Managing Proliferative Vitreoretinopathy With Retinal Detachment

There is a high probability of anatomic success after repair.

BY TAREK S. HASSAN, MD

In this issue of Retina Today, Tarek S. Hassan, MD, provides surgical pearls for managing proliferative vitreoretinopathy with retinal detachment.

We extend an invitation to readers to submit pearls for publication in Retina Today. Please send submissions for consideration to Ingrid U. Scott, MD, MPH (iscott@psu.edu); or Dean Elliott, MD (dean_elliott@meei.harvard.edu). We look forward to hearing from you.

—Ingrid U. Scott, MD, MPH; and Dean Elliott, MD

Proliferative vitreoretinopathy (PVR) is the most common complication causing retinal reattachment after primary retinal detachment (RD) surgery. It is reported to occur in approximately 7% to 8% of patients following primary RD surgery. In a tertiary referral practice, as many as 25% to 30% of patients who present with RD may have associated PVR.

PVR is a process of cellular proliferation, brought on by the zealous healing process within the posterior segment of the eye, stemming from the presence of partial or full-thickness retinal breaks or holes, in which glial and retinal pigment epithelial (RPE) cells proliferate and form membranes on and/or beneath the retinal surface. The epiretinal membranes and subretinal fibrosis contract and cause shortening of the retina; they distort and hold open the retinal breaks and can lead to persistent or progressive RD. This proliferative process can occur in untreated eyes as well as eyes that have undergone primary RD repair with scleral buckling or other interventions.

Significant visual loss occurs frequently after PVR-associated redetachment. Although anatomic repair is possible in most cases, the management can be challenging. This article presents some of the strategies I use in our referral practice to manage PVR in RD.

RISK FACTORS FOR PVR

Significant risk factors for PVR in RD include chronic RD, numerous and/or large retinal breaks, giant retinal tears, trauma, aphakia, previous intraocular surgery, vitreous hemorrhage, and uveitis. Patient age is also a factor. In younger patients, the proliferative healing response is more aggressive, and a young patient with trauma has a high potential for the development of PVR. Pediatric patients with retinal dystrophies such as retinopathy of prematurity or familial exudative vitreoretinopathy are also at higher risk for PVR.

GOALS OF MANAGEMENT

My main goals in managing RD with PVR are to reattach the retina and to relieve all traction, both from epiretinal membranes and from subretinal fibrosis. It is also important to relieve any foreshortening of the retina. Sometimes surgeons repair the RD and remove all the visible scar tissue but fail to adequately relax the retina. In these cases, the retina may be reattached intraoperatively, but it may redetach postoperatively when the potential for further foreshortening exists. I aim to make sure the retina is as free of as much traction and foreshortening as possible with such thoughts for the future in mind.

There has been a stigma regarding performing signifi-
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cant retinotomies or retinectomies in these types of procedures because outcomes were poor a couple of decades ago. However, much of that negative attitude was based on old data, old equipment, and old techniques. With current small-gauge, high-speed, more flow-controlled vitrectomy technology, we can do a better job of safely repairing RDs and can use retinotomy and retinectomy techniques as needed to relieve traction and foreshortening of the retina in any dimension.

MANAGEMENT OPTIONS

When faced with PVR today, I generally assume an intraocular procedure is or will be required if traction-based RD occurs. The management of PVR with RD depends on the duration of the RD. If the patient has a chronic RD, the epiretinal membranes and subretinal fibrosis are more mature. Although these membranes are easier to divide into sections and peel than membranes that have not been in place as long, eyes with such chronic scar tissue typically have poorer long-term visual outcomes.

I currently perform small-incision sutureless vitrectomy with either 23- or 25-gauge instrumentation. State-of-the-art vitrectomy equipment allows us to reach just as far into the periphery and to do as many manipulations as we could with 20-gauge vitrectomy but with less trauma to the eye. In addition, with the smaller-gauge equipment, we can work into spaces under the retina or in the midst of epiretinal scar tissue using the vitreous cutter to perform functions we used to do with scissors.

It is important to make sure that any posterior hyaloid still attached to the retina is removed. PVR is much more prevalent in eyes in which some posterior hyaloidal elements remain attached to the retina. Often, particularly after scleral buckling, post-RD PVR develops in eyes in which a posterior vitreous detachment was not complete.

In addition to there being many advantages to using small-gauge instrumentation for PVR repair, there are minimal disadvantages compared with older 20-gauge techniques; the latest generation of equipment has reduced earlier limitations of smaller-gauge instruments. With the most recent generation of vitrectomy gear, light and laser probes are sufficiently stiff, illumination is excellent, bimanual surgery is enhanced with the use of lighted knives and chandelier lights, and there is a full range of other instrumentation in 23- and 25-gauge sizes, with the exception of a fragmatome.

We can even introduce silicone oil using the smaller-gauge cannulas. Silicone oil is useful when the PVR is significant, recurrent, and/or predominantly in the inferior quadrants. Silicone oil is probably underutilized in the United States compared with other parts of the world, although I think it is a terrific tool for long-term tamponade. It helps to stabilize difficult eyes that have undergone multiple recurrent refractory PVR-associated RDs.

Scleral buckling is sometimes helpful as part of a PVR repair. A buckle can reinforce the vitreous base and peripheral retina and help to relieve foreshortening of the retina in addition to retinotomy. If the retina has a significant amount of anterior proliferation that cannot be removed satisfactorily with peeling, or if the patient is to be left phakic, often a buckle can serve as an adjunct to the intraocular repair. If all the pathology is posterior and the traction and foreshortening can be relieved to my satisfaction with vitrectomy, I often do not use a buckle.

It is possible to repair PVR with scleral buckling alone, especially when the PVR is limited to a single quadrant. However, this approach has largely been supplanted by effective, safe vitrectomy in recent years and has now become a technique that is used only rarely.

COMPLICATIONS OF PVR REPAIR

Complications of PVR repair can occur from the creation of an excessively large retinectomy or retinotomy, for example. These can be made larger than needed to relax the retina, thereby taking away too much viable retina. This can have several potential effects, including the reduction of postoperative visual functioning, induction of more PVR, and creation of the most challenging complication of PVR surgery: postoperative hypotony.

Most often, postoperative hypotony occurs for 2 basic reasons: either the ciliary body shuts down due to repeated anatomic insults from RDs and ischemia and does not produce aqueous adequately, or the ciliary body is affected by extensive scar tissue that limits aqueous production.

In PVR repair, however, hypotony can occur when large areas of bare RPE are left following large retinectomies that lead to an increase in uveoscleral outflow. Any 1 or combination of these mechanisms may lead to hypotony as a complication of PVR. The management
of such hypotony is often challenging and may be ineffective. If it is due to extensive fibrous proliferation on the ciliary body and/or cyclitic membrane, anterior membrane peeling to remove this tissue can, in some cases, result in improved intraocular pressure. Often, however, the ciliary body processes experience significant ischemia, and this approach may be ineffective or not indicated. Similarly, no effective treatment can anatomically address hypotony caused by excessive uveoscleral outflow. I have attempted to cause an intraocular pressure rise with repeated high-dose topical steroid application or sub-Tenon's steroid injections. Marginally successful results have been seen with these approaches, and thus I am often left filling such eyes with long-term silicone oil tamponade if they are fluid-filled, or leaving the silicone oil indefinitely if they are already oil-filled.

Another potential complication of extensive PVR repair is damage to the optic nerve due to decreased perfusion. This can result in significant optic atrophy due to the repeated accumulation of ischemic insults from multiple RDs and the procedures to repair them.

**RECENT REPRESENTATIVE CASE REPORT**

A 72-year-old man with significantly reduced vision for at least 1 month presented to a referring physician. At presentation, the patient had a macula-off RD with subretinal fibrosis and some anterior PVR in one quadrant temporally, for which the referring surgeon performed a scleral buckle procedure. The surgeon reported that this intervention worked to treat the RD for approximately 1 week, but then the retina redetached. By the time the patient came to us for repair, the area of subretinal fibrosis had detached the entire temporal retina, and the patient had developed surface PVR inferiorly and over the macula.

The patient was taken to the operating room for 23-gauge vitrectomy. The scleral buckle was left in place; no buckle revision was performed. I first examined the retinal surface for signs of posterior hyaloid attachments. I occasionally inject a small amount of triamcinolone onto the retinal surface to help identify isolated areas of posterior hyaloidal attachment if I feel that there may be some present and I cannot see them. I also sometimes inject a small amount of a very diluted indocyanine green stain to help identify the internal limiting membrane (ILM), as I feel that ILM removal can be very helpful in minimizing recurrence of PVR membranes in the future. Using a 2-handed combination of intraocular forces and a 23-gauge lighted knife, along with a peripheral chandelier for illumination, I removed the surface PVR from the macula and inferior retina. A very small peripheral retinotomy was created, through which I was able to use standard 23-gauge forces to effectively remove the subretinal fibrosis.

The retina was foreshortened inferiorly, which is common in chronic RD cases, so I made a retinotomy of approximately 4 clock hours along the posterior edge of the scleral buckle. Then the retina was flattened with perfluorocarbon liquid, and the areas of the repair and retinotomy were treated with 2 to 3 concentric rows of laser.

The eye was filled with silicone oil to a level just posterior to the iris plane, and the surgery was completed. I chose to leave the eye with silicone oil rather than gas because the RD had been chronic and 1 prior surgery had failed, with worsening of the PVR. The pathology was largely inferior, and I did not think that patient positioning would be effective in keeping that area tamponaded with gas.

With follow-up of 4 months, the patient is doing well with complete anatomic reattachment, no recurrent PVR, and visual improvement to 20/50 from a preoperative vision of counting fingers. The patient is now contemplating wanting the silicone oil removed in the next few weeks.

Patients with PVR-associated RD should be informed preoperatively about the possibility of recurrence, poor visual prognosis, refractive changes, and potential long-term complications. The patient should be examined on day 1 postoperatively for increased IOP, hypotony, and signs of endophthalmitis. Periodic examinations should occur thereafter.

**CONCLUSION**

Today, we are able to repair eyes with RD and PVR with modern vitreoretinal surgical techniques and instrumentation that lead to a high likelihood of anatomic success—perhaps in excess of 90% to 95%. Unfortunately, patients will have varying degrees of visual recovery, with poorer visual results generally seen if multiple procedures are required.

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