Branch Retinal Vein Occlusion

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Retinal Blood Supply

When we talk about the Branch Vein occlusion of retina, we might as well devote a couple minutes on the blood supply by the arterial system and the drainage by the venous system.

The central retinal artery enters the globe from the center of the optic nerve, immediately adjacent and parallel to the exiting central retinal vein.

The blood supply to the inner layers of the retina is exclusively from the central retinal artery except in 15% to 30% where cilioretinal artery is present. The outer retina is supplied by the choroidal circulation.

After the first or second bifurcation, the so called retinal arteries and veins are actually arterioles and venules due to loss of elastic lamina and a continuous smooth muscle layer.

Occlusion of the venous drainage system.

Occlusion of the Central venous drainage system has a peak incidence in individuals of 60 years plus, and predominantly men.

Close proximity of the central retinal artery and vein in the region of the lamina and their common adventitial sheath are critical anatomic factors.

The venous occlusion may effect by a common pathology-- The central retinal vein, the dual trunk of central retinal vein (seen in about 20% individuals) and a branch vein. Names are given accordingly. CRVO, Hemispheric Retinal vein occlusion (HRVO) and BRVO.

Branch Retinal Vein Occlusion

BRVO

A relatively common cause of retinal vascular disease. Incidence - male and females - equal. Age incidence - 60 plus. Site of occlusion is rarely other than at A-V crossing. Frequently a unilateral condition.

Cause

Compression of the vein by the sclerotic artery, leading to turbulent blood flow, endothelial damage, and thrombus formation is the postulated etiology.

Risk Factors

History of systemic hypertension, cardiovascular disease, glaucoma, increased body mass index at 20 yrs. old, higher serum levels of alpha-2, and shorter axial lengths of the eye.

Clinical Features

1. Branch retinal vein occlusion has many similarities to diabetic retinopathy, including edema, capillary nonperfusion, neovascularization, and vitreous

2.
2. Diabetic retinopathy is a continued progressive abnormality of the microcirculation that may involve the entire fundus, whereas in branch vein occlusion the abnormalities are caused by a single acute event.

3. Almost always of sudden onset of blurred vision or field defect, segmentally distributed intraretinal hemorrhage. Intraretinal hemorrhage is less marked in nonischemic occlusion and much more marked if occlusion is ischemic.

4. The location of obstruction determines the distribution of the intraretinal hemorrhage; obstruction at the optic nerve head, will involve two quadrants of the fundus whereas occlusion at peripheral to the disc, one quadrant or less may be involved.

5. If the venous blockage is peripheral to tributary veins draining the macula, there may be no macular involvement and no decrease in visual acuity. An incomplete block at the A/V crossing may progress to more complete occlusion and hemorrhage becomes more extensive in succeeding weeks to months.

6. After an year or so the, fundus picture may become more benign and the intra retinal hemorrhages may be absorbed but the venous abnormalities that occurred may persist including capillary non perfusion, dilation of capillaries and collateral vessel formation.

Vision Limiting Complications

1. Macular edema.
2. Macular non perfusion.
3. Vitreous hemorrhage from neovascularization.

In the acute phase of the disease with a substantial intraretinal hemorrhage, it may be impossible to evaluate potential vision, patient should be followed every 2 to 3 months until there is sufficient clearing of hemorrhage to allow evaluation by fluorescein angiography.

Although it may be difficult to provide a prognosis in the acute phase, it is helpful to recognize that about one third to one half of patients with BRVO have a return of vision to 20/40 or better without therapy.

Fluorescein Angiography and its prognostic value

After the acute phase of the BRVO has passed and intra retinal hemorrhage has mostly absorbed, which usually takes 3 to 6 months, Fluorescein angiography should be obtained.

Fluorescein angiography is the only technique that will accurately define the capillary abnormalities in BRVO; it is therefore particularly important that high-quality angiography be obtained.

When Fluorescein angiography demonstrates macular edema with cystoid involvement of the fovea, but no capillary nonperfusion, it is presumed that the macular edema is the cause of vision loss and about one third of patients will spontaneously regain some vision.

Foveal leak

However, patients who have had decreased vision for over 1 year as a result of macular edema are much less likely to regain vision spontaneously.

Complications of BRVO

Retinal neovascularization may develop when the capillary non-perfusion area is more than five disc diameters as visualized by Fluorescein angiography.

Of large branch vein occlusions (involving a quadrant or more), about 50% are associated with a large area of capillary non-perfusion; of this 50% about 40% will develop neovascularization.
Retinal or disc neovascularization, or both, may develop at any time within the first 3 years after an occlusion but are most likely to appear within the first 6 to 12 months after the occlusion.

Those who develop neovascularization, about 60% of them experience episodes of vitreous hemorrhage. If neo-vascularization left untreated it can lead to prolonged visual disability in the affected eye.

Iris neovascularization is a rare complication of BRVO; diabetes may increase this risk. Retinal neovascularization is particularly difficult to recognize in BRVO because the collaterals that develop frequently may mimic neovascularization.

In BRVO Neovascularization on the retina frequently mimics the collaterals on the retina.

F.F.A differentiates between the collaterals and neovascularization

Ret. Neo Vascularization

TREATMENT of BRVO

(a). Medical
(b) LASER
(c). Surgical

Medical –Control of provocative factors like hypertension, Diabetes, Cardiovascular diseases and blood cholesterol have been recommended. Anticoagulant therapy has not shown to be beneficial in either the prevention or the management of BRVO

Laser therapy in BRVO -

Familiarity with the laser treatment technique is required. Important variables, such as residual intra-retinal hemorrhage, thickness and extent of retinal edema, location of neovascularization and presence of retinal traction they all influence the exact mode of therapy.

High quality FFA is essential before any planning for Laser therapy. Laser therapy is indicated only where the following criteria are met

Eligibility Criteria for Laser therapy

1. Fluorescein proven per fused macular edema involving the foveal center,
2. Absorption of intraretinal hemorrhage from the foveal center,
3. Branch retinal vein occlusion of 3 to 18 months’ duration.
4. No diabetic retinopathy,
5. Vision reduced to 20/40 or worse after best refraction.

Treatment of Macular Edema

Grid laser applied through out the leaking area. Extending from major vascular arcade to the Edge of FAZ.

In grid laser, energy absorption occurs at R.P.E level

In grid P.C laser is not applied directly and immediately close to leaking and dilated capillary vasculature.

Grid Laser PC FA Transient Face

Laser Parameter for Grid.

1. Duration 0.1 sec.  
2. Spot Size 100 micron  
3. Power Medium white burn

Mechanism of Grid Laser in macular edema

How laser P.C. reduces edema is not clear. Probable mechanisms are: -

1. Laser tickles and activates R P E pump to reduce edema.
2. Laser produces thinning of retina and Choroidal vasculature helps in reducing edema by auto regulatoryconstriction of retinal vasculature in the leaking area.
Treatment of Neovascularization in BRVO study

Prophylactic scatter laser P.C. can lessen subsequent neovascularization and, if neovascularization already exists, then peripheral scatter laser P.C. can reduce subsequent vitreous hemorrhage.

If peripheral scatter laser photocoagulation is applied in eyes with large areas of nonperfusion, the incidence of neovascularization can be reduced from about 40% to 20%.

In prophylactic treatment view many eyes (60%) that would never develop neovascularization would receive peripheral scatter laser. For this reason, it is recommended that laser P.C. be applied only after neovascularization is clearly documented by F.F.A.

In confirmed neovascularization by FFA, peripheral scatter laser P.C. can reduce the likelihood of vitreous hemorrhage from 60% to 30%.

Scatter Photocoagulation

![Laser setting for Scatter photo coagulation](image)

- **Laser setting for Scatter photo coagulation**
  1. Spot size 200-500
  2. Duration 0.1 second
  3. Intensity Medium white color
  4. Placement - one burn apart at the entire Capillary non-perfusion area

**Caution-complications and side effects**

Careful consideration and discussion with the patient before initiation of treatment is required.

With proper attention to detail, complications are infrequent. Side effects of treatment include scotoma production. On average, vision will improve from 20/70 to 20/40.

It is particularly important to recognize that laser photocoagulation should never be placed over extensive intraretinal hemorrhage in the acute phase of branch vein occlusion As it can produce preretinal fibrosis.

**Wait period**

Branch Vein Occlusion Study emphasize waiting at least 3 to 6 months before considering laser therapy after the BRVO

**Surgical Treatment of BRVO**

A recent study by Kumar and associates postulated that removal of the compressive factor by sectioning the adventitial sheath (sheathotomy) might be an effective treatment for BRVO. This is still a very specialized arena.

**Conclusion**

BRVO is a common cause of visual loss in Pts. over the age of 60 yrs. through neovascularization, macular edema and macular ischemia. Adequate and timely treatment of such conditions like Hypertension, Diabetes mellitus, raised blood cholesterol and retinal inflammation are the precautions to be advised to the patient.