Cryotherapy is much

more inflammatory than laser photocoagulation. It may promote the production of preretinal fibrin membranes and result in a traction retinal detachment.

Pharmacologic Vitreolysis

Ideally, liquefaction of the vitreous gel (syneresis) could be achieved by medical or pharmacologic means, as could the creation of a vitreous separation from the retina. The prospect of “chemical” vitrectomy is very exciting, with its promise of a nonsurgical method for clearing a vitreous hemorrhage. Currently, the enzymes plasmin, dispase, chondroitinase, and hyaluronidase are under clinical investigation. It is unclear whether any of these agents will effectively eliminate the need for a surgical vitrectomy or whether they will be used as adjuvants to facilitate vitreous surgery. We can look forward to the results of these studies and perhaps to the introduction of new agents with the potential of increased efficacy and safety.

Conclusion

The sudden loss of vision from a vitreous hemorrhage is a source of great concern to both the patient and ophthalmologist. Depending on the etiology of the hemorrhage, the visual loss may be considered a temporary nuisance or it may mask an underlying disease that has sight- or even life-threatening consequences. After a careful patient history, ocular examination, and ultrasonography, a differential diagnosis can be formulated and a strategy for management pursued.

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The Clinician's Corner section is intended to provide additional viewpoints on the subject covered in this issue of Focal Points. Consultants have been invited by the Editorial Review Board to respond to questions posed by the Academy's Practicing Ophthalmologists Advisory Committee for Education. While the advisory committee reviews the modules, consultants respond without reading the module or one another's responses.—Ed.

1. When should gonioscopy be performed in the patient with vitreous hemorrhage?

Dr. Marcus: In practice, I do not routinely perform gonioscopy to rule out neovascularization of the angle in every case of vitreous hemorrhage.

Each case must of course be taken individually. On all new patients with a vitreous hemorrhage, I routinely check the intraocular pressure (IOP) and perform slit-lamp biomicroscopy of the anterior chamber to assess for iris neovascularization. If there is elevated IOP or iris neovascularization, then gonioscopy is required, regardless of the suspected cause. Gonioscopy may be considered in cases of vitreous hemorrhage when a proliferative retinopathy, such as diabetic retinopathy, ocular ischemic syndrome, or retinal venous occlusive disease, is suspected. Gonioscopy is not generally required for vitreous hemorrhage having nonproliferative causes, such as posterior vitreous detachment, retinal tears, or detachment. Gonioscopy should be considered after clearing of a traumatic vitreous hemorrhage or hyphema to rule out angle recession.

Dr. Thomas: Gonioscopy in a patient with a spontaneous vitreous hemorrhage should be performed in both eyes if the clinical scenario permits. Infants and uncooperative adults may make this impossible. A contact lens may often be necessary for examination of the peripheral retina. The anterior viewing lens is readily available for viewing the angle at the same time. Examination of the angle architecture can yield evidence of traumatic iridodialysis, recession, cyclodialysis, or angle neovascularization.

2. How do you manage the patient with neovascular glaucoma and a dense vitreous hemorrhage?

Dr. Marcus: I consider a differential diagnosis: proliferative diabetic retinopathy, posterior vitreous detachment, retinal venous occlusive disease, ocular ischemic syndrome, chronic retinal detachment, and other less likely entities such as uveal melanoma, retinal artery occlusion, and uveitis. Echography is performed to rule out tumor and retinal detachment. The patient's risk factors and history may dictate a nonemergent workup for diabetes, hypertension, and carotid occlusive disease. The majority of cases are caused by a proliferative retinopathy, and emergent or

urgent treatment is indicated. In general, for presumed proliferative retinopathies, I perform "blind" 360° retinal cryopexy without conjunctival cutdown under retrobulbar anesthesia for dense vitreous hemorrhages (measuring 9,11,13 mm from the limbus in each quadrant). Patients given topical cycloplegics, steroids, and multiple IOP-lowering agents (e.g., beta-blocker, alpha-2 agonist, and carbonic anhydrase inhibitor). I avoid pilocarpine and prostaglandin topical agents in this setting so as not to exacerbate the inflammatory component of neovascular glaucoma. The need for IOP-lowering medication or glaucoma surgery is then decided on an individual basis. Cyclophotocoagulation or cyclocryotherapy should, in general, be considered only in eyes with a known poor visual prognosis. These decisions are based on follow-up IOP, gonioscopy, and the previous status of the optic nerve. Neovascular glaucoma in this setting should be treated aggressively. Consideration for additional retinal cryopexy with or without conjunctival cutdown, laser retinopexy with clearing of the vitreous hemorrhage, or vitrectomy with endolaser/cryopexy should be based on the status of the vitreous hemorrhage, IOP, gonioscopy findings, status of the optic nerve, visual potential (if known), and etiology.

Dr. Thomas: In an eye with neovascular glaucoma, the etiology of the neovascularization and hemor-

Clinician's Corner



rhage is critical in planning a course of treatment. When there is no visual potential and if neovascular glaucoma can be determined by clinical evidence prior to the onset of hemorrhage, palliative procedures to control discomfort from elevated IOP should be considered. When there is visual potential, aggressive early intervention may salvage potential vision. The underlying pathology can be treated surgically with pars plana vitrectomy to clear the vitreous hemorrhage and anterior segment opacities, including cataract, pupillary membranes, and opacified capsules after intraocular lens implants. Use of wide-field panoramic viewing systems has improved our visualization in eyes with anterior segment opacities and smaller pupils. Use of iris retractors, however, and lensectomies may still be necessary to manage the retinal pathology found at surgery. It is important to consider simultaneous glaucoma seton placement or cyclophotocoagulation as part of preoperative planning. Treating the underlying pathology is essential to removing the stimulus for neovascularization and hemorrhage. Such treatment may involve removing pre-retinal membranes, eliminating traction and rhegmatogenous components of the pathology, if found, and performing panretinal laser photocoagulation to reduce neovascularization of the anterior segment.

3. What are the common causes of vitreous hemorrhage in a 2-year-old? How long do you wait to perform vitreous surgery, given the risk of amblyopia? What challenges might one expect with vitrectomy surgery in such a setting?

Dr. Marcus: Common causes include trauma (accidental or non-accidental), Terson syndrome, or previous proliferative retinopathy, such as retinopathy of prematurity (ROP) and familial exudative vitreoretinopathy (FEVR). In a 2-year-old, I would certainly wait at least 2 weeks for clearing of the vitreous hemorrhage. Density and vision (if measurable) determine whether vitrectomy to prevent amblyopia is considered. If the hemorrhage progressively clears, a conservative approach is taken unless amblyopia is present already. Patching the uninvolved eye is decided in consultation with a pediatric ophthalmologist. Challenges with vitrectomy surgery in this age

group include a nonliquefied vitreous, postoperative cataract, and need for general anesthesia.

Dr. Thomas: The role of the ophthalmologist should be one of looking at the whole patient, not just the eye, to determine the source of hemorrhage. Historical information, especially family history, is important to know. Trauma should be considered first in an otherwise healthy 2-year-old child. Many times, there are other signs of external trauma in the face and neck and other parts of the body. However, overdiagnosis of child endangerment must be avoided, given the array of other potential causes such as ROP with contraction of the peripheral fibrovascular tissue, juvenile retinoschisis with peripheral neovascularization, and other unusual retinopathies, including leukemia. Intervention with vitrectomy should be considered within a few weeks of onset once the pathology has been elucidated. Anatomically, before age 7, the eye does not have a definable pars plana, and a more anterior approach through the pars ciliaris is necessary. There is a high risk of cataract and need for intraoperative lensectomy. Maintaining the phakic status of the eye has, in my experience, been very difficult. Amblyopia is a problem if you do not operate early (less than 1 month after detection) and if you create surgical aphakia which needs to be corrected postoperatively. These complications may be significantly reduced in the future by the use of enzymatic vitreous dissolution, obviating the need for urgent surgical intervention.

4. How do you manage a diabetic patient with a large subhyaloid hemorrhage?

Dr. Marcus: Options include observation, YAG laser hyaloidotomy, or vitrectomy. Based on the literature (which is mixed and controversial), I observe patients with foveal sparing and watch them closely (every 2 or 3 weeks) after panretinal ablation. If the hemorrhage increases or the fovea is involved, I prefer vitrectomy surgery in a timely fashion. In patients with a large subhyaloid hemorrhage involving the fovea, I might observe closely for clearing, but in general perform vitrectomy within a few weeks or sooner of diagnosis. While YAG laser hyaloidotomy is an alternative, many of these patients still require vitrectomy. All patients

require retinal ablation (PRP or cryotherapy) at diagnosis.

Dr. Thomas: Obviously, the clinical scenario will differ from patient to patient. The clinical status of the patient, expected longevity, and status of the other eye are important, but as a general guideline I intervene in 7–14 days after onset. Clinical experience has shown that extensive subhyaloid hemorrhages have a poor prognosis because of tractional and contractional effects on the posterior pole architecture. Early intervention with pars plana vitrectomy and removal of the entire posterior hyaloid face and cortical vitreous as far as possible peripherally is advantageous to eliminate the anticipated mechanical forces. Visual acuity is severely affected in all cases because the macula is totally obscured by blood. Because our surgical techniques are so advanced—with instrumentation and the concepts of en bloc posterior hyaloid delamination—the risk of complications is very low and the benefit of stabilization of the retinal architecture and the likelihood of visual recovery are high.

5. Should aspirin or Coumadin be stopped in a patient with vitreous hemorrhage from diabetic retinopathy, ARMD, or PVD? Should one check the anticoagulation status?

Dr. Marcus: Based on results of the Early Treatment Diabetic Retinopathy Study (ETDRS), I do not recommend stopping aspirin in patients with any of these conditions. I extrapolate the ETDRS results to Coumadin and also do not recommend stopping it. For Coumadin especially, I feel that a stroke, myocardial infarction, or pulmonary embolism would be catastrophic given how little we know about any visual benefit from discontinuing anticoagulants. In addition, given our inability to effectively treat hemorrhagic, exudative ARMD, the risk-to-benefit ratio for stopping anticoagulants in this setting is questionable. I seldom consider checking the PT/PTT in patients on Coumadin.

Dr. Thomas: Because vitreous hemorrhage was not increased in patients on aspirin in the ETDRS, I do not stop aspirin in patients with vitreous hemorrhage from any source. Although Coumadin use increases the risks of submacular hemorrhage in

ARMD, the clinician must weigh the risks of stopping Coumadin. Since atrial fibrillation, mechanical heart valves, carotid vascular disease, and history of stroke and deep vein thrombosis create life-threatening risks in the absence of Coumadin, I am very reluctant to stop Coumadin under any circumstances. I would certainly check the anticoagulation status of the patient and have the patient's primary physician adjust the Coumadin appropriately. Over the past 10 years, I have not stopped Coumadin prior to vitrectomy surgery—initially because the patients, for medical reasons, could not tolerate being stopped, but ultimately because I found no significant increase in intraoperative or postoperative complications. Management of intraocular hemorrhage with current vitrectomy techniques allows completion of surgery without additional life-threatening risks.

6. How do you manage the patient with a dense vitreous hemorrhage associated with acute posterior vitreous detachment?

Dr. Marcus: The literature supports a high likelihood of retinal tears with PVD-associated vitreous hemorrhage. In my experience, however, the majority of vitreous hemorrhages in this setting clear without developing retinal tears or detachment. Careful examination with scleral depression and three-mirror contact lens biomicroscopy should be performed in all cases. All tears and detachments should be promptly treated. Retinal tears in this setting often require cryotherapy because the laser will be blocked by the hemorrhage. In dense vitreous hemorrhage, scleral depression will likely be unrevealing. Echography should be performed in this setting to evaluate for retinal tear or detachment. If no tears or detachment are found, I follow the patient closely (initially every few days, then weekly) for clearing or the development of retinal tears or detachment. If the hemorrhage is nonclearing and dense by 1 month follow-up, I will consider vitrectomy or continued observation. Patients should always be counseled as to the symptoms of retinal traction.

Dr. Thomas: When an acute posterior vitreous detachment occurs with a dense vitreous hemorrhage, I am usually aggressive and intervene surgically in the first 2–4 weeks. If at 7 days there is evidence of peripheral clearing and some improved

visualization of the peripheral retina with scleral depressed examination and no evidence of retinal detachment on ultrasound, I may consider waiting further. Usually, though not always, dense hemorrhages are associated with significant pathology. With many retinal vascular diseases, proliferative diabetic retinopathy, and vein occlusions—in addition to retinal tears—the underlying pathology carries risk for progression during an observation period. Again, the patient's overall clinical status should be taken into consideration. Pre-existing ARMD-associated disciform scars that produce a vitreous hemorrhage at the time of PVD would certainly warrant an extended observation period prior to intervention. Barring selected cases, for these reasons, early intervention is warranted.

7. A patient presents with vitreous hemorrhage, but a superior quadrant retinal detachment can be visualized with a single horseshoe tear. What are the roles for scleral buckle, vitrectomy, and pneumatic retinopexy in this patient?

Dr. Marcus: Given that all can be visualized, I would consider any of these options. In general, for this simple, primary retinal detachment, I would favor either buckle or pneumatic retinopexy as an initial surgery. In pseudophakes, I favor buckle over pneumatic retinopexy. I would avoid initial vitrectomy, especially in phakic eyes, because the hemorrhage will often clear. I would more likely consider vitrectomy, possibly combined with scleral buckle, in aphakes or pseudophakes or in patients who require quicker visual rehabilitation (I would use short-acting gas). If the hemorrhage is on the denser side, I might be more inclined to consider vitrectomy.

Dr. Thomas: Before I decide how to manage an eye with visible pathology that is amenable to pneumatic retinopexy, but in which vitreous hemorrhage has obscured detail elsewhere, I consider the patient's comfort level with the examination, and ability of the patient to be cooperative, as well as the possibility of unseen pathology or induced pathology after pneumatic retinopexy. If the patient understands the inherent risks of all of these procedures and the potential benefit of the more limited procedure of the pneumatic retino-

pexy, then I consider the pneumatic retinopexy first. If, at any time, additional complicating factors arise—for example, inferior detachment on ultrasound behind the hemorrhage, new tears visible, or additional vitreous hemorrhage—intervention with vitrectomy to clear the hemorrhage and scleral buckling should be performed.

8. How does one differentiate melanoma from subretinal hemorrhage on ultrasound examination of an eye with a dense vitreous hemorrhage?

Dr. Marcus: The internal reflectivity of a melanoma will be low, whereas the internal reflectivity of a subretinal hemorrhage will be medium to high. Melanomas are unlikely to present with vitreous hemorrhage. With subretinal hemorrhage, the contralateral eye will often demonstrate ARMD.

Dr. Thomas: Low and high internal reflectivity are the classic differentiating findings; the melanoma having low to medium reflectivity and the hemorrhage high reflectivity. The presence of the “collar button” configuration also makes melanoma a more likely diagnosis. The final ultrasound techniques are sequential examinations over time. Ectopic disciform processes with breakthrough vitreous hemorrhages may be difficult to differentiate from a melanoma on ultrasound in an eye with dense vitreous hemorrhage. Do not depend on the ultrasound alone. You have many additional pieces of information, especially the patient's clinical history and the fellow eye to look at for pathology that may be the etiology of the subretinal hemorrhage, especially advanced ARMD. Clinically, the subretinal hemorrhage will evolve and begin to resolve, albeit slowly. The melanoma will probably change very little during a short observational period. The vitreous hemorrhage may also resolve to an extent that visualization is possible. In most cases, the melanoma has been present for an extended period of time and can be observed with ultrasound. It may not change. Most subretinal hemorrhages will begin to resolve and change configuration with flattening and decreasing in size. If there is any indication that the mass is resolving, continued observation is the best plan.

9. What are your recommendations with regard to activity for the diabetic patient with a new vitreous hemorrhage?

Dr. Marcus: I don't limit activity. I recommend head-of-bed elevated positioning during sleep.

Dr. Thomas: Because rapid-eye-movement sleep produces a high risk of vitreous hemorrhage through mechanical traction, I restrict the patient's activity with bed rest and head elevation for only a few days. I want to see if enough clearing occurs so that panretinal photocoagulation with contact lens or indirect ophthalmoscopic delivery can be performed. I always have the patient refrain from strenuous impact activities such as jogging, sky-diving, boxing, weight lifting, all-terrain-vehicle (ATV) riding, and bike riding. I do allow them to read and watch TV, since the eye movements associated with these activities are significantly less than in REM sleep. I also instruct them to avoid any position in which their head is below their heart so that ambient intravascular pressure within the eye does not become elevated. It is important to apprise the patient and his or her family that the changes in visual function of an eye with vitreous hemorrhage increases the risk of injury and necessitates lifestyle changes to accommodate for it. Such information may help the patient avoid such sequela as loss of depth perception (though most patients have already experienced the phenomenon), which can lead to slips, falls, broken bones, and difficulty with insulin injections.

10. How can one differentiate vitreous hemorrhage from ROP and shaken-baby syndrome in an infant?

Dr. Marcus: A history is important in determining if prematurity existed or if non-accidental trauma is possible. The retinal hemorrhages of a shaken baby are more likely bilateral, and more likely demonstrate hemorrhages throughout different layers of the retina, optic nerve, and vitreous. Signs of ROP may be present in the uninvolved eye. Echography may sometimes be helpful in assessing

the extent and location of hemorrhages in the setting of vitreous hemorrhage. Shaken-baby syndrome will sometimes present with a distinctive traumatic retinoschisis. ROP more likely presents with peripheral traction. Other systemic signs of, and workup for, shaken-baby syndrome should be sought with pediatric consultation.

Dr. Thomas: The clinical scenario and the clinical examination are the critical means of differentiating ROP from shaken-baby syndrome. Knowledge of prematurity and pre-existing ROP enhance the reliability of this differential diagnosis. Often, the physical evidence suggests shaken-baby syndrome but is not conclusive. To reduce or eliminate ambiguity the ophthalmologist will need the help of the pediatrician and may have to determine whether social service personnel have ever been involved in the baby's welfare. Such information aids not only in the diagnosis but, if positive for the syndrome, also in management of the family environment during the recovery period. Multiple hemorrhages scattered throughout the posterior pole and periphery at different levels—intraretinal, subretinal, and subhyaloid—are typical for shaken-baby syndrome, while peripheral retinal neovascular tissue associated with peripheral vitreous hemorrhage, seen with scleral depression, is typical for the ROP baby. As always, look at the whole patient and the prior information available about the child and family. When the eye examination is done, you are able to place the clinical findings within the frame of the complete clinical scenario.

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