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EMERGING DATA ON NEW THERAPIES AND TREATMENT PARADIGMS FOR RVO TREATMENT

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An Evidence-based Approach for RVO: Corticosteroids, Implants, and Anti-VEGF Therapies

BY ALLEN C. HO, MD

Macular edema in retinal vein occlusion (RVO) is the second most common cause of vision loss due to retinal vascular disease.[1-3] The two major types of RVO are branch retinal vein occlusion (BRVO) and central retina vein occlusion (CRVO). BRVO is more common than CRVO, with a 5-year incidence in 0.6% of the general population, compared with a 0.2% 5-year incidence of CRVO.[3]

BRVO presents with dilated and tortuous retinal venous system in a particular quadrant or hemisphere of the retina, and it is often associated with macular edema. Cotton wool spots, disc edema, and neovascularization may also be present. CRVO presents with hemorrhagic changes in all four quadrants of the retina and dilated and tortuous retinal veins, often described as a "blood and thunder" fundus. As with BRVO, associated cotton wool spots, optic disc edema, and neovascularization may also be present.

The exact pathogenesis is not known, but possible causes of RVO include external vascular compression, disease of the vein wall, or intravascular thrombus formation.[1-3] Once an obstruction has occurred, increased vascular pressure behind the occlusion can cause fluid and small molecules to leak across the vascular wall and into the surrounding retinal tissue, causing macular edema. Macular edema is a common complication of RVO.[4] Low-grade, chronic inflammation may also play a role in exacerbating the disease process.[3,4] This includes the production of inflammatory mediators (such as prostaglandins and IL-6), increased amounts of vascular permeability factors such as vascular endothelial growth factor (VEGF),[5] and may also include the loss of endothelial tight junction proteins.[6]

The natural history of CRVO was described first in the CVOS (Central Vein Occlusion Study), which showed that patients with perfusion who had good visual acuity at baseline, 20/40 or better, had the tendency to have better visual acuity later on.[7] Patients with baseline visual acuity worse than 20/200 for example, tended not to show improvement—80% could be expected to have visual acuity of 20/200 or worse over time. Those in the intermediate category of 20/50 to 20/200 generally stayed the same or worsened. Only about 20% would improve with better visual acuity over 3 years.[7]

The natural history of BRVO is more variable, as shown in the BVOS (Branch Vein Occlusion Study). One-third to one-half of untreated patients can return to a visual acuity of 20/40 or better within the first 6 months, and as many as 70% of patients can gain some vision over time in the first year.[8]

Diagnostic evaluation of patients with RVO includes fluorescein angiography to identify areas of leakage and also to identify areas of nonperfusion. In addition, optical coherence tomography (OCT) has become an essential tool, allowing physicians to monitor macular edema, which causes swelling and visual dysfunction and can be associated with lipid exudation and hemorrhage, and the effectiveness of therapy.

CLINICAL TRIALS IN RVO

Until recently, treatments for BRVO and CRVO were primarily guided by the BVOS and CVOS, studies that were published in the 1980s and 1990s, respectively. Over the past 2 years, there have been no fewer than five randomized clinical trials evaluating the use of pharmacologic agents for RVO. The SCORE (Standardized Care vs Corticosteroid for Retinal Vein Occlusion) BRVO and CRVO studies tested the standard of care, laser photocoagulation (BRVO) or observation (CRVO), to intravitreal triamcinolone (Trivaris, Allergan Inc.) injections; the Geneva study evaluated the use of the dexamethasone intravitreal implant (Ozurdex, Allergan Inc.) vs sham for CRVO and BRVO; and BRAVO (A phase 3, multicenter, randomized, sham injection-controlled study of the efficacy and safety of ranibizumab injection compared with sham in patients with macular edema secondary to BRVO) and CRUISE (A phase 3, multicenter, randomized, sham injection-controlled study of the efficacy and safety of ranibizumab injection compared with sham in patients with macular edema secondary to CRVO) evaluated ranibizumab (Lucentis, Genentech) vs sham for BRVO and CRVO,

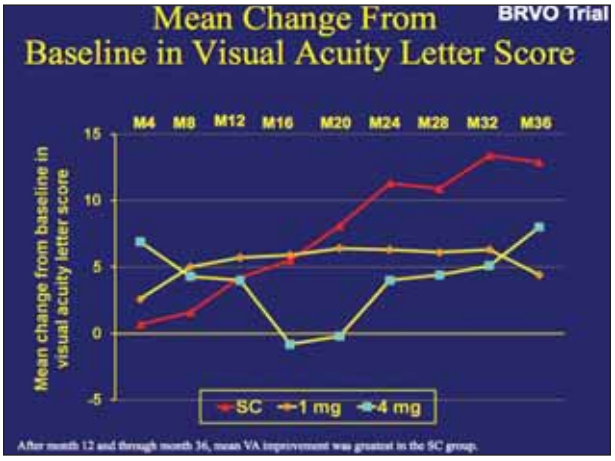

Figure 1. SCORE-BRVO. Over a period of 3 years, laser produced superior visual acuity results when compared to either 1 mg and 4 mg triamcinolone acetonide.

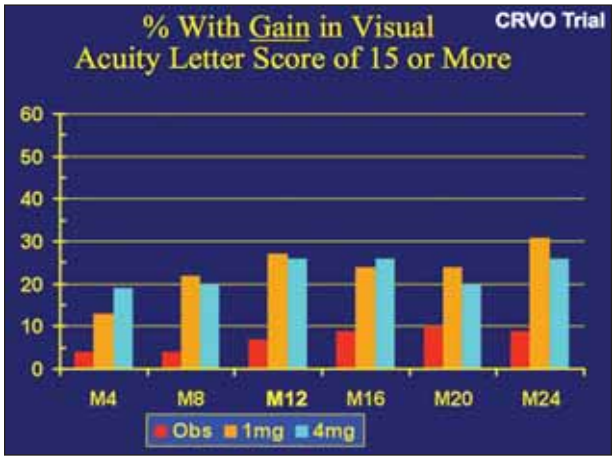

Figure 2. SCORE-CRVO. Both triamcinolone acetonide groups achieved results superior to the observation group at 12 months, as early as 4 months and continued to 24 months.

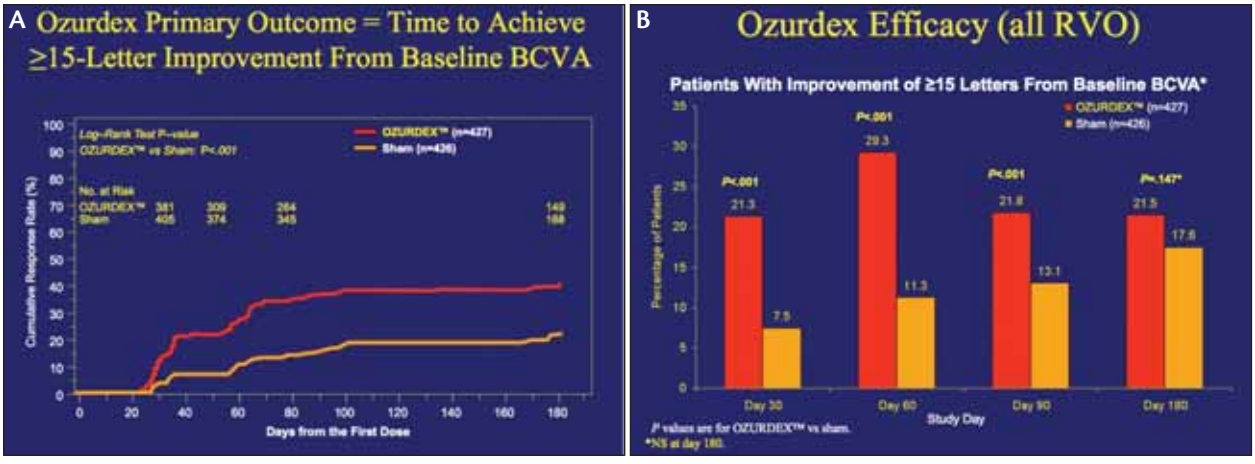

Figure 3. Geneva. The time to a 15-letter gain was significantly shorter for patients in the dexamethasone groups than in the sham groups from day 30 through day 90 (A), with peak efficacy of dexamethasone at day 60 (B).

respectively. Most recently, the Copernicus study evaluated aflibercept (VEGF Trap-Eye, Regeneron) vs sham for CRVO.

The rationale for using steroids to treat macular edema secondary to RVO is that corticosteroids provide stabilization of the blood-retinal barrier, thereby reducing macular edema. Steroids may also have an anti-angiogenic effect, reducing the vascular endothelial growth factor (VEGF) mediated increase in vascular permeability.

SCORE

SCORE compared 1 mg triamcinolone acetonide with 4 mg triamcinolone vs laser photocoagulation for BRVO and CRVO. The results of SCORE-BRVO showed that over the period of 3 years, the laser produced superior visual acuity results when compared to either 1 mg and 4 mg triamcinolone acetonide (Figure 1).[9] Additionally, the safety profile of laser was better, with fewer cataracts or increased intraocular pressures (IOPs), leading to the recommendation that laser remain the standard of care for BRVO.

The results of SCORE-CRVO showed that both triamcinolone acetonide groups achieved results superior to the observation group for vision at 12 months.[10] The visual benefit was enjoyed as early as 4 months and continued to 24 months (Figure 2). The 1 mg dose had a better safety profile than the 4 mg dose and therefore, the 1 mg dose was recommended over observation.

GENEVA

There are other steroids that have been explored for macular edema due to RVO. The 6-month Geneva trial compared the dexamethasone 0.7 mg or 0.35 mg intravitreal implant to sham for the treatment of macular edema following BRVO and CRVO.

The dexamethasone intravitreal implant at both doses

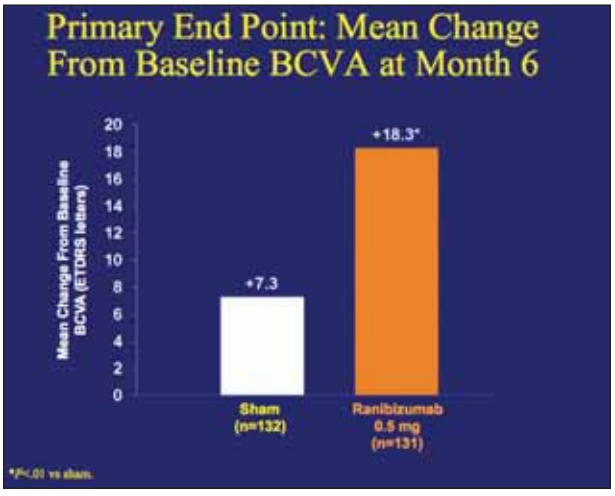

Figure 4. BRAVO. Patients in the ranibizumab 0.5 mg group achieved a mean 18.3-letter gain at month 6, compared with 7.3 letters in the sham group.

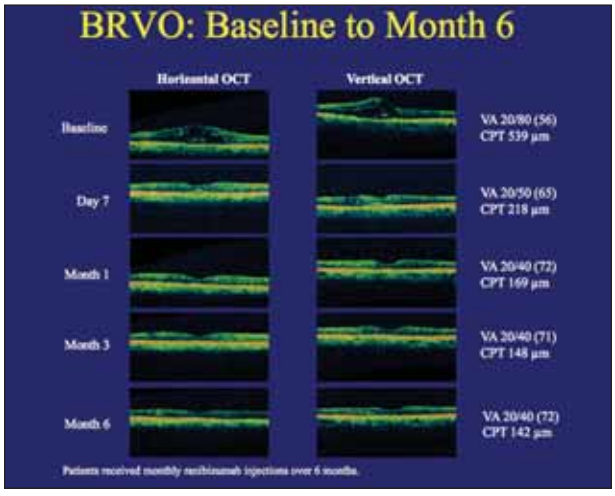

Figure 5. BRAVO. Horizontal and vertical OCTs from baseline to month 6.

provided a benefit to patients, and the time to a 15-letter gain was significantly shorter for patients in the dexamethasone groups than in the sham groups from day 30 through day 90, with peak efficacy of dexamethasone at day 60 (Figure 3).[11] Additionally, sustained-release dexamethasone reduced the incidence of 15-letter vision loss by 50% compared with sham by day 90. The persistence of efficacy was 21% for patients with BRVO and 17% CRVO at month 12.

The incidence of adverse events was relatively low, with increased IOP being the most frequently reported (25% of patients vs 1% with sham), but this is consistent with steroid therapy.

BRAVO AND CRUISE

BRAVO was a phase 3, 6-month trial with 6 months of follow-up. Patients with BRVO were randomized to treatment with either 0.3 mg or 0.5 mg ranibizumab monthly or sham for the first 6 months and then as-needed (PRN) ranibizumab in the second 6 months, with the sham group being converted to PRN 0.5 mg ranibizumab. Rescue laser was available during the first 6 months beginning at month 3 and PRN in the second 6 months beginning at month 9.

Patients in the ranibizumab 0.5 mg group achieved a mean 18.3-letter gain at month 6, compared with 7.3 letters in the sham group, which was statistically significant (Figure 4).[12] Visual acuity improvement and anatomic improvement was evident as early as day 7 for the patients treated with ranibizumab.

The horizontal and vertical OCTs of a patient from baseline to month 6 are shown in Figure 5 and are demonstrative of the typical response of patients treated with ranibizumab in BRAVO. During the 6-month period, the patient's centerpoint thickness decreased from 539 µm at

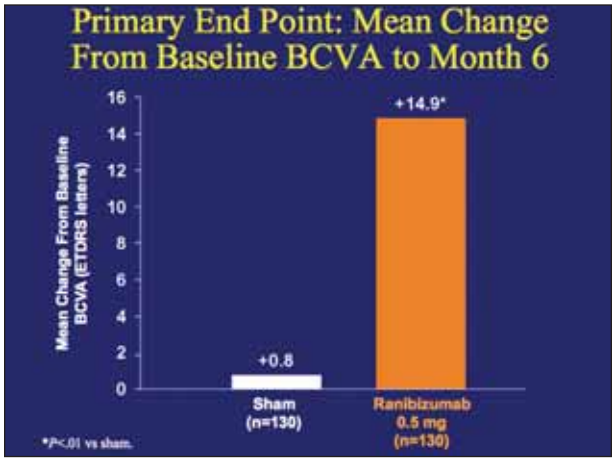

Figure 6. CRUISE. Patients in the 0.5 mg group achieved a 15-letter improvement at 6 months, compared with a less than 1 letter gain in the sham group.

baseline to 142 µm at month 6. Overall, this patient's visual acuity improved from 20/80 to 20/40.

Sixty-one percent of patients in the ranibizumab 0.5 mg group gained three or more lines of vision from baseline to month 6, compared with 29% in the sham treatment group.

Adverse events in the BRAVO study showed comparable safety side-effects and are consistent with the safety of ranibizumab for patients with AMD.

Like BRAVO, CRUISE randomized patients to either 0.3 mg or 0.5 mg ranibizumab or sham and also had a 6-month treatment period and a 6-month follow-up period with PRN dosing of ranibizumab for all groups (0.5 mg for the crossover sham group).

Patients in the 0.5 mg group achieved a 15-letter improvement at 6 months, compared with a less than 1 letter gain in the sham group (Figure 6). As with BRAVO, the

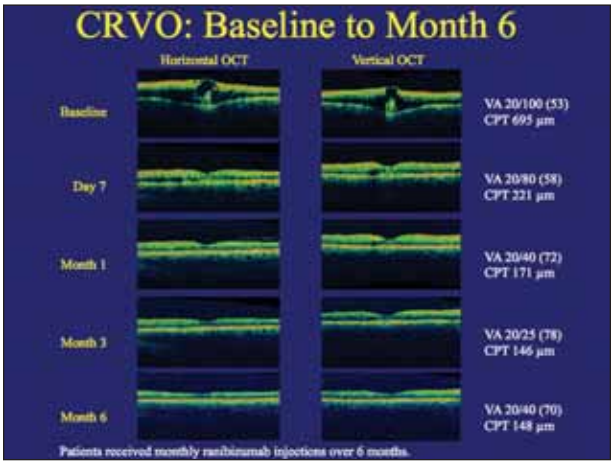

Figure 7. CRUISE. Horizontal and vertical OCTs from baseline to month 6.

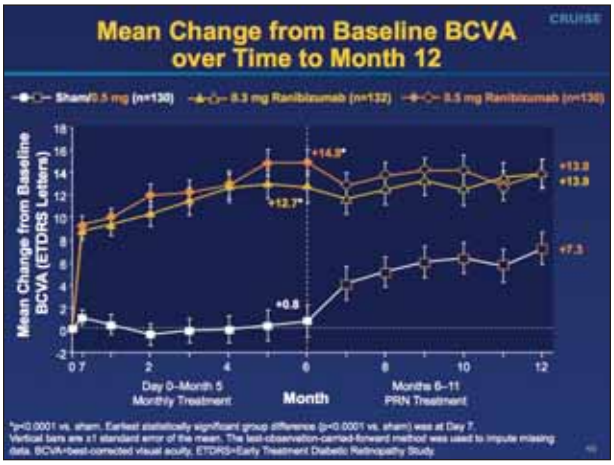

Figure 8. CRUISE. Conversion to PRN ranibizumab in the second 6 months resulted in improvement in visual acuity, but the benefit was not nearly as great as it was for those patients who received ranibizumab in the first 6 months.

change in visual acuity and anatomic improvement occurred as early as day 7 in the ranibizumab group.[13]

Figure 7 shows the horizontal and vertical OCTs from baseline to month 6 in a patient with CRVO treated with ranibizumab. The patient's center point thickness decreased from 695 µm at baseline to 148 µm at month 6, and the visual acuity improved from 20/100 to 20/40.

When patients in the CRUISE sham group converted over to the PRN ranibizumab phase in the second 6 months, there was some improvement in visual acuity, but the benefit was not nearly as great as it was for those patients who received ranibizumab in the first 6 months (Figure 8).

Adverse events in CRUISE were comparable between the sham and the ranibizumab groups.

COPERNICUS

Recently, the 6-month results of a trial comparing aflibercept to sham for CRVO were released. Copernicus randomized patients with CRVO to receive 2 mg aflibercept every 4 weeks vs sham.

At 6 months, 56% of patients in the aflibercept group gained three or more lines of vision compared with only 12% in the sham group.[14] The mean change in visual acuity for patients in the aflibercept group was a gain of 17.3 letters at 6 months vs a loss of four letters in the sham group. There was also significant and rapid reduction in retinal thickness on OCT in the aflibercept group, whereas although there was some reduction in the sham group, it was not significant.

SUMMARY

The recent data that are available have changed our thinking about the treatment of macular edema RVO. When treating macular edema secondary to RVO, we have historically been guided by data that are decades old. It is the hope that we will soon have several treatment options at our disposal so that our patients may benefit by improved outcomes. ■

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CRVO and BRVO Management

BY MICHAEL S. IP, MD

CASE 1: CRVO

A 53-year-old man with diabetes and hypertension presented with visual changes in his right eye for 1 week. His visual acuity measured 20/20 with an intraocular pressure (IOP) of 17 mm Hg and a blood pressure of 148/105. Figure 1A is a composite of the fundus photograph of the right eye showing dilated and tortuous veins in all four quadrants, mild disc edema, and dot blot hemorrhages throughout the posterior pole, indicating central retinal vein occlusion (CRVO). Optical coherence tomography (OCT), however, shows a completely flat macula with a normal foveal architecture and a good foveal reflex and depression (Figure 1B), which likely accounts for the patient's 20/20 visual acuity.

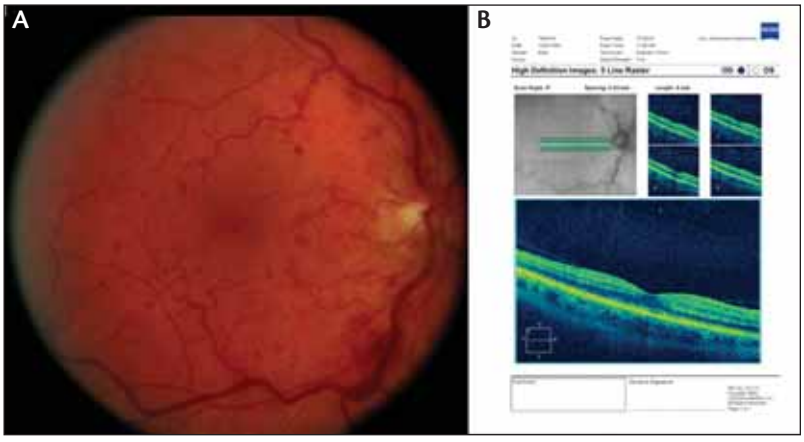

Figure 1. Case 1: Dilated and tortuous veins in all four quadrants, mild disc edema, and dot blot hemorrhages indicate CRVO (A). OCT, however, shows a flat macula (B).

We initially chose to observe this patient. Three weeks later, however, the patient presented with worsening symptoms and visual acuity decreased to 20/25. Although this was still relatively good visual acuity, the OCT showed distinct retinal thickening (Figure 2).

The treatment options that we considered were: A) observation; B) anti-VEGF therapy with either ranibizumab or bevacizumab; or C) steroid therapy with either the intravitreal dexamethasone implant or intravitreal triamcinolone acetonide.

We chose to treat the patient with bevacizumab. Although the patient had a good initial response, at 6 months follow-up, his visual acuity continued to decline to 20/80. There was worsening intraretinal hemorrhage in the macula and significant retinal thickening and retinal cyst formation (Figure 3).

At this point, we decided to administer an intravitreal injection of 1 mg triamcinolone acetonide. The patient had visual acuity and retinal thickness improvement, but had subsequent relapse that required a number of intravitreal triamcinolone acetonide injections.

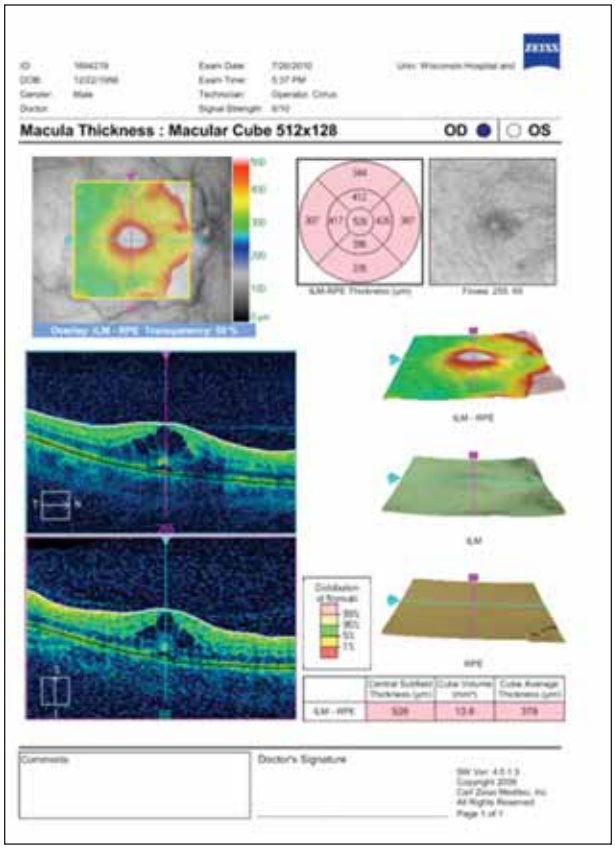

Figure 2. Case 1: Three weeks after initial presentation, the OCT shows distinct retinal thickening.

CASE 2: BRVO

An 86-year-old man presented with visual acuity that had been decreased to 20/50 for 3 weeks. His IOP was 9 mm Hg, and he had a blood pressure of 162/80. On his fundus image, a small branch retina vein occlusion (BRVO) could

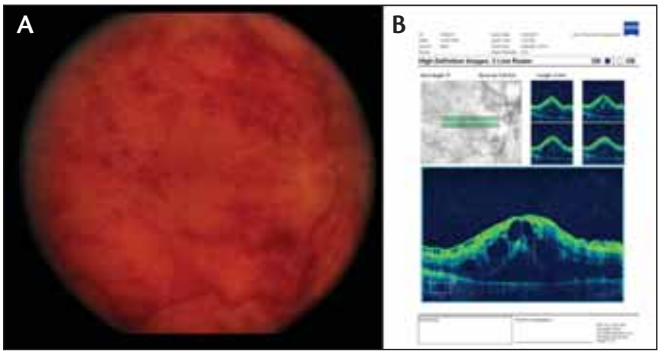

Figure 3. Case 1: At 6 months, fundus shows worsening intraretinal hemorrhage in the macula (A) and OCT shows significant retinal thickening and retinal cyst formation (B).

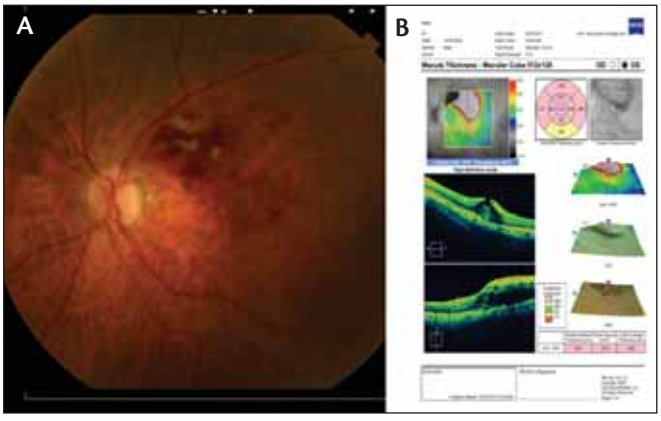

Figure 4. Case 2: At presentation, fundus shows a small BRVO with more focal intraretinal hemorrhage and a more focal area of cotton wool sports compared with CRVO (A). Retinal thickening is apparent on OCT (B).

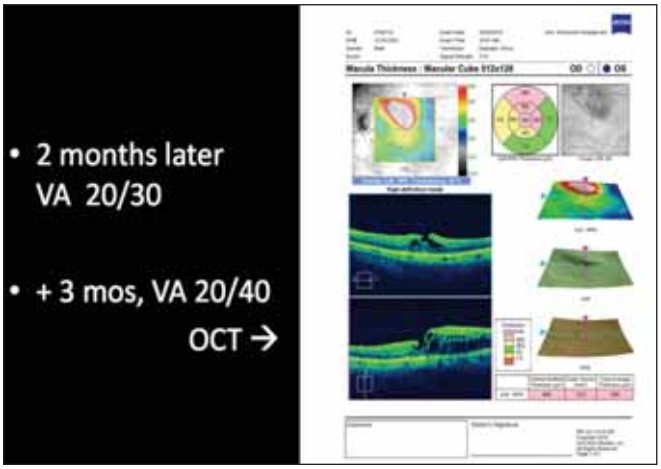

Figure 5. Case 2: The patient received two injections over 2 months and visual acuity improved to 20/30. After three more injections over 3 months, visual acuity was stable at 20/40 and OCT showed improvement.

be seen (Figure 4A) with more focal intraretinal hemorrhage and a more focal area of cotton wool spots compared with CRVO. Retinal thickening is apparent on OCT (Figure 4B); the hemorrhage can be seen in the inner layer of the retina with shadowing of the outer retina. The fovea is split in half in typical BRVO cases, one half being normal and the other half showing significant distortion of the architecture. In contrast, the entire fovea is abnormal in CRVO.

Treatment options for this patient are slightly different and include not only A) anti-VEGF; and B) the intravitreal dexamethasone implant or intravitreal triamcinolone acetonide; but also C) grid laser photocoagulation.

Grid laser photocoagulation was proven effective for patients in the original BVOS (Branch Vein Occlusion Study),[1] and these findings were supported by the SCORE (Standard Care vs Corticosteroid for Retinal Vein Occlusion)-BRVO trial,[2] which showed that laser therapy alone over 2 to 3 years was successful in treating macular edema secondary to BRVO. The effect of laser photocoagulation, however, from our clinical anecdotal experience, is gradual and was substantiated by the SCORE-BRVO study. Thus, for many patients, we choose to use anti-VEGF therapy, which was shown to provide faster visual acuity improvement for patients with BRVO in the BRAVO trial (a phase 3, multicenter, randomized, sham injection-controlled study of the efficacy and safety of ranibizumab injection compared with sham in patients with macular edema secondary to BRVO).[3]

The patient received two injections over 2 months, and his visual acuity improved to 20/30. He received three more injections over the next 3 months, and his visual acuity was stable at 20/40 with improvement on OCT (Figure 5). ■

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CRVO Management

BY RICHARD F. SPAIDE, MD

CASE 1: CRVO

The patient in Figure 1 presented to me with widespread hemorrhaging and a swollen optic nerve. All these features are consistent with CRVO. The patient also had collateral vessels on the surface of the nerve. The patient's visual acuity was 20/250 on the Early Treatment for Diabetic Retinopathy (ETDRS) scale. Fluorescein angiography (FA) showed dilated tortuous veins and a capillary leakage (Figure 2).

Our management options were: A) observation; B) antivascular endothelial growth factor (VEGF) therapy with either bevacizumab (Avastin, Genentech) or ranibizumab (Lucentis, Genentech); or C) steroids, using the intravitreal dexamethasone implant (Ozurdex, Allergan Inc) or triamcinolone acetonide. We think that observation would have no expected visual acuity improvement in this patient,

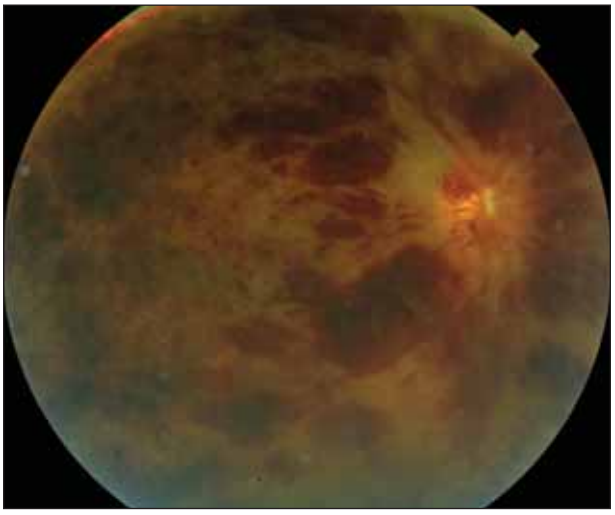

Figure 1. Case 1: Fundus photo at presentation shows widespread hemorrhaging and a swollen optic nerve.

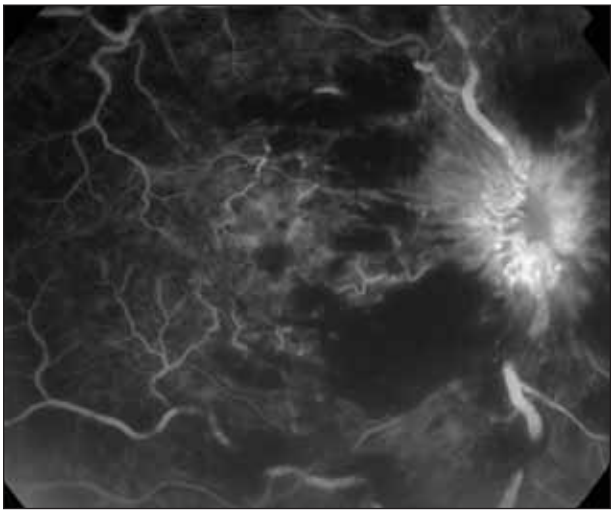

Figure 2. Case 1: FA at presentation shows dilated tortuous veins and a capillary leakage.

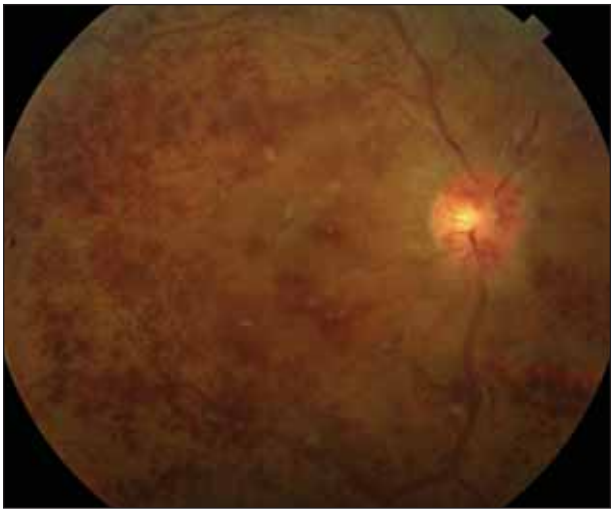

Figure 3. Case 1: After two monthly injections with ranibizumab, the fundus photo shows improvement.

Figure 4. Case 1: FA shows less dilated and tortuous veins and less vascular leakage.

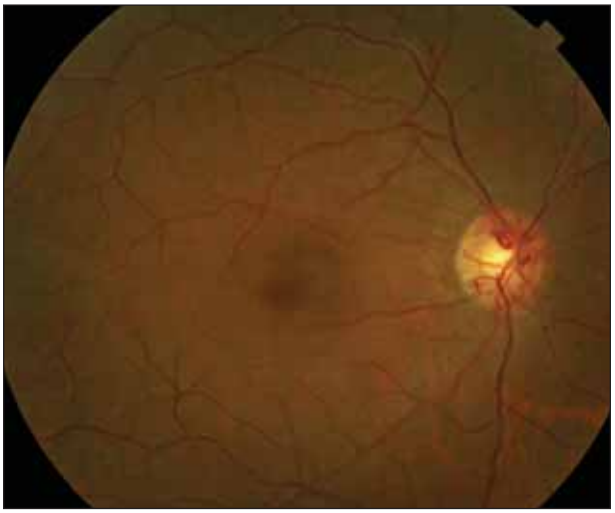

Figure 5. Case 1: At 4 months follow-up, there is an improvement in optic nerve health, but collateral vessels remain.

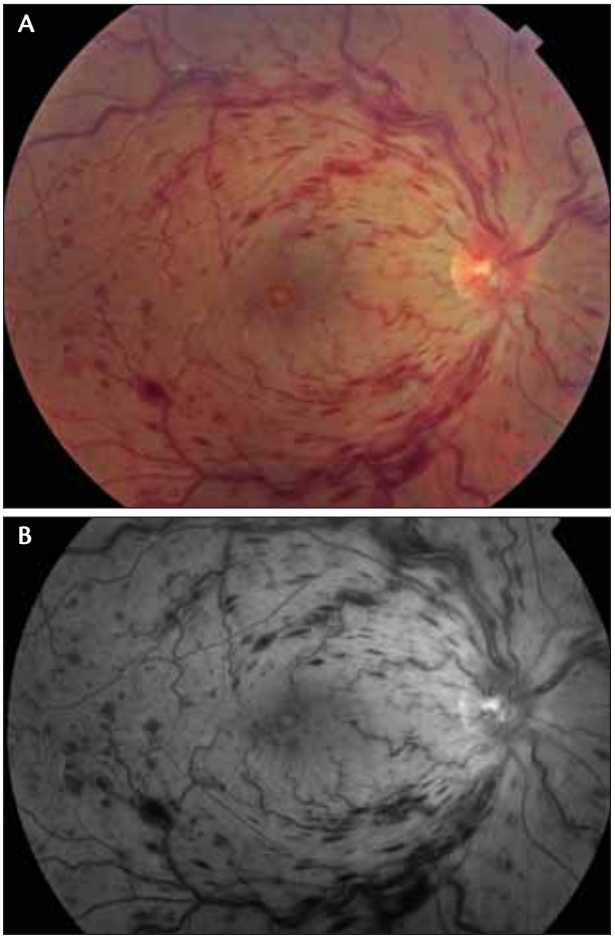

Figure 6. Case 2: Note that the central macula shows cystoid changes in the fovea (A); this cystoid change, particularly in the center part of the fovea, is more evident in the red-free photograph (B).

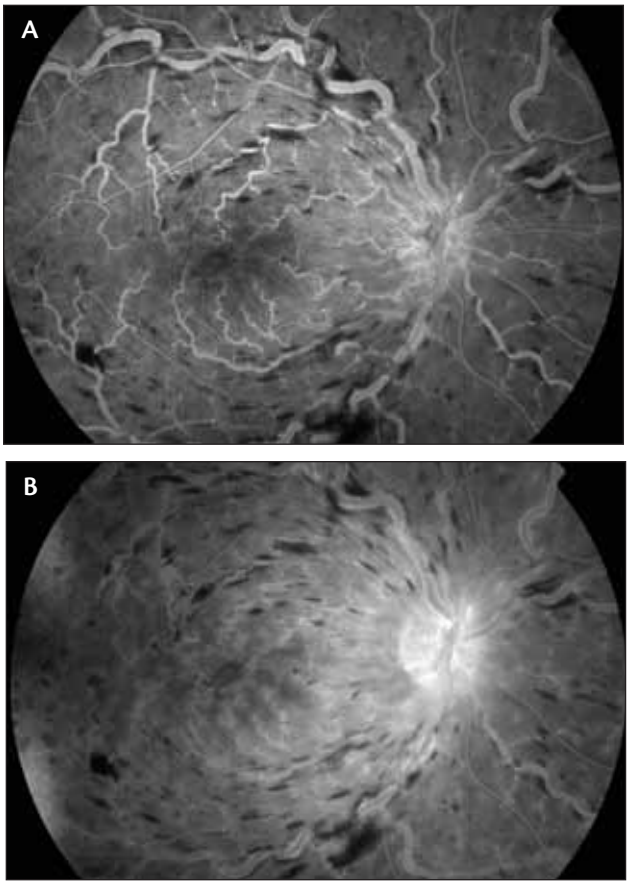

Figure 7: Case 2. The FA shows blockage by the intraretinal hemorrhage but demonstrates pronounced amounts of venous dilation and tortuosity (A). The later phase FA shows a significant amount of leakage into the retina (B).

based on past studies such as the Central Retinal Vein Occlusion Study. Anti-VEGF therapy has a high proportion of long-term response, with a marked improvement in visual acuity in treated patients, and it has a very low risk of side effects. Corticosteroids have a relatively poor long-term visual acuity response, and triamcinolone acetonide in particular has a high proportion of side effects, including the potential for glaucoma and cataracts.

For this patient, we chose anti-VEGF therapy with ranibizumab.

By 2 months, at follow-up after monthly injections, the patient's clinical appearance improved (Figure 3). The veins were much less dilated and tortuous, and there was less vascular leakage present on FA (Figure 4). There was remarkable resorption of the intraretinal hemorrhages were remarkable in this patient. In addition, the optic nerve swelling had abated, although the collaterals were still present on the surface of the nerve. The patient's visual acuity improved to 20/80 ETDRS.

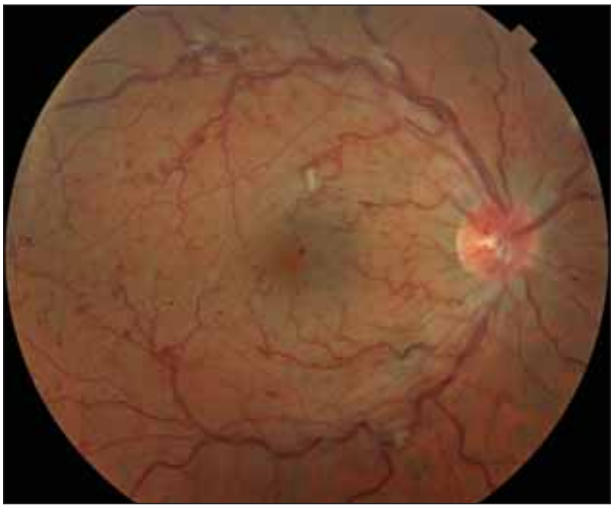

Figure 8. Case 2:At 2 months after treatment,the patient did not have any collateral vessels.

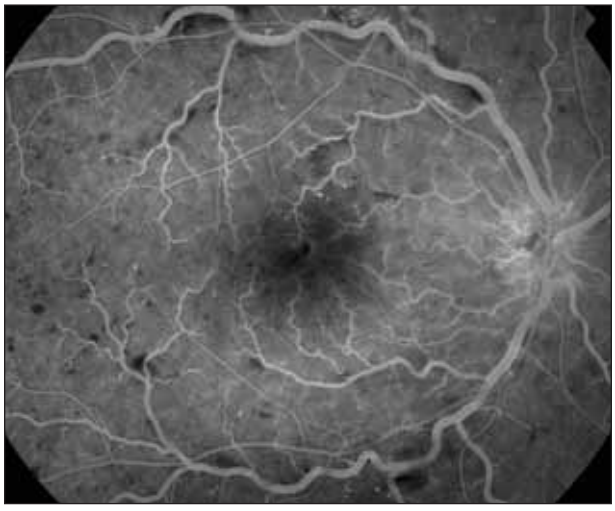

Figure 9. Case 2:At 2 months after treatment, FA shows a near cessation of leakage into the retina and hemorrhages are no longer blocking the background fluorescein.

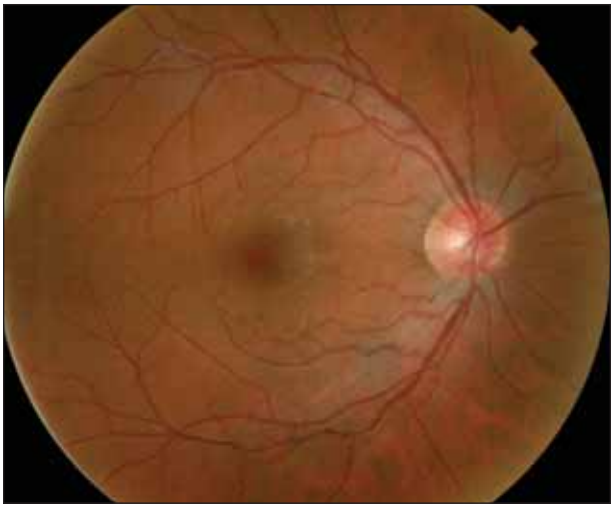

Figure 10. Case 2: Fundus photography at 38 months after treatment shows no macular edema and no intraretinal hemorrhages.

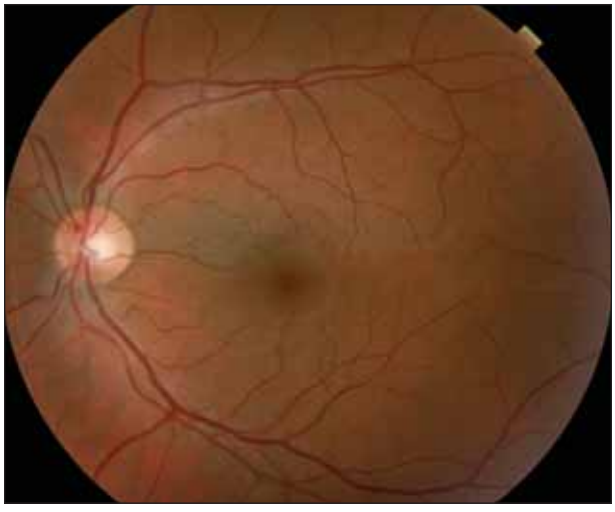

Figure 11. Case 2: The fundus photo of the fellow eye appears similar to the treated eye at 38 months in Figure 11.

At 4 months follow-up, the patient's optic nerve continued to show improvement; however, the collateral vessels remained (Figure 5). There was a continuation of remarkable intraretinal hemorrhage resorption. The patient's visual acuity was 20/50, and has remained stable for 3 years.

CASE 2: CRVO

Another patient with CRVO presented with dilated tortuous veins and intraretinal hemorrhages, and his optic nerve was swollen and erythematous. Visual acuity was 20/200. Note that the central macula in Figure 6A shows cystoid changes in the fovea; this cystoid change, particularly in the center part of the fovea, is more evident in the red-free photograph (Figure 6B).

The FA showed blockage by the intraretinal hemorrhage but demonstrates pronounced amounts of venous dilation and tortuosity (Figure 7A). A later phase FA shows a significant amount of leakage into the retina (Figure 7B).

Management options are: A) observation; B) anti-VEGF therapy; and C) either the intravitreal dexamethasone implant or intravitreal triamcinolone acetonide. Again, based on the experience from the CVOS,

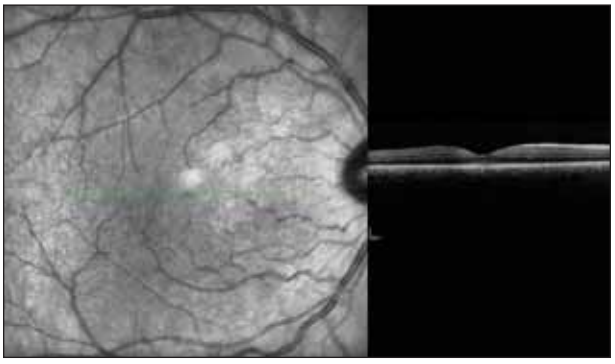

Figure 12. Case 2: At 38 months, OCT shows normal foveal contour with no increase in thickness.

we did not expect to see visual acuity improvement with observation for a patient with this level of visual acuity loss. Anti-VEGF therapy, on the other hand, has been shown to have a high proportion of long-term response with a low risk of side effects. For this patient, we chose anti-VEGF therapy using ranibizumab because we thought this treatment had the highest potential for visual acuity improvement and the lowest risk for side effects.

At 2 months, the patient had less venous dilation and tortuosity and the intraretinal hemorrhages are also starting to resorb. The patient did not have any collateral vessels (Figure 8). The FA showed a near cessation of leakage into the retina, and hemorrhages are no longer blocking the background fluorescein (Figure 9). The patient's visual acuity at 2 months had improved to 20/40.

Thirty-eight months after initial presentation, the patient's retina looked almost normal, with no collaterals in the no longer swollen disc. The patient's visual acuity was 20/20. We found it interesting that the blood vessels leading into the disc all appear to be normal. There is no macular edema and no intraretinal hemorrhages (Figure 10). The fellow eye (Figure 11) appeared similar to the treated eye, which on optical coherence tomography (Figure 12), has a normal foveal contour with no increase in thickness. ■

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