Current Ophthalmic Management for Retinal Vein Occlusion

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Abstract
Retinal vein occlusion is associated with ocular morbidity and blindness as a result of macular oedema, macular ischaemia and neovascular glaucoma. New treatment options have become available, particularly for the management of the associated macula oedema; however, there is no consensus as to the best therapeutic option. Ongoing trials will provide further evidence to aid decision-making as to the most cost-effective treatment option with the best visual outcome. We present a synopsis of the most recent trial data for the management of retinal vein occlusion. At present it is recommended that treatment choices are tailored to individual patient needs.

Keywords
Retinal vein occlusion, ranibizumab, dexamethasone implant, aflibercept, laser

Retinal vein occlusion (RVO) is a common retinal vascular disease second only to diabetic retinopathy, affecting the elderly population.1 Blockage of the retinal venous circulation has the potential to cause significant loss of vision. The introduction of newly approved anti-vascular endothelial growth factor (anti-VEGF) agents such as ranibizumab (Lucentis; Novartis, Switzerland) and aflibercept (Eylea; Bayer, Germany), and the slow-release dexamethasone implant (Ozurdex; Allergan, US) offer more treatment options to improve the potential for visual recovery. The remit of this article is to present a summary of the latest evidence on the ophthalmic management for RVO.

Epidemiology and Pathophysiology
A recent study on the prevalence of RVO has shown an age and sex standardised prevalence of 5.20 per 1,000 for any form of RVO. This was a large study of 68,751 individuals, ranging from 30 to 101 years of age. The prevalence of branch RVO (BRVO) was higher than central RVO (CRVO) (4.42 versus 0.80 per 1,000), increased with age and there was no difference between the sexes.2 Over 50 % of cases of RVO occur in patients older than 65 years of age.3 It has also been shown that the chance of developing an RVO in the contralateral eye within 4 years of the first occlusion to be around 7 %.4 The exact pathogenesis of RVO still remains ill-defined. It is thought to occur due to a combination of venous stasis, degenerative changes of the vessel wall and blood hypercoagulability. This is known as Virchow’s triad. In CRVO, the thrombotic event can occur in the central retinal vein, either at the lamina cribosa, posterior to the lamina cribosa or within the optic nerve. In BRVO, arterial compression of a vein as an arteriovenous crossing, in combination with pre-existing vascular endothelial damage, is thought to lead to thrombus formation. The endothelial damage may be secondary to systemic cardiovascular risk factors. In patients with arteriosclerosis, thickening and hardening of the retinal arterial walls can lead to retinal vein compression with haemostasis and resultant thrombus formation since the arteries and veins share a common adventitial sheath at crossing points.

The risk of RVO is higher in patients with hypertension, diabetes and hypercholesterolaemia.5 O’Mahoney et al.4 performed a meta-analysis that showed that the prevalence of hypertension in RVO patients was 63.6 % compared with 36.2 % in controls. Hyperlipidaemia was twice as common among patients with RVO (35.1 %) compared with controls (16.7 %). Diabetes was slightly more prevalent among patients with RVO (14.6 %) than unaffected controls (11.1 %).

Rarer associations include thrombophilia,6 oral contraceptive pill,7 optic disc vasculitis,8 multifocal choroiditis9 and systemic inflammatory disorders.10 Myeloproliferative disorders cause increased blood viscosity and systemic vasculitides, such as Behçets and polyarteritis nodosa, cause retinal vasculitis leading to RVO, especially in the younger age group.

Thrombophilia refers to the propensity to develop thrombosis due to an abnormality of the coagulation system. Antiphospholipid antibody syndrome and hyperhomocystinaemia are the two haematological disorders with the strongest evidence for association with CRVO.11-13 The Blue Mountains Eye Study11 also showed an association between CRVO and glaucoma. It was hypothesised that deformation of the lamina cribosa in glaucoma might distort the central retinal vein as it exits the eye, and thus lead to CRVO.

Occlusion of the retinal vein causes elevation of venous pressure, slowing of arterial blood flow and impedes capillary perfusion. These changes result in retinal ischaemia. Macula oedema is a common cause of vision loss in both BRVO and CRVO. The pathogenesis of macula oedema is multifactorial and is not completely understood. The factors involved include increased venous hydrostatic pressure, the presence of inflammatory cytokines...
Table 2: Summary of the Major Clinical Trials of Treatments for Macula Oedema Due to Central Retinal Vein Occlusion – 12-month Data

<table>
<thead>
<tr>
<th>Study</th>
<th>CRUISE23</th>
<th>COPERNICUS42</th>
<th>GENEVA26</th>
<th>GAULEO44</th>
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</thead>
<tbody>
<tr>
<td>Drug</td>
<td>Ranibizumab</td>
<td>Aflibercept</td>
<td>Dexamethasone</td>
<td>Aflibercept</td>
</tr>
<tr>
<td>Number of patients</td>
<td>392</td>
<td>189</td>
<td>437</td>
<td>177</td>
</tr>
<tr>
<td>Treatment arms</td>
<td>0.3 mg</td>
<td>0.5 mg Sham</td>
<td>2 mg Sham</td>
<td>0.7 mg</td>
</tr>
<tr>
<td>Mean change in visual acuity</td>
<td>13.9</td>
<td>13.9 7.3</td>
<td>-4</td>
<td>0</td>
</tr>
<tr>
<td>% patients ≥15 letters gain</td>
<td>46.2</td>
<td>47.7 16.9</td>
<td>56.1 12.3</td>
<td>18 17 12</td>
</tr>
</tbody>
</table>

PRN = pro re nata.

(e.g. prostaglandins and interleukin-6), dysregulation of endothelial tight junction proteins and increased amounts of VEGF. Vision loss can occur via the following three mechanisms:

- Serous exudation distal to the point of obstruction and excessive VEGF production, leading to excessive vascular permeability causing macula oedema.
- Retinal haemorrhages and/or retinal ischaemia may lead to retinal pigment epithelium atrophy and scarring.
- Retinal ischaemia resulting in optic disc, retinal, iris and angle neovascularisation. Retinal neovascularisation can cause vitreous haemorrhage and tractional retinal detachment. Iris neovascularisation may cause neovascular glaucoma.

Diagnosis and Assessment

Patients presenting with RVO typically complain of sudden, painless loss or distortion of vision. BRVO, however, is often asymptomatic. The presentation of RVO can consist of any combination of retinal vascular tortuosity, retinal haemorrhages, cotton wool spots, optic disc swelling and macula oedema. In BRVO, haemorrhages are largely localised to an area drained by the occluded branch retinal vein and in a hemispheric vein occlusion these are restricted superiorly or inferiorly.

The main signs of CRVO are widespread retinal haemorrhages in all four quadrants and dilated, tortuous retinal veins. A number of parameters can be used to assess the degree of ischaemia, such as degree of visual loss, presence of a relative afferent pupillary defect, extent of capillary non-perfusion on fluorescein angiography and electroretinography, showing reduced b wave amplitude, reduced b:a ratio and prolonged b wave amplitude. Ischaemia in CRVO is defined as greater than 10 disc areas of retinal capillary non-perfusion on fluorescein angiography.

Natural History and Complications

The visual outcome of CRVO depends on visual acuity (VA) at presentation. In the Central Vein Occlusion study (CVOs), 65% of patients’ eyes maintained 20/40 vision or better if acuity at the time of presentation was 20/40 or better, but only 1% achieved this level if acuity was initially 20/200 or worse.
Ischaemic BRVO patients should be monitored carefully every 3 months for 12 months, especially if the area of retinal ischaemia is >4 disc diameters. Retinal neovascularisation was found to occur in 62 % of patients with >4 disc diameter area of non-perfusion. In the presence of retinal neovascularisation, sectorial laser photoacoagulation to the ischaemic retina may be used to cause regression of retinal new vessels. At present there is anecdotal evidence for the unlicensed use of intravitreal bevacizumab (Avastin; Roche, Switzerland), to accelerate new vessel regression. It is unclear at what stage and how frequently injections should be given.

The management of hemispheric vein occlusion is similar to that described for BRVO. The risk of ruberosis in ischaemic hemi-central vein occlusion is, however, greater than that of ischaemic BRVO. The management of angle or iris neovascularisation and neovascular glaucoma is the same as for CRVO.

In an eye with no visual potential and established neovascular glaucoma, the aim is to keep the eye pain-free with the use of topical steroids and/or cycloplegics. However, if the eye has any visual potential, it is important that attempts are made to control the intraocular pressure with topical pressure-lowering agents, cyclo-ablative procedures or glaucoma filtration surgery, in order to preserve vision.

Management of Macular Oedema Secondary to Retinal Vein Occlusion

The current ophthalmic management of RVO is based on the presence or absence of ischaemia. With the availability of newly licensed drugs, such as dexamethasone 0.7 mg and ranibizumab, we are able to offer patients treatment at an earlier stage, where required, with the possibility of reducing associated complications. Tables 1–4 summarise the major clinical trials for the treatment of macula oedema.

Laser Photoacoagulation

Until recently the Central Retinal Vein Occlusion Study (CVO) and Branch Retinal Vein Occlusion (BVOS) studies have guided our understanding of the ophthalmic management of RVO. In patients with CRVO, no improvement in VA has been demonstrated from treating macular oedema with grid laser photoacoagulation in randomised controlled trials. However, in BRVO, grid laser photoacoagulation in the distribution of leaking capillaries has been shown to be beneficial, but it is recommended only after a period of 3 to 6 months following the initial event and following absorption of the majority of haemorrhage. In the BVOS study, the number of patients who gained at least two lines of VA from baseline, and maintained this for two consecutive visits, was significantly greater for those treated with laser than for controls. (p=0.00049) Laser photoacoagulation is unlikely to benefit patients in the presence of severe visual loss (<6/60 vision) and in whom symptoms have been present for more than 1 year. 22

Intravitreal Corticosteroids

Triamcinolone Acetonide

Corticosteroids have multiple anti-inflammatory effects that can reduce vascular permeability, inhibit fibrin deposition and leukocyte movement, and reduce migration of inflammatory cells. In addition they can stabilise endothelial cell tight junctions and inhibit the synthesis of VEGF, prostaglandins and other cytokines. Unfortunately, intravitreal steroids have significant ocular side effects including ocular hypertension, primary open angle glaucoma and development of cataract. They should therefore be used with caution in phakic patients and those with a history of glaucoma or ocular hypertension.

Table 3: Summary of the Major Clinical Trials of Treatments for Macula Oedema due to Branch Retinal Vein Occlusion- 6 Month Data

<table>
<thead>
<tr>
<th>Study</th>
<th>Drug</th>
<th>Number of patients</th>
<th>Mean change in visual acuity</th>
<th>% patients ≥15 letters gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>BRAVO24</td>
<td>Ranibizumab</td>
<td>397</td>
<td>16.6 18.3 7.3 7.5 7.5</td>
<td>52.2 61.1 28.8 23 21 20</td>
</tr>
<tr>
<td>GENEVA25</td>
<td>Dexamethasone</td>
<td>830</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The Standard Care vs Corticosteroid for Retinal Vein Occlusion (SCORE) study compared 1 mg and 4 mg of intravitreal Trivans (TV), a preservative-free form of triamcinolone, with standard care (grid photoacoagulation for BRVO and close observation for CRVO). Over 25 % of patients with CRVO patients treated with TV (at either dose) gained ≥15 letters compared with just 6.8 % of patients receiving sham injections. The study recommended injection of 1 mg TV for patients with macula oedema due to CRVO, with repeat injections every 4 months for persistent or recurrent oedema. In the BRVO group, TV injections were not superior to grid laser at either dose. In the 4 mg group there was a higher rate of elevated intraocular pressure and cataract compared to the 1 mg group.

Currently TV is not available for use in clinical practice instead Kenalog is available, a preserved form of triamcinolone. There is no convincing evidence that outcomes seen with TV in SCORE-CRVO would also be seen if Kenalog was used in the same way. The Score Study Investigative Group concluded that grid photoacoagulation should remain the benchmark against which other treatments are compared in clinical trials in eyes with macula oedema secondary to BRVO.

Dexamethasone Biodegradable Implant (Ozurdex, Allergan, Irvine, CA)

Dexamethasone is a potent, water-soluble corticosteroid which can be injected into the vitreous cavity in the form of an implant. Dexamethasone is three times more potent than triamcinolone on a molar basis. The implant is composed of a biodegradable copolymer of lactic acid and glycolic acid containing micronised dexamethasone. This gradually releases the total dose of dexamethasone over a period of 6 months.

At the 6-month primary endpoint, the percentage of patients who gained ≥15 letters was 23 % (0.7 mg) and 21 % (0.35 mg) in the implant groups and 20 % in the sham group. In the CRVO subgroup, the percentage of patients who gained ≥15 letters was 18 % (0.7 mg) and 17 % (0.35 mg) in the implant groups and 12 % in the sham group (see Table 1).

However, both patient populations showed some evidence of benefit at earlier timepoints. The peak effects of the drug were at 60 days. The proportion of eyes achieving at least a 15-letter improvement from baseline best-corrected VA (BCVA) was greater in treatment groups at month 1 (21 % in 0.7 mg versus 18 % in 0.35 mg group versus 8 % in the sham group; p<0.001) and month 3 (22 % in the 0.7 mg group versus 23 % in the 0.35 mg group versus 13 % in the sham group; p<0.001).
The effect was no longer statistically significant at month 6, the primary endpoint. The maximal visual benefit effect of treatment with Ozurdex was seen at 60 days, leading some to question the logic behind using it as a treatment every 6 months, and further studies with repeated injections at shorter intervals are underway.

At 12 months the mean gain in VA of patients receiving dexamethasone 0.7 mg for CRVO was 2.3 letters compared with a loss of 1 letter without any treatment. For BRVO, the mean gain in VA at 12 months was 6 letters for those treated with dexamethasone 0.7 mg and no change for those treated with sham injection (see Table 2).31

Haller and colleagues concluded that dexamethasone 0.7 mg would be most efficacious in patients who have had macula oedema for a short period of time.32 Dexamethasone 0.7 mg is now licensed for use in non-ischaemic RVO in Europe. It has also been demonstrated to be effective for the treatment of diabetic macula oedema in vitrectomised eyes.33

Intravitreal Anti-vascular Endothelial Growth factors
Ranibizumab (Lucentis, Novartis)

Ranibizumab is a humanised, affinity-matured anti-VEGF antibody fragment that blocks all isoforms of VEGF-A. VEGF released by the ischaemic retina in patients with CRVO or BRVO is thought to contribute to the development of macula oedema.

In the Efficacy and Safety of Ranibizumab Injections in Patients with Macula Oedema Secondary to CRVO (CRUISE) and Efficacy and Safety of Ranibizumab Injections in Patients with Macula Oedema Secondary to BRVO (BRAVO) randomised, controlled trials, which investigated the visual outcomes of ranibizumab in the treatment of CRVO and BRVO-related macula oedema, respectively, patients were given doses of 0.3 mg or 0.5 mg ranibizumab every month for 6 months. The effect was compared with sham injection. There were 392 patients in the CRUISE and 397 in the BRAVO studies.

A greater percentage of patients with BRVO (61.1 %) gained ≥15 letters after 6 months of treatment with monthly ranibizumab versus sham compared with CRVO patients (47.7 %). 28.8 % of BRVO sham patients gained ≥15 letters in comparison with only 16.9 % of CRVO sham patients.

The CRUISE study demonstrated that six sessions of monthly injections of 0.3 mg ranibizumab or 0.5 mg ranibizumab reduced macula oedema and provided visual benefit. The percentage of patients with centre point thickness ≤250 microns at 6 months was 75 % (0.3 mg), 76.9 % (0.5 mg) and 23.1 % (sham injection) (p<0.0001).

In the BRAVO study there was a greater reduction of macula oedema in the ranibizumab groups. Centre point thickness was reduced by 337.3 microns (0.3 mg ranibizumab) and 345.2 microns (0.5 mg ranibizumab) compared with 157.7 microns in the sham group. Ranibizumab showed significant visual benefit in both conditions and the visual gains were maintained over 12 months with additional injections (see Tables 3 and 4).31,35

Patients with BRVO had better outcomes if the time from diagnosis to treatment was less than 3 months; however, there appeared to be little difference in outcome for CRVO patients. These studies suggest that the long-term treatment outcomes with ranibizumab are excellent, but it may be difficult to completely wean patients off injections even after 2 years.30 CRVO patients are likely to require more frequent follow up and a greater number of injections to control oedema and maintain visual benefits than BRVO patients.32

Ranibizumab is also licensed in the EU for the treatment of RVO. However, a few questions remain unanswered, mainly as a result of the exclusion criteria used in the CRUISE and BRAVO studies. Patients with visual acuities better than 20/40 or those with an afferent pupillary defect were excluded, meaning that the efficacy of ranibizumab in treating patients with VA better than 20/40, or those with severely reduced VA and advanced macula oedema, is unclear.

Bevacizumab (Avastin, Roche)
The pan-VEGF blocker bevacizumab is licensed for intraocular use. The Pan American Collaborative Retina Study Group has evaluated the effects of bevacizumab for both CRVO and BRVO in large retrospective comparative multi-centre studies. For CRVO, 56.8 % of eyes treated with 1.25 mg of bevacizumab gained ≥3 lines of VA, while 57.1 % of eyes treated with 2.5 mg of bevacizumab achieved similar gains.34

Combination treatments of dexamethasone 0.7 mg and bevacizumab have shown promising results. A recent study34 evaluated the efficacy and safety of a dexamethasone implant 0.7 mg alone or in combination with bevacizumab over 12 months. Sixty-four eyes were prospectively investigated. Group 1 were treated with Ozurdex and group 2 with three consecutive bevacizumab injections followed by Ozurdex. Recurrences were treated with Ozurdex only. In group 1, BCVA improved to a greater extent in the BRVO patients compared with the CRVO patients. An opposite outcome was observed in group 2 with a greater improvement in BCVA with CRVO patients compared to the BRVO patients. Recurrence after the first Ozurdex injection occurred after 3.8 months (CRVO) and 3.5 months (BRVO) in group 1 versus 3.2 and 3.7 months in group 2. Raised intraocular pressure (>21 mmHg) was detected in approximately 40 % and cataract progression requiring surgery occurred in 50 % of eyes after three Ozurdex injections. One year after the initiation of treatment, a significant difference in number of letters gained was seen only for patients with CRVO, favouring patients who were treated with bevacizumab first.42

### Table 4: Summary of the Major Clinical Trials of Treatments for Macula Oedema Due to Branch Retinal Vein Occlusion – 12-month Data

<table>
<thead>
<tr>
<th>Study</th>
<th>Drug</th>
<th>BRAVO40</th>
<th>SCORE41</th>
<th>GENEVA43</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>397</td>
<td>411</td>
<td>437</td>
<td></td>
</tr>
<tr>
<td>Treatment arms</td>
<td>0.3 mg 0.5 mg Sham/0.5 mg</td>
<td>1 mg 4 mg Laser</td>
<td>0.7 mg Sham/0.7 mg</td>
<td></td>
</tr>
<tr>
<td>Mean change in visual acuity</td>
<td>16.4 18.3 12.1</td>
<td>5.7 4 4.2 6 6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>% patients ≥15 letters gain</td>
<td>56 60.3 43.9</td>
<td>26 27 29 0 0</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
A prospective interventional case series of 41 of 34 eyes of 33 patients treated with initial injection of bevacizumab followed by a 0.7 mg dexamethasone implant has demonstrated similar findings. VA improved from 11 letters to a maximum of 25 letters during the study period. These studies suggest that the combination of treatments might provide a synergistic action that increases VA and prolongs the time between injections compared with the single treatment with either medication.

Currently the Comparison of Intravitreal Dexamethasone Implant and Ranibizumab for Macular Oedema in Branch Retinal Vein Occlusion (COMO) study is underway. It is a randomised, 12-month study, comparing the safety and efficacy of dexamethasone 0.7 mg versus ranibizumab in the management of macular oedema in BRVO. The results of this head-to-head study will provide guidance on how to direct further treatment.

**Afiblercept (Eylea, Bayer)**

Intravitreal afiblercept injection, also known as VEGF-Trap Eye, is a fusion molecule which has anti-VEGF effects. It is a 115 kDa decoy receptor fusion protein, composed of the second domain of human VEGF receptor 1 and the third domain of VEGF receptor 2 fused to the Fc domain IgG1. The binding affinity of afiblercept for VEGF is greater than that of either bevacizumab or ranibizumab, thereby offering a theoretically longer interval between doses. It is licensed in the EU for use in patients with CRVO.

Afiblercept in under evaluation for the treatment of CRVO in two phase III studies: Controlled Phase III evaluation of Repeated Intravitreal administration of VEGF Trap-Eye in Central Retinal Vein Occlusion (COPERNICUS) and VEGF Trap-Eye: Investigation of efficacy and safety in CRVO (GALILEO). In the COPERNICUS study, patients were randomised to receive one injection per month for 6 months injections of either 2 mg intravitreal afiblercept or sham. This study involved 189 patients. The monthly 2 mg dose showed significant visual benefit in patients with CRVO at 6 months, with 56.1% of patients achieving ≥15 letters compared with 12.3% of patients receiving sham injections. There was a large mean VA gain of 17.3 letters in those receiving treatment compared with a loss of four letters in those without treatment.

From week 24 to 52, all patients received 2 mg intravitreal afiblercept as needed. At week 52, 55.3% of the treatment group had gained ≥15 letters compared with 30.1% in the sham group. There was a higher mean VA of 16.2 letters compared with 3.8 letters in the sham group (p<0.001).

The authors concluded that at 24 weeks, monthly intravitreal injection of 2 mg afiblercept improved VA and central retinal thickness, prevented neovascularisation and was associated with a low rate of adverse events related to treatment.

Similar results have been shown in the GALILEO study. This was a double-masked study in which 177 patients either received 2 mg intravitreal afiblercept or sham every 4 weeks until week 24. During the first 24 weeks of GALILEO, monthly afiblercept treatment resulted in rapid and sustained gains in BCVA. Disappointingly, this improvement in VA declined between weeks 52 and 76. Overall, the results of these trials provide a promising treatment for patients with CRVO.

**Combination Treatments**

In addition to the combined treatment of dexamethasone 0.7 mg and bevacizumab mentioned earlier, bevacizumab has also been studied in combination with laser and triamcinolone acetonide.

A small, prospective study of 18 eyes with macular oedema due to BRVO, in which eyes were randomised to receive treatment with either bevacizumab alone or in combination with macula grid laser, has shown that combining laser treatment with intravitreal bevacizumab injections results in a lower number of re-injections than intravitreal bevacizumab treatment alone. A randomised, prospective study of bevacizumab injection alone versus combined injection of bevacizumab and triamcinolone acetonide (2 mg/0.05 ml) in patients with CRVO showed no significant differences between the groups.

**Surgical Treatments**

Other treatments which have been considered include laser-induced chorioretinal anastomosis, radial optic neurotomy with pars plana vitrectomy and thrombolytic therapies. Laser-induced chorioretinal anastomosis has been shown to increase the risk of choroidal neovascularisation and requires a high-powered laser, which is no longer commercially available. Experimental trials of radial optic neurotomy with pars plana vitrectomy and thrombolytic therapies are underway, but these treatments are not currently recommended.

**Conclusion**

Recent trials have provided us with multiple methods of treating macular oedema in RVO. It is difficult to compare the treatments as all the various trials and studies have differences in their design and methodology.

With our current knowledge of the treatment options, corticosteroids and anti-VEGF agents appear to be at the forefront of management. However, a higher incidence of intraocular pressure rise and cataract formation makes corticosteroids the less desirable option. Anti-VEGFs have a better ocular side-effect profile, but there is no defined treatment end-point and more frequent injections are required. There is also the theoretical risk of an increased rate of arterial thromboembolic events, such as myocardial infarction or stroke with anti-VEGF treatment. This poses some concerns as many RVO patients may already have underlying hypertension, hyperlipidaemia and/or diabetes.

Studies are currently underway which will provide additional guidance as to how our treatment regimens can be modified to further enhance visual outcomes and simultaneously reduce the burden of frequent follow up and requirement for injections. Certainly the results of some of the head-to-head studies will shed further light as to which direction to take in terms of anti-VEGF and dexamethasone 0.7 mg injections.

At present we believe that the best strategy is to tailor the treatment according to the individual patient, the aim being to prevent irreversible damage caused by chronic macular oedema. For younger patients without significant systemic risk factors, a history of ocular hypertension or glaucoma, anti-VEGF might be the most appropriate first-line treatment. Pseudophakic patients with a previous history of stroke and reluctance or inability to attend the eye clinic monthly might benefit from initial treatment with a dexamethasone 0.7 mg implant.
Retina