Chorioretinal Venous Anastomosis for Central Retinal Vein Occlusion: Transvitreal Venipuncture

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Abstract. The authors describe an eye with a central retinal vein occlusion that developed chorioretinal anastomoses following transvitreal venipuncture, a vitreoretinal surgical technique. [Ophthalmic Surg Lasers 1999; 30: 52–55.]

The creation of a vascular anastomosis between a retinal vein and a choroidal vein may bypass the venous obstruction in eyes with a vein occlusion. Laser photocoagulation has been successful in creating an anastomosis with a corresponding improvement in visual acuity in some eyes with a central retinal vein occlusion (CRVO). 1,2 We describe transvitreal venipuncture, a vitreoretinal surgical technique that resulted in the creation of a chorioretinal anastomosis in an eye with a CRVO.

CASE REPORT

A 21-year-old woman was referred to the Wilmer Vitreoretinal Surgical Service with a 6-month history of progressive visual acuity loss from a CRVO in the right eye. Corrected visual acuity was 1/200 in the right eye and 20/20 in the left eye. There was a right afferent pupillary defect. Gonioscopy, intraocular pressure, and the results of slit-lamp examination were normal in both eyes. No Weiss ring was present. Ophthalmoscopy of the right eye (Fig. 1) disclosed marked optic disc edema, venous tortuosity, moderate intraretinal hemorrhage in all four quadrants, and cystoid macular edema with early subfoveal fibrosis. No neovascularization was present. Fluorescein angiography disclosed blocked fluorescence from the intraretinal hemorrhage. The parafoveal vascular architecture (arrow) was largely intact.

Figure 1. (A) Ophthalmoscopy of the right eye disclosed marked optic disc edema, cystoid macular edema with early subfoveal fibrosis (arrow), and moderate intraretinal hemorrhage in all four quadrants. (B) Fluorescein angiography disclosed blocked fluorescence from the intraretinal hemorrhage. The parafoveal vascular architecture (arrow) was largely intact.

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Figure 2. (A) Two weeks postoperatively, ophthalmoscopy disclosed less disc edema and intraretinal hemorrhage without an obvious chorioretinal anastomosis. At five puncture sites, fine preretal neovascularization was present. Subfoveal fibrosis was again noted. The boundaries of the subretinal blood were discernible superior and inferior to the disc (arrows). The blood did not extend subfoveally. Compare with Figure 2B. (B) Fluorescein angiography demonstrated neovascularization at five treatment sites. It was not possible to discern if the neovascular growth was retinal or choroidal in origin. An anastomosis could not be identified. Blocked fluorescence from the subretinal blood was present superior, inferior, and nasal to the disc. The blocked fluorescence within the macula was due to the subfoveal fibrosis and the parafoveal and perifoveal intraretinal hemorrhage; the subretinal blood did not extend under the foveal center. Compare with Figure 2A.

ophthalmoscopy disclosed blocked fluorescence from intraretinal hemorrhage and a largely intact parafoveal vascular network. More than 10 disc areas of retinal capillary nonperfusion were present nasally and inferiorly. Late parafoveal dye accumulation was present. Ophthalmoscopy of the left eye was normal.

The diagnosis of an ischemic CRVO with perfused macular edema was made. Using a standard 3-port pars plana approach, the posterior hyaloid was engaged and detached from the retina and removed with the vitreous cutter. In six locations along the larger second and third order retinal vessels, intraocular diathermy was used to coagulate the locations to be penetrated. A 20-gauge needle tip was used to puncture the retinal vein and underlying Bruch’s membrane in each location. During puncture of the superonasal treatment site, subretinal bleeding developed but did not extend toward or under the macula; instead, the hemorrhage extended into the superior and nasal subretinal space and was controlled by raising the height of the infusion bottle. Any preretinal blood was aspirated through a soft-tipped cannula. Hemostasis was achieved.

Two weeks postoperatively, the corrected visual acuity was 6/200. Ophthalmoscopy disclosed less disc edema and intraretinal hemorrhage without an obvious chorioretinal anastomosis (Fig. 2A). Preretinal neovascularization with early preretinal fibrosis developed at five puncture sites. Subretinal fibrosis was again noted in the foveal center. Fluorescein angiography (Fig. 2B) demonstrated fine, new vessels emanating from five of the puncture sites. It was not possible to determine ophthalmoscopically or angiographically if these vessels were retinal or choroidal in origin. An obvious chorioretinal anastomosis could not be identified. Blocked fluorescence from the subretinal blood was present superior, inferior, and nasal to the disc. The subretinal blood did not extend under the foveal center (Fig. 2B).

Six weeks postoperatively, the corrected visual acuity was 20/400. No neovascularization of the iris or the angle was present. Ophthalmoscopy demonstrated further resolution of the disc edema, intraretinal hemorrhage, venous tortuosity, and cystoid macular edema (Fig. 3). The subfoveal fibrosis was more evident. The neovascular proliferation had largely involuted. Preretinal fibrosis at each treatment site made identification of a chorioretinal anastomosis difficult.

Four months postoperatively, the corrected visual acuity was 20/400. There was no iris or angle neovascularization. Ophthalmoscopy disclosed no disc edema, macular edema, intraretinal hemorrhage, venous tortuosity, or neovascularization (Fig. 4A). Subfoveal fibrosis was again identified. Preretinal fibrosis was present at each treatment site with some surrounding traction. The presence of an inferonasal
Chorioretinal anastomosis was strongly suggested ophthalmoscopically because of the wider diameter of the central venous segment that dipped down into the choroid at the puncture site, compared with the narrower diameter of the peripheral portion of that vein (Fig. 4A). The other treatment sites were more difficult to assess ophthalmoscopically for the presence of an anastomosis, partly because of overlying preretinal fibrosis. The central venous segment at the 5 other treatment sites was either the same diameter (1 site) or had markedly narrowed (4 sites) compared with the diameter of the more peripheral segment and, at 3 of the 4 sites, was ophthalmoscopically devoid of blood (Fig. 4A). Fluorescein angiography disclosed preferential fluorescein filling of the central venous segment that dipped down into the choroid inferonasally compared with the peripheral portion of the vein (Fig. 4B), suggesting the presence of an anastomosis. The central venous segment at 4 of the other 5 treatment sites filled preferentially with fluorescein dye before the peripheral venous segment filled, also suggesting the presence of a chorioretinal anastomosis at these puncture sites. At one treatment site, however, the central venous segment was not patent angiographically (Fig. 4B), although the peripheral venous segment was patent and seemed to be draining into the choroidal circulation.

Seven months postoperatively, the visual acuity remained 20/400. The ophthalmoscopic and fluorescein angiographic findings were unchanged.
DISCUSSION

A chorioretinal anastomosis provides an alternate route for retinal blood flow to exit an eye with an occluded central retinal vein. Chorioretinal anastomoses have been created using laser photocoagulation in eyes with a perfused CRVO.1,2 In an eye with an ischemic CRVO, however, anastomosis formation using laser photocoagulation has not been advocated, because the likelihood of developing neovascular complications of the procedure may be higher in ischemic eyes and reestablishing venous outflow from these eyes would not likely reperfuse the areas of retinal capillary dropout.

Despite this, laser photocoagulation to create an anastomosis has been attempted in eyes with ischemic CRVO.3 Perhaps to improve perfusion of the existing retinal vasculature and decrease the likelihood of developing iris and/or angle neovascularization. Moreover, perfused macular edema may coexist in an eye with a peripheral ischemic CRVO, and in these cases, as in our case, anastomosis formation may lead to some visual improvement. However, given the widespread ischemia, the potential to develop chorioretinal neovascularization may be greater than in eyes with nonischemic CRVO, especially if the posterior hyaloid is attached, because it may provide a scaffold for neovascular proliferation.4

We describe a vitreoretinal surgical technique that resulted in the formation of chorioretinal venous anastomoses in an eye with a CRVO. Intraoperative detachment of the posterior hyaloid during this procedure may lessen the likelihood of developing chorioretinal neovascularization,4 and, if neovascular proliferation should develop at the treatment site, possibly lessen the severity. In our case, some neovascular proliferation occurred at five of the treatment sites postoperatively, despite intraoperative removal of the posterior hyaloid, and may have been either retinal or choroidal in origin. The neovascularization involuted without intervention in this case.

In this patient, the procedure was not without complication. A subretinal hemorrhage developed intraoperatively and may have been avoided by elevating the intraocular pressure just before puncturing the retinal vein and Bruch's membrane instead of after the bleeding began. Neovascular proliferation of either retinal or choroidal origin was identified at most of the treatment sites postoperatively and regressed without intervention. The severity of the neovascularization may have been subdued, in part, by the absence of the posterior hyaloid in this case; perhaps the concurrent placement of panretinal laser endophotocoagulation may lessen the likelihood of neovascular complications. Preretinal fibrosis was not avoided using this technique.

Although the visual and ophthalmoscopic improvement in our case may not be a direct result of intervention, this technique of transvitreal venipuncture resulted in ophthalmoscopic and angiographic evidence of chorioretinal anastomosis formation in an eye with a CRVO.

REFERENCES