Retinal vein occlusion: Treatment

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INTRODUCTION

Retinal vein occlusion (RVO) is an important cause of visual loss among older adults throughout the world [1,2]. RVO is the second most common cause of vision loss from retinal vascular disease, following diabetic retinopathy [3]. Despite many proposed interventions, there are no treatments proven to reopen occluded retinal veins. Management is directed at secondary complications of RVO that affect vision, including macular edema, retinal neovascularization, and anterior segment neovascularization. Effective treatment for macular capillary nonperfusion, a fourth cause of visual loss in RVO, is not available.

Treatment modalities, including medical therapies, laser photocoagulation, and other surgical therapies, will be discussed here. Epidemiology, pathophysiology, clinical manifestations, and diagnosis of RVO are discussed separately. (See "Retinal vein occlusion: Epidemiology, clinical manifestations, and diagnosis".)

MANAGEMENT

Overview — There are three major anatomic types of retinal vein occlusion (RVO): central retinal vein occlusion (CRVO), hemiretinal vein occlusion (HRVO), and branch retinal vein occlusion (BRVO). CRVO occurs due to thrombus within the central retinal vein at the level of the lamina cribrosa of the optic nerve, leading to involvement of the entire retina. Less commonly, HRVO may occur when the superior and inferior retinal drainage does not merge into a single central retinal vein and one of the two trunks is occluded (generally more similar to CRVO). BRVO occurs when a vein in the distal retinal venous system is occluded, leading to hemorrhage

along the distribution of a small vessel of the retina. (See "Retinal vein occlusion: Epidemiology, clinical manifestations, and diagnosis", section on 'Classification'.)

Newly diagnosed patients with ischemic CRVO can be categorized by their degree of visual loss and findings on examination:

- **Severe visual loss** – In patients with severe visual loss (eg, 20/400 visual acuity or less), relative afferent pupillary defect (eg, greater than 1 log unit as tested with neutral density filters), diffuse retinal hemorrhage (often representing indeterminate perfusion status), or retinal capillary nonperfusion (eg, greater than 10 disk areas by fluorescein angiography), monthly examinations for six to eight months after initial diagnosis is advised in order to detect development of retinal or anterior segment neovascularization. If there is progression of visual decline or increasing hemorrhage, monthly examinations should be extended until stability can be established for at least six to eight months.

- **Non-severe visual loss** – In patients without severe visual loss or any of the other above characteristics, a follow-up examination in one to three months with subsequent lengthening of intervals is advised until stability can be established at least 6 to 12 months.

In patients with BRVO or CRVO without macular edema or neovascularization, we suggest observation rather than initiation of treatment.

Treatment is indicated in patients with RVO for macular edema, retinal neovascularization, and anterior segment neovascularization. Goals of treatment are to maintain central visual acuity by minimizing the effects of chronic macular edema, reducing the risk of bleeding into the vitreous cavity by producing regression of retinal neovascularization, and preventing neovascular glaucoma that can occur in eyes with severe disease. Treatment also involves management of predisposing risk factors, such as diabetes and hypertension. (See "Overview of general medical care in nonpregnant adults with diabetes mellitus" and "Overview of hypertension in adults".)

Macular edema — Pharmacologic treatment with intravitreal anti-vascular endothelial growth factor (VEGF) agents is first-line therapy for macular edema [4-8]. Intravitreal glucocorticoid therapy is considered an alternative for patients with edema refractory to anti-VEGF monotherapy [9]. Grid laser photocoagulation therapy is another distant alternative for treatment of BRVO [10] but has limited, if any, benefit in patients with CRVO. There is no established role for prophylactic therapy for macular edema with either anti-VEGF therapy or laser therapy.

Retinal neovascularization — Scatter laser photocoagulation is recommended to reduce the risk of visual impairment from vitreous hemorrhage (secondary bleeding into the vitreous

cavity) for established neovascularization of the retina or optic disc due to RVO [11]. In BRVO, scatter laser photocoagulation is usually applied to the portion of the retina in the distribution of the occluded vein where retinal capillary nonperfusion most likely exists. The laser application is often guided by fluorescein angiography. In CRVO, the scatter laser treatment is applied throughout the fundus periphery, known as panretinal scatter laser retinal photocoagulation.

Anti-VEGF treatment also leads to regression of neovascularization and is often used initially to supplement laser treatment for that purpose, particularly if there is macular edema or extensive intraretinal or vitreous hemorrhage limiting ability to perform laser treatment [12].

Anterior segment neovascularization — Patients with established anterior segment neovascularization are at risk of glaucoma due to neovascularization that obstructs the trabecular meshwork and produces synechial angle closure glaucoma. Once neovascularization is observed in the anterior segment, scatter retinal photocoagulation is recommended to reduce this risk. In addition, intravitreal anti-VEGF therapy may be used as an adjunct to laser treatment. Similar to above, anti-VEGF therapy may be used as a temporizing measure because the effects of laser take two to four weeks to develop. Additionally, in some patients laser cannot be performed initially because of anterior segment bleeding, elevated intraocular pressure, corneal edema, miosis, or extensive retinal hemorrhage preventing laser uptake.

Patients with severe (ischemic) CRVO are at particularly high risk for neovascular glaucoma, often within the first few months of diagnosis, and should be observed at least monthly for development of anterior segment neovascularization during this period. Such patients often present with severe visual loss, retinal hemorrhage, or evidence of extensive retinal capillary nonperfusion. Routine panretinal photocoagulation in patients with CRVO, prior to onset of anterior segment neovascularization, has not been shown to be beneficial [13].

MEDICAL THERAPY

Intravitreal injections of pharmacologic agents have become first-line treatment for symptomatic macular edema from retinal vein occlusion (RVO). Long-term efficacy results (over two years) are available only for ranibizumab and aflibercept. Data are limited regarding combination therapy with intravitreal anti-vascular endothelial growth factor (VEGF) and corticosteroids, although one study showed no clear differences between anti-VEGF treatment combined with sub-Tenon triamcinolone compared with anti-VEGF monotherapy [14]. Technology assessments for the American Academy of Ophthalmology found that intravitreal anti-VEGF treatment was safe and effective for treatment of macular edema associated with

central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO) and that earlier treatment was associated with improved visual outcomes [15,16].

Drawbacks of intravitreal therapy include the need for multiple injections over periods of months to years and a remote risk of infectious endophthalmitis from the injection procedure that can result in catastrophic visual loss. Other risks of intravitreal injection include cataract, retinal detachment or tears, and intraocular hemorrhage. Safety related to the systemic effects of repeated courses of intravitreal anti-VEGF therapy remains a topic of debate.

Vascular endothelial growth factor inhibitors — Because of clinical trials indicating the potential for visual improvement, intravitreal anti-VEGF drugs are first-line therapy for macular edema associated with BRVO and CRVO.

VEGF is a potent mitogen and vascular permeability factor that plays a pivotal role in neovascularization. Levels of VEGF are elevated in patients with CRVO [17]. Anti-VEGF molecules limit the destructive effects of choroidal neovascular membranes in patients with exudative age-related macular degeneration. (See "Age-related macular degeneration", section on 'VEGF inhibitors'.)

VEGF inhibitors in patients with RVO are hypothesized to limit macular edema and improve vision by decreasing vascular permeability. Several anti-VEGF intravitreal agents are available for clinical use: bevacizumab, ranibizumab, aflibercept, faricimab, and ranibizumab-nuna. Only ranibizumab (or its biosimilars) [18,19], faricimab [20], and aflibercept are approved for treatment of RVO by the US Food and Drug Administration (FDA). There are no evidence-based criteria for determining which of these drugs to use, and clinical decisions are based mostly on medication cost.

Treatment is instituted soon after diagnosis of macular edema because a treatment delay of six months was found to be associated with failure to achieve the same degree of visual recovery when compared with immediate treatment [5,8,21]. In many clinical practices, periodic intravitreal injections are performed as necessary until macular edema remits, at which time treatment is discontinued and reinstituted as needed (a "treat and observe" approach). In one study in patients with either BRVO or CRVO whose disease was stable after seven months of monthly ranibizumab injections, administering ranibizumab on an as-needed schedule, compared with continued monthly injections, resulted in no difference in visual acuity at 15 months [22]. In an alternative approach, intervals between treatments are gradually increased to a point when the clinician deems observation to be appropriate (a "treat and extend" approach). There are no studies comparing these two approaches in RVO management.

Evidence of effectiveness for BRVO — Several observational studies and randomized trials have found intravitreal anti-VEGF drugs to be effective and well-tolerated for the treatment of macular edema from BRVO [23-28].

In the six-month BRAVO trial, in which 397 patients with macular edema from BRVO were randomly assigned to ranibizumab (0.3 or 0.5 mg intraocularly) or sham, more patients receiving low-dose and high-dose ranibizumab had improvements in visual acuity of at least 15 letters (55 and 61 versus 29 percent) and achieved visual acuity of 20/40 or better (68 and 65 versus 42 percent) [25]. Treatment with ranibizumab results in durable benefit lasting four years or more [26]. The addition of laser therapy to ranibizumab does not lead to better functional outcomes in patients with BRVO [24].

In a 52-week trial, monthly intravitreal aflibercept for the first 24 weeks, followed by aflibercept every eight weeks thereafter, was shown to be superior to grid laser with a mean gain of 17.1 letters in the aflibercept group compared with 12.2 letters in the laser group [29]. Beyond 24 weeks, rescue therapy with opposite-arm treatment was permitted, with intravitreal aflibercept rescue therapy showing substantial benefit in patients originally assigned to grid laser.

Intravitreal bevacizumab has also been used to treat BRVO with macular edema. Favorable outcomes were reported in a two-year observational study [28]. Although not approved for intravitreal use, bevacizumab is considerably less expensive than ranibizumab and aflibercept. In patients with neovascular age-related macular degeneration, bevacizumab has demonstrated equivalent efficacy in comparison with ranibizumab. (See "Age-related macular degeneration", section on 'VEGF inhibitors'.)

Evidence of effectiveness for CRVO — Several observational studies and randomized trials have found intravitreal anti-VEGF drugs to be effective and well-tolerated for the treatment of macular edema from CRVO [4-6,8,30,31].

In the six-month CRUISE trial, in which 392 patients with macular edema from CRVO were randomly assigned to ranibizumab intraocular injections (0.3 or 0.5 mg) or sham injections, more patients receiving ranibizumab had greater improvements in visual acuity of at least 15 letters (46 and 48 percent versus 17 percent) [5]. Ranibizumab-treated eyes were also more likely to achieve visual acuity of ≥20/40 (44 and 47 percent versus 21 percent). Patients received intraocular ranibizumab injections as often as monthly if they met prespecified criteria (best-corrected visual acuity ≤20/40 or mean central subfield thickness >250 micron on optical coherence tomography).

An update to the CRUISE trial has extended and confirmed the results through month 12, with greater improvements in visual acuity in the ranibizumab group [6]. In a long-term follow-up of

a subset of patients from the CRUISE trial, edema resolution occurred at 49 months in 44 percent of patients with CRVO treated with ranibizumab, but 56 percent still required frequent injections [26].

A similarly designed study (COPERNICUS) involving intravitreal aflibercept in patients with CRVO has also shown a beneficial effect on visual acuity at six months and one year [8], although visual acuity diminished somewhat when the dosing schedule changed at 24 weeks from monthly injections to injections as needed and to less frequent monitoring (at least quarterly) beyond one year [32]. Intravitreal aflibercept for CRVO was also compared with sham injections in the GALILEO Study, in which patients received injections every four weeks to week 24 and then as needed up to 76 weeks; patients in the aflibercept group had more visual and anatomic improvement at both 24 and 76 weeks, compared with the sham group [30].

In the United States, bevacizumab is not approved to treat CRVO with macular edema, but it appears to be as effective as aflibercept and is considerably less expensive. In a randomized controlled clinical trial of 362 patients with CRVO or hemiretinal vein occlusion, improvements in the visual acuity letter score were comparable in patients using intravitreal bevacizumab and aflibercept after six months of therapy (mean increase from baseline of 18.6 versus 18.9, respectively) [31]. A beneficial effect at 12 months persisted, though not definitely to a similar degree, with either bevacizumab or aflibercept in patients who responded well after the initial six months of treatment, even when switched over the subsequent six months from a monthly to a treat-and-extend (TAE) regimen [33].

Intravitreal glucocorticoids — Intravitreal glucocorticoids are considered second-line pharmacologic therapy for RVO. Randomized trials and cohort studies have found that intravitreal glucocorticoid injections may improve visual acuity in patients with BRVO [7,9] and with CRVO [34]; however, a systematic review identified substantial loss of follow-up data in one of the randomized trials and concluded that evidence of visual improvement from intravitreal steroids for CRVO with macular edema should be interpreted with caution [35]. Intravitreal glucocorticoid injections, however, may cause adverse effects not commonly observed with anti-VEGF therapy, including cataract formation and moderate or severe increases in intraocular pressure [7]. Patients require periodic monitoring for elevation of intraocular pressure and treatment of glaucoma, if present [36].

Intravitreal glucocorticoids are administered as injections or as a slow-release dexamethasone implant delivered via a proprietary injection device. Some forms of intravitreal corticosteroid treatment, such as the dexamethasone intravitreal implant or intravitreal injections of triamcinolone acetonide, involve less frequent injections (every three to four months rather than monthly) than do intravitreal VEGF inhibitors. Reasonable candidates for intravitreal

glucocorticoid therapy include patients who have responded poorly to anti-VEGF therapy or who are adverse to frequent intraocular injections. Pseudophakic patients without glaucoma are considered especially favorable candidates for glucocorticoid therapy because they are no longer at risk for steroid-induced cataract formation. Similarly, patients who have had previous glaucoma filtering procedures, and are thus at lower risk of steroid-induced elevated intraocular pressure, may be appropriate candidates.

Effectiveness of intravitreal injection of triamcinolone — In the Standard Care versus Corticosteroid for Retinal Vein Occlusion (SCORE) trial for BRVO, intravitreal triamcinolone in doses of 1 or 4 mg were compared with macular grid laser photocoagulation in 411 patients with visual acuity loss from BRVO-related macular edema [9]. There was no difference in visual acuity between the treatment arms. Increasing intraocular pressure and cataract formation were more common in the triamcinolone arms, particularly the 4 mg treatment group. Given these complications, other therapies such as intravitreal anti-VEGF agents or macular grid laser photocoagulation are preferred options for patients with persistent vision loss secondary to BRVO-related macular edema. (See 'Vascular endothelial growth factor inhibitors' above and 'Retinal laser photocoagulation' below.)

The SCORE trial for CRVO compared intravitreal triamcinolone (1 or 4 mg) with observation in patients with macular edema secondary to CRVO [34]. Patients randomly assigned to triamcinolone had improved visual acuity after 12 months. The 4 mg triamcinolone group had higher rates of intraocular pressure elevation and cataract compared with 1 mg and control groups. After initial injection of triamcinolone, patients were retreated as needed at four-month intervals. This trial suggests that patients with visual acuity loss from macular edema secondary to CRVO may benefit, relative to observation alone, from treatment with 1 mg of preservative-free triamcinolone at baseline and at four-month intervals as needed for one year.

Effectiveness of glucocorticoid implant — In two six-month randomized trials in which 1267 patients with macular edema from either BRVO or CRVO were randomly assigned to dexamethasone intravitreal implant (0.35 or 0.7 mg) versus sham, patients receiving implant treatment were more likely to have improvement of at least 15 letters over a 60- to 90-day observation period [37]. At the end of six-month follow-up, improvement of visual acuity persisted for both treatment groups compared with the control group. A single treatment was administered, and a low rate of adverse effects was observed.

An intravitreal glucocorticoid implant may provide a more convenient dosing regimen and possibly a safer alternative to more frequent injections of other agents. In a retrospective observational study of patients with BRVO and macular edema, functional and anatomic outcomes at 12 months were the same for patients treated with monthly intravitreal

bevacizumab or dexamethasone implant injection at six-month intervals [38]. A dexamethasone implant (0.7 mg) is available for treatment of CRVO and BRVO. Although the risk of elevated intraocular pressure and cataract formation is higher than that for anti-VEGF agents, the risk profile of the dexamethasone implant may be acceptable in some patients because of failure to respond to anti-VEGF therapy, even for durations of use longer than those studied in clinical trials.

Antithrombotic and thrombolytic therapy — Thrombosis is one of the main pathogenic causes of RVO. (See "Retinal vein occlusion: Epidemiology, clinical manifestations, and diagnosis", section on 'Pathophysiology'.)

Antithrombotic and thrombolytic medications, including aspirin, ticlopidine, heparin, streptokinase, and recombinant tissue plasminogen activator, have been studied for the treatment of both BRVO and CRVO. A systematic review including four small randomized trials in BRVO and five small randomized trials in CRVO, however, found limited evidence of effectiveness [39,40]. Thus, except in cases in which the primary etiology of vein occlusion is due to underlying coagulopathy or inflammatory disease, there is no current indication for antithrombotic, thrombolytic, or immunosuppressive medication for the treatment of RVO.

RETINAL LASER PHOTOCOAGULATION

Retinal laser photocoagulation is first-line therapy for neovascular complications of retinal vein occlusion (RVO).

For the treatment of macular edema from RVO, grid laser photocoagulation is generally considered to be second-line to anti-vascular endothelial growth factor (VEGF) and intravitreal glucocorticoid therapies and often reserved for patients who are averse to intravitreal injections. Combination intravitreal therapy and grid laser photocoagulation is used in some patients as well but lacks robust clinical data. Laser treatment is noninvasive and accomplished in one or more clinic sessions, whereas anti-VEGF therapy may require multiple intravitreal injections over a period of months or years without a known, pre-established endpoint for a given patient.

There is no established role for laser photocoagulation as prophylaxis for macular edema or neovascularization.

There are three main forms of laser photocoagulation treatment:

- Peripheral or sector scatter photocoagulation, which involves moderate laser burns distributed in a regularly spaced pattern throughout the portion of the peripheral retina in the distribution of the occluded vein. It is used for neovascularization in patients with branch retinal vein occlusion (BRVO).

- Panretinal scatter photocoagulation, which involves photocoagulation of the entire peripheral retina and is used for retinal or anterior segment neovascularization in patients with central retinal vein occlusion (CRVO).

- Macular grid photocoagulation, which creates small, mild burns in a grid-like distribution over the macula and is used for macular edema in patients with BRVO who are adverse to intravitreal therapy. Grid laser photocoagulation may cause symptoms of permanent paracentral scotoma. Withholding grid laser for a period of three months after onset of symptoms is often recommended because macular edema can spontaneously remit. Grid photocoagulation has become second- or third-line treatment for macular edema after intravitreal anti-VEGF therapy and possibly intravitreal corticosteroid therapy.

Complications — Although laser treatments for RVO are well-tolerated, there are rare complications (<1 percent) that can lead to worsening visual acuity:

- Retinal scarring
- Choroidal neovascularization
- Subretinal fibrosis
- Visual field deterioration
- Paracentral scotomata

SURGERY

Surgical intervention is not considered as primary therapy and is reserved for cases where neovascular complications are unable to be sufficiently treated by medical or laser approaches.

Surgery for branch retinal vein occlusion (BRVO) is largely restricted to vitrectomy (surgical removal of the vitreous humor) with or without an arteriovenous sheathotomy, in which the overlying retinal artery is separated from the vein to improve blood flow. In a randomized trial comparing intravitreal triamcinolone versus vitrectomy/arteriovenous sheathotomy in 40 eyes with BRVO-related macular edema, no differences were found in visual acuity or foveal thickness at three and six months [41]. Definitive conclusions cannot be drawn from this study due to the short follow-up period and small sample size.

Studies evaluating surgical options in central retinal vein occlusion (CRVO) are limited to small case series [42]. These surgical options include:

- Vitrectomy
- Radial optic neurotomy
- Chorioretinal venous anastomosis to bypass the site of obstruction of venous outflow
- Direct injection of thrombolytic medication into the retinal vein via retinal vein cannulation

In clinical practice, vitrectomy is commonly performed for removal of persistent vitreous hemorrhage or management of neovascular complications secondary to BRVO or CRVO. In those eyes where the vitreous hemorrhage had blocked the ability to perform needed sector or panretinal scatter laser photocoagulation for control of the neovascular process, laser treatment can be done intraoperatively after the hemorrhage has been surgically evacuated.

Sheathotomy, radial optic neurotomy, chorioretinal venous anastomosis, and direct injection of thrombolytic medication are rarely done in clinical practice given unclear benefit and high risks associated with these procedures.

We consider vitrectomy to be indicated for complications of BRVO or CRVO such as persistent vitreous hemorrhage or traction retinal detachment from severe neovascularization despite laser photocoagulation and medical therapy. (See 'Retinal laser photocoagulation' above and 'Medical therapy' above.)

SUMMARY AND RECOMMENDATIONS

- In newly diagnosed patients with ischemic central retinal vein occlusion (CRVO) with severe visual loss (eg, 20/400 visual acuity or less), relative afferent pupillary defect, diffuse retinal hemorrhage (often representing indeterminate perfusion status), or retinal capillary nonperfusion (eg, greater than 10 disk areas by fluorescein angiography), monthly examinations for six to eight months after initial diagnosis is advised in order to detect development of retinal or anterior segment neovascularization. If there is progression of visual decline or increasing hemorrhage, monthly examinations should be extended until stability can be established for at least six to eight months. (See 'Overview' above.)

- In patients without severe visual loss or any of the other above characteristics, a follow-up examination in one to three months with periodic monitoring or lengthening of intervals is advised until stability can be established for at least 6 to 12 months. (See 'Overview' above.)

- In patients with branch retinal vein occlusion (BRVO) or CRVO without macular edema or neovascularization, we suggest observation rather than initiation of treatment (**Grade 2C**). (See 'Overview' above.)

- In patients with macular edema from BRVO or CRVO that has caused symptomatic visual loss, we recommend intravitreal anti-vascular endothelial growth factor (VEGF) treatment as first-line therapy (**Grade 1A**). Second-line therapies are dexamethasone 0.7 mg implant or intravitreal triamcinolone acetonide. In patients with BRVO and visual loss who are averse to intravitreal injection treatments, if duration of symptoms is at least three months, we suggest macular grid laser photocoagulation treatment rather than observation (**Grade 2B**). (See 'Macular edema' above.)

- In patients with neovascularization from BRVO, we suggest peripheral scatter photocoagulation rather than observation (**Grade 2C**). In patients with neovascularization from CRVO, we suggest panretinal photocoagulation rather than observation (**Grade 2C**). (See 'Retinal neovascularization' above and 'Retinal laser photocoagulation' above.)

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