Chorioretinal Anastomosis After Radial Optic Neurotomy for Central Retinal Vein Occlusion

José García-Arumí, MD; Anna Boixadera, MD; Vicente Martínez-Castillo, MD; Reinaldo Castillo, MD; Antonio Dou, MD; Borja Corcostegui, MD

Objectives: To evaluate the incidence of chorioretinal anastomosis after radial optic neurotomy and to determine its effect on visual acuity and foveal thickness in patients with central retinal vein occlusion.

Methods: We conducted a prospective, uncontrolled, interventional study of 14 patients with preoperative visual acuities below 20/125. Pars plana vitrectomy and radial optic neurotomy were performed. Fluorescein angiography and optical coherence tomography were used to monitor the evolution of macular edema.

Results: All patients underwent radial optic neurotomy with no major complications. Eight patients (57.1%) gained 1 or more lines of visual acuity while the visual acuity of 6 patients (42.9%) improved by 2 or more lines (mean visual acuity, 20/80; P<.001) (mean visual acuity gain, 3 lines). The decrease in macular thickness was shown to be statistically significant (P<.001) (median, 282 µm). Retinochoroidal shunts developed in 6 eyes (42.9%) at the site of the radial optic neurotomy.

Main Outcome Measures: Improvement in visual acuity and a decrease in foveal thickness seen on optical coherence tomography.

Conclusions: Surgical decompression of central retinal vein occlusion via radial optic neurotomy seems to be a promising technique that improves or at least stabilizes the course of severe central retinal vein occlusion. Improvement may occur because of optic nerve decompression, vitrectomy, and by inducing new chorioretinal shunts that drain retinal circulation to the choroid and accelerate resolution of retinal edema.

Arch Ophthalmol. 2003;121:1385-1391

CENTRAL RETINAL vein occlusion (CRVO) is the third most common blinding vascular retinal disorder1,2 after diabetic retinopathy and branch retinal vein occlusion. Among patients with CRVO, 34% develop capillary nonperfusion and retinal ischemia. Iris neovascularization and neovascular glaucoma may occur in 45% to 85% of the eyes affected by ischemic CRVO and only in 5% of the nonischemic eyes.2-3 The main known risk factors of CRVO are hypertension and open-angle glaucoma.2-5

The Central Vein Occlusion Study reported that the final visual acuity (VA) mainly depends on the VA at the initial presentation.6 In patients with a VA greater than 20/40, the VA stabilizes in 65% of the eyes. If the initial VA is less than 20/200, 80% of the eyes have a final VA that is unchanged or worse and a high risk of developing ischemic CRVO. In patients with intermediate VA (20/50 to 20/200), 44% of the eyes remain stable while 37% of the eyes worsen. Moreover, 30% of the eyes convert from nonischemic to ischemic CRVO; this percentage increases as VA worsens.

The pathogenesis of CRVO is yet not very well understood. It is thought to be a compartment syndrome, since in a 1.5-mm-diameter area, the central retinal artery, the central retinal vein, and the optic nerve coexist. Thrombotic occlusion is thought to develop as the result of an increase in the arterial diameter, changes in the scleral ring, and the presence of anatomical anomalies and possible systemic factors, which together cause a decrease in the venous lumen, increased turbulence, and damage to endothelium and thrombus formation. This is supported by histologic studies that localize the thrombus in the lamina cribrosa in most or all cases.7,8

There is no effective treatment for CRVO. Panretinal laser photocoagulation has only been effective in managing...
METHODS

A prospective, uncontrolled, interventional study was designed to treat 14 patients with nonischemic CRVO and severe loss of VA. Inclusion criteria were a VA of 20/125 or worse caused by macular edema and hemorrhages secondary to CRVO of fewer than 12 months from onset. Patients with CRVO and a VA higher than 20/125 were observed and excluded from the study unless the VA decreased. Exclusionary criteria were other associated vasculopathies, previous laser photocoagulation and vitreous hemorrhage, or retinal neovascularization secondary to CRVO. All patients provided informed consent.

Preoperative data recorded from the patients included age, sex, race, eye affected, bilaterality, time from onset, refraction, VA measurement using the Early Treatment Diabetic Retinopathy Study letter chart, and the presence of an afferent pupillary defect and/or risk factors such as hypertension, open-angle glaucoma, hyperlipidemia, or antiphospholipid syndrome.

All 14 eyes underwent indirect ophthalmoscopy and indirect slitlamp examination, including biomicroscopy of the vitreous and retina. The degree of macular edema, extension of hemorrhages, and presence of submacular hemorrhage were recorded. For each patient fundus photographs were taken and fluorescein angiography was performed.

Optical coherence tomography (OCT) (model 2000; Humphrey Instruments, San Leandro, Calif) was performed to measure the foveal thickness in all patients, and the best-corrected VA was obtained at the same time in all eyes within 1 week before surgery. Optical coherence tomography was performed and the best-corrected VA was measured postoperatively at 1, 2, 6, and 12 months. The final VA was the 1 measured at the 6-month follow-up visit.

Patients were fully informed of all relevant aspects of the procedure. The same surgeon (J.G.-A.) performed all procedures. A standard 3-port pars plana vitrectomy was performed in all cases and, if the cortical vitreous was adhering to the posterior pole, the posterior hyaloid was dissected from the retina using a vitreous probe or a silicone-tipped cannula under active aspiration; the dissection began over either the optic disc or the temporal vascular arcade. A standard microvitreoretinal blade was used to perform radial optic neurotomy (a single radial cut was made on the nasal aspect of the optic disc to avoid damage to the major nasal retinal vessels). After performing the radial optic neurotomy, intraocular pressure was increased to avoid bleeding. No corticosteroids were injected during the procedure either intraocularly or periocularly.

The intraoperative factors recorded were intraoperative bleeding and a change in the caliber of the central retinal vein and color of the optic nerve after decompression. We evaluated the following postoperative factors: changes in best-corrected VA, decreased macular edema measured clinically and by OCT, decreased or resolved intraretinal hemorrhages, and angiographic assessment of venous reperfusion.

The results were calculated with nonparametric statistical methods. The relation between preoperative and postoperative VA and foveal thickness (measured by OCT) was calculated using the Spearman correlation test and scatterplot graphics. P<.05 was considered statistically significant.
period ranged from 6 to 16 months postoperatively (mean follow-up period, 9.6 months).

Preoperative best-corrected VA ranged from hand motions to 20/125 (mean, 20/250; median, 20/250). Preoperative foveal thickness measured by OCT varied from 418 to 1000 µm (median, 707 µm).

Each patient had had a symptomatic decrease of VA in the affected eye due to a CRVO of less than 8 months’ duration (range, 1-32 weeks; mean, 2.1 weeks from onset). Of the 14 patients, systemic hypertension was present in 5 (35.7%), open-angle glaucoma in 4 (28.6%), dyslipidemia in 2 (14.3%), diabetes mellitus in 1 (7.1%), and antiphospholipid syndrome in 1 (7.1%). One of the patients had no risk factors. Four patients (28.6%) had an afferent pupillary defect. Central retinal vein occlusion was bilateral in 4 patients (28.6%). The patients’ demographic data are listed in the Table.

Slight vitreous hemorrhage was observed in the early postoperative period in 1 (7.1%) of 14 patients and cleared spontaneously in 3 weeks. In 3 patients (21.4%) a small subretinal hemorrhage was present at the site of the radial cut. Visual acuity increased to 20/80. Clinical improvement of the macular edema and hemorrhages was observed in all patients (Figures 1, 2, and 3).

Postoperative VAs ranged from 20/400 to 20/25 (median, 20/80; mean, 20/80). The difference between the preoperative and postoperative VA levels was statistically significant (P<.001, Spearman rank correlation value, $r=0.81$). Eight patients (57.1%) had an improved best-corrected VA of 1 or more lines gained while 6 patients’ (42.9%) VA improved by 2 or more lines. No patient had a postoperative decrease in VA (Figure 4). The preoperative foveal thickness ranged from 418 to 1000 µm (median, 707 µm; 25th percentile, 644 µm; and 75th percentile, 976 µm); the postoperative thickness ranged...
from 206 to 559 µm (median, 467 µm; 25th percentile, 386 µm; and 75th percentile, 508 µm).

Visual acuity recovery was statistically related to the decrease in macular edema. There seemed to be a tendency to achieve better best-corrected VA when the foveal thickness values decreased below 260 µm during follow-up (Figure 5).

Six patients (42.9%) developed postoperative chorioretinal shunts at the site of the radial optic neurotomy. These cilioretinal shunts appeared between 3 weeks and 3 months after surgery, with a mean of 35 days. The median best-corrected VA achieved by the group with these shunts was 20/60 vs 20/110 in those patients who did not develop a chorioretinal anastomosis. Although the best-corrected VA was better in the patients who developed an anastomosis, the difference was not statistically significant ($P = .28$). The patients who did not show a significant increase in best-corrected VA had macular ischemia (Figure 6) or subretinal hemorrhage.

No patient developed retinal or iris neovascularization by the end of the follow-up period. Six patients (42.9%) developed a cataract.

**COMMENT**

It is clear from the Central Vein Occlusion Study\(^6\) that, when left to follow its natural course, the vision in patients with CRVO will most likely worsen or remain unchanged and that those patients with poor vision initially have little hope of significant spontaneous recovery. There is no known effective treatment for CRVO. Several treatments have been attempted apart from those approved by the CRVO study.\(^6,9\) McAllister et al,\(^12\) Fekrat et al,\(^13\) and Browning\(^14,15\) tried to create a
chorioretinal venous anastomosis using high-energy argon laser combined or not with neodymium:YAG laser to bypass the site of the obstruction to venous outflow at the level of the lamina cribrosa, but they achieved variable results and several associated complications developed, especially significant neovascular ones.

Other therapeutic attempts included the use of thrombolytic agents, mostly recombinant tissue plasminogen activator. Recombinant tissue plasminogen activator was either injected into the vitreous cavity or cannulated into a retinal vein as was done by Weiss and Byone, and previously proved by experimental studies by Tang and Han. Nevertheless, these studies have provided only preliminary results.

Fekrat and de Juan described a technique to treat ischemic CRVO consisting of surgically induced chorioretinal vein anastomosis at the larger second- and third-order retinal vessels. Lastly, optic nerve sheath decompression was attempted using an external approach by surgical interventions.
Vasco-Posada, later by Arciniegas, and recently by Dev and Buckley. It is a difficult technique with important potential complications. Our patients underwent radial optic neurotomy, described by Opremcak et al, that consists of optic nerve decompression via an internal vitreoretinal approach.

Optical coherence tomography enabled accurate assessment of the surgical outcome of macular edema. Measurements of central foveal thickness with sequential OCTs allowed us to accurately measure increases or decreases in retinal thickness with more sensitivity than slit-lamp biomicroscopy, as shown by Hee et al. Visual acuity recovery was correlated with decreased macular edema ($P = .03$). Overall, it related to better best-corrected VA in patients in whom foveal thickness values were less than 260 µm postoperatively. A previous article reported that foveal thickness measured by OCT in patients with retinal vascular occlusion did not correlate with VA, but it did serve to assess the progress of the disease in patients with low VA. Green et al and Glacet-Bernard et al suggested that the VA depends mainly on the state of the remaining circulation or the speed of its regeneration. In diabetic maculopathy the decline in VA seems to be correlated with the amount of fluid accumulation within the retinal layers. Moreover, using the retinal thickness analyzer, some correlation has been reported between macular thickness and VA in cases of retinal vascular occlusion, although with a lower linear regression value than in other diseases.

We hypothesized that improvement in our patients may occur by the following different mechanisms: by relieving mechanical pressure exerted on the central retinal vein by the distended optic nerve, thereby improving retinal blood flow; moreover, by using vitrectomy and posterior hyaloid peeling that have been shown experimentally to decrease macular edema. Since these studies have localized carbon anhydrase activity at the Mueller cells apart from at the retinal pigmentary epithelium, most probably, after vitrectomy, the exchange between the retina and the vitreous cavity is easier and helps decrease macular edema.

New chorioretinal shunts developed in 6 (42.9%) of 14 patients at the site of the radial optic neurotomy, creating a new pathway of venous outflow. We believe the collaterals formed after the neurotomy are more active in draining the edema and hemorrhages than those achieved after application of argon laser spots. In addition, their location close to the optic nerve makes them more effective. In patients in whom we observed newly formed anastomosis, the median VA achieved was better than in those patients in whom they did not exist (20/60 vs 20/110). The patients with collaterals in whom VA did not improve had subretinal macular hemorrhage and macular ischemia. This is a new finding that was not observed by Opremcak et al.

Nevertheless, the improvement in the VA of our patients has not been as spectacular as that reported by Opremcak et al. Best-corrected VA in their patients had improved by 73% vs 57.1% in our patients. Of those with increased VA, the improvement ranged from 3 to 7 lines (mean, 5 lines) in the study by Opremcak et al, whereas in our patients, the improvement ranged from 1 to 7 lines (mean, 3 lines). In their group 45% had a final best-corrected VA better than or equal to 20/70, and this result was obtained in 7 (50%) of our 14 patients, although in our group the mean preoperative VA was slightly higher.

Our results seem to be better than the natural history data; although without a randomized controlled trial and more patients, the efficacy and safety of this procedure cannot be proved. Nevertheless, as these preliminary data suggest, radial optic neurotomy seems to be a potential treatment for selected cases of this otherwise untreatable disease.

Figure 6. Patient 13. A, Fundus view of the left eye of an 80-year-old man with a visual acuity of 20/400 due to complete hemorrhagic central retinal vein occlusion. Macular and optic disc edema, venous dilation and tortuosity, and intraretinal hemorrhages are present. B, Four months after surgery, the fundus view shows resolution of retinal hemorrhages and macular edema, but the patient’s visual acuity remains the same. A chorioretinal anastomosis is clearly seen at the nasal neurotomy. C, Fluorescein angiography 4 months after surgery shows retinal ischemia involving the macular area and neovascularization in the upper nasal and temporal arcades.
Submitted for publication January 7, 2003; final revision received June 16, 2003; accepted June 20, 2003.

We thank Eduardo Hermosilla, PhD, Vall d’Hebron Hospital, for his assistance with the statistical analysis.

Corresponding author and reprints: José García-Arumi, MD, Instituto de Microcirugia Ocular, C/ Munner n° 10, 08022, Barcelona, Spain (e-mail: 17215jga@comb.es).

REFERENCES