RAD for Central Retinal Vein Occlusion

Novel venous circulation is achieved via creation of multiple venous anastomotic sites.

BY CALVIN GRANT, MD

Central retinal vein occlusion (CRVO) remains a significant cause of vascular-related visual loss in many patients. Ischemia, edema, and blood are at the root of this loss.[1] As with many of our treatments, the main focus of therapy for CRVO has been selected elements in the pathophysiologic tree.[2] Antivascular endothelial cell growth factor (VEGF) strategies have had some success in diminishing macular edema and blood, but visual function improvement has been modest, especially in cases of ischemia.[3] Furthermore, pharmacologic strategies do not address the underlying cause of the ischemia. The laser and surgical methods for addressing CRVO have attempted to rework the venous drainage or address the underlying thrombus. These approaches have been limited secondary to complications or ineffectiveness. Additionally, previous strategies that relied on systemic or regional administered thrombolytics have largely failed.

An alternative method that employs widely used surgical principles to restore venous blood flow has been described: Revascularization by anastomotic decompression (RAD).[4] In RAD, multiple anastomotic sites between the retinal venous circulation and the choroid are induced in the "far" posterior pole. With improving retinal venous circulation, edema, and even ischemia, may be reversed or improved (Figure 1). Simply put, the procedure creates a novel venous circulation by the induction of multiple venous anastomotic sites. The key steps are vitrectomy, induction of chorioretinal venous anastomoses, aspiration of minimal blood, laser photocoagulation, and air-fluid exchange.

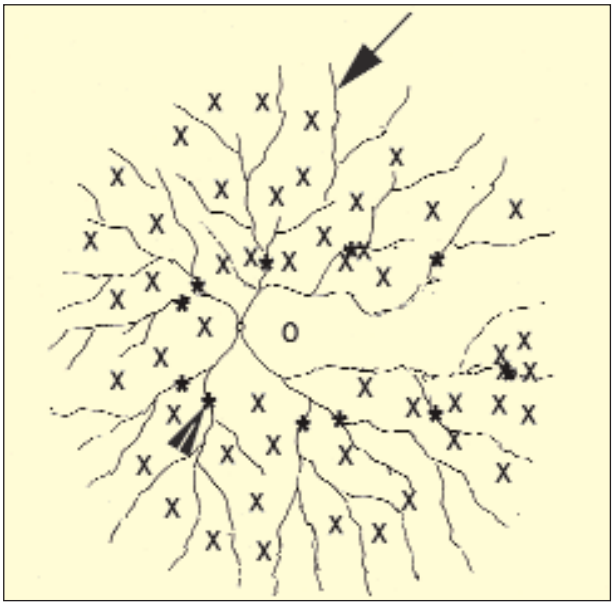

Figure 1. The goal is to diffusely decompress upstream macular vascular congestion (venous and arterial). Chorioretinal venous anastomoses are induced anterior to the vascular arcades over the entire venous trunk.

VITRECTOMY

Through a 20- or 23-gauge vitrectomy, a posterior vitreous detachment is created, and the vitrectomy is completed to well beyond the equator. Given that slit-lamp–derived laser procedures were plagued by vitreochoriodal neovascularization, double-checking for complete elevation of the vitreous with the lack of the

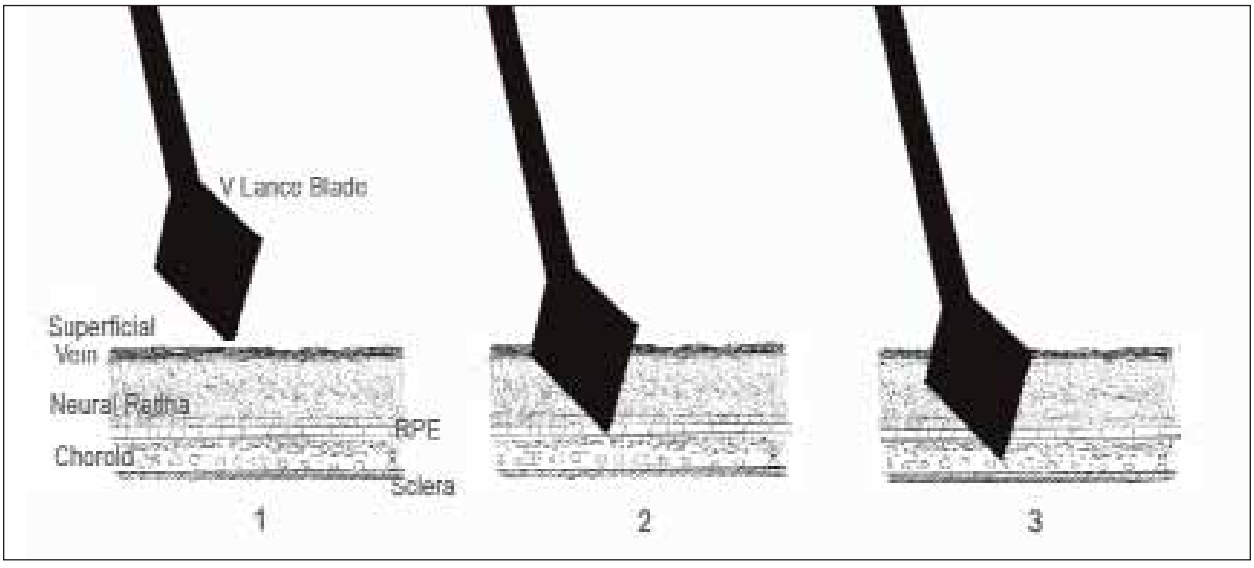

Figure 2. Sites are selected where retinal veins overlap choroidal veins. An MVR blade enters the vein and Bruch's is interrupted. Deeper choroidal vascular structures are engaged.

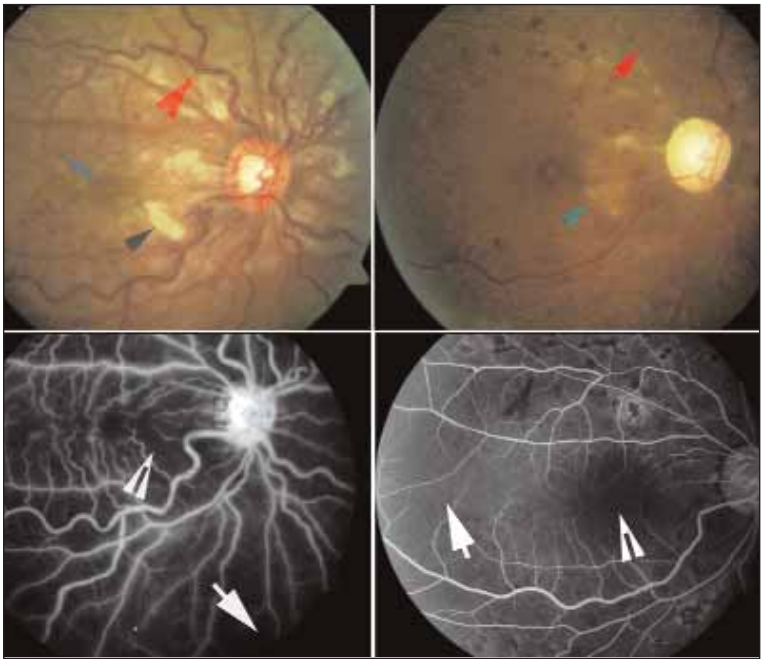

Figure 3. Before (left images) and 2 weeks after RAD (right images).

fish-strike sign is of the utmost importance. The vitrectomy is then continued to the far periphery.

CHORIORETINAL VENOUS ANASTOMOSES

Attention is then directed to the selection of the anastomotic sites. It is apparent from this procedure that peripheral impedence has a tremendous bearing on macular venous congestion. The collaterals may be able to drain macula blood flow once the venous column from the periphery is diverted or the macular circulation empties into the newly formed anastomoses.

The prime sites are 2 to 3 disc diameters anterior to the vascular arcades. Ideally, the sites are selected where a vein crosses a deeper choroidal vessel. If intraretinal blood obscures the choroidal vasculature, then the stab incisions are made blindly. Preoperative indocyanine green angiography may be useful in delineating posterior ciliary arteries and vortex veins to be avoided. On occasion, I increase the intraocular pressure to 70 mm Hg prior to creating the wound. In most cases, however, I wait for the first sign of hemorrhage before elevating the pressure. In ischemic patients, it is often difficult to see the venous tree with pressure elevation. Hemorrhaging, if it occurs, is usually minimal and easily controlled.

Stab incisions transect the retinal vein with one or two passes down to the mid-choroid. The microvitreoretinal (MVR) knife passes perpendicularly through these structures (Figure 2). For a standard 20-gauge MVR, the blade is advanced one-quarter to one-third of the tip. A 23-gauge MVR may be advanced to the bevel. Edema and individual anatomical differences must be considered with these relationships. It is important not to drag the blade. If you feel something firm, it is usually

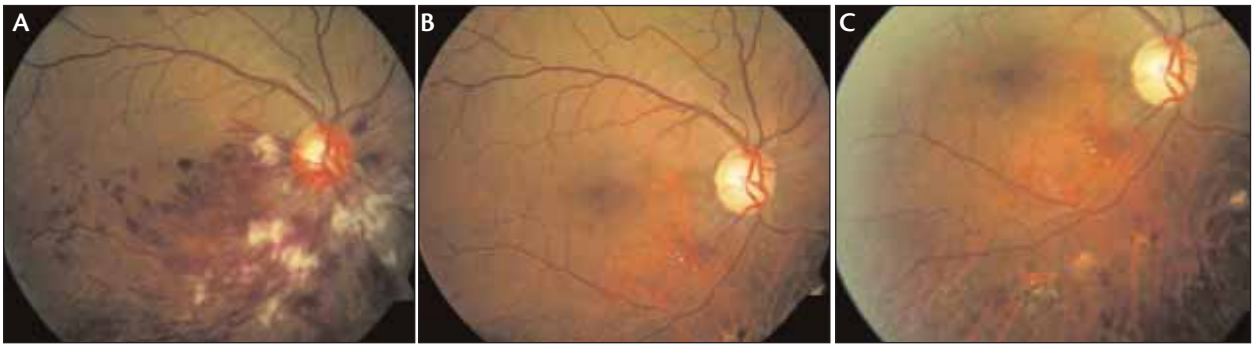

Figure 4. Six weeks after RAD.

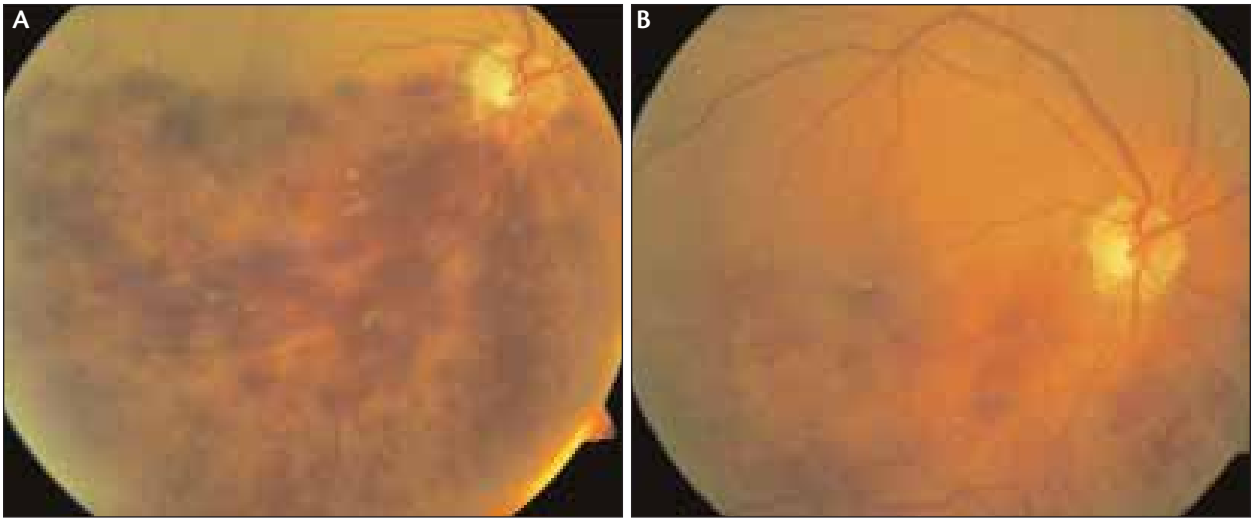

Figure 5. Six weeks after RAD.

the sclera—do not advance beyond this point. Some patients have a more rigid Bruch's membrane that may provide some resistance. Penetration of Bruch's membrane may be associated some minor bleeding. This may serve as an endpoint for stab depth. Stab incisions are created around the entire venous tree for ischemic patients. For patients with non-ischemic occlusions, some vessels nasal to the disc may be skipped.

ASPIRATION, PRP, AND AIR-FLUID EXCHANGE

The next step in RAD is aspiration of blood, which is performed with a silicone soft-tip catheter. There is usually some blood that remains on the surface of the stab incision. I generally do not remove this drop of coagulated blood. Chasing the blood may lead to continued bleeding and is not necessary. Two to three rows of semi-confluent PRP intensity laser are placed surrounding each incision site and PRP is performed to the midperiphery. Lastly, a complete air fluid exchange is performed.

For postoperative recovery, I recommend face-down positioning until the air bubble has dissipated. I do not recommend using steroids or anti-VEGF agents for at least 8 weeks postoperatively. Typically, pharmacologic agents are not required for recovery and shunts are functional within 2 weeks (Figure 3). In my experience, most patients demonstrate maximal benefit from RAD in 6 to 8 weeks (Figures 4 and 5). ■

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