Vein Occlusions

The underlying condition for the majority of vein occlusions is hypertension. If you think about how the vasculature works on the retina, arteries cross over veins. As we get hardening of the arteries, eventually that puts pressure on the vein underneath, we get nicking and a thrombus that forms, occluding off that blood vessel. Vein occlusion can also be associated with coronary artery disease, diabetes, and peripheral vascular disease. It is usually seen in elderly patients (60-70 YO), with a slight predilection for males and hyperopes. Vein occlusion is the second most common vascular disease after diabetic retinopathy.

What we see with a branch retinal vein occlusion (BRVO) is a sudden, painless loss of vision in a quadrant area, with an, in the case of Figure 10, is an inferior visual field defect. We will get tortuosity of the retinal blood vessels, particularly the veins, with superficial hemorrhages and cotton wool spots (CWS). The occlusion typically happens at an A/V crossing.

Table 1

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<th>Causes of Visual Loss in a BRVO</th>
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With BRVO’s, most patients end up retaining relatively good vision. More than 50% of your patients are going to have 20/40 vision or better. Vision loss typically happens from things such as persistent macular edema, development of a vitreous heme or other problems going on (Table 4), but the majority of patients end up doing very well.

Question: What would you recommend to a patient with a non-ischemic BRVO who has decreased vision secondary to macular edema?

1. Monitor as it will resolve on its own
2. Monitor for 5-6 months then macular grid laser if not resolved
3. Macular grid laser immediately
4. Monitor for 5-6 months then initiate anti-VEGF if not resolved
5. Initiate anti-VEGF treatment immediately

(Scroll to next page for answer)
The majority of polled doctors went with option #5, which is initiate anti-VEGF immediately. I think there is some debate on this one. There was a study that came out which is called the BRAVO study. The BRAVO study looked at the use of anti-VEGF for the treatment of macular edema secondary to BRVO’s. From the BRAVO study, we know that macular grid will improve a patient’s vision, so we typically waited 5-6 months, and at that point if there was not much resolution we would go ahead and do the macular grid lasers. With that, we could typically get 2-3 lines of improvement in vision. This study did a sham injection for the first 5 months, then in the 6th month, it gave an injection of anti-VEGF vs two concentrations of anti-VEGF from day 1. The results showed that the patients who aren’t treated (white line, Fig 11 Bottom) DO get better. In month #6, when they were given the anti-VEGF, there is another little peak in their VA’s that goes above. That’s compared to the patients who had anti-VEGF from day 1, and there was no difference in success between the two different
concentrations of anti-VEGF, but you can see that overall there was better vision if anti-VEGF was given from Day 1.

With a central retinal vein occlusion (CRVO), we have a different story. The patient’s vision is going to be significantly reduced – usually 20/200 to finger counting range. This is happening closer down to the lamina area, just prior to the lamina cribrosa. We have dilated blood vessels, lots of blood in the retina, edema, and potentially cotton wool spots.

Thinking about patients who present with a CRVO, what we get concerned about is patients who have ischemic ones. Approximately 20% of our patients who have CRVO’s are going to be ischemic. Half of those patients are going to run into other complications. Complications include things such as neovascular glaucoma, persistent macular edema, and macular ischemia. Patients don’t tend to get better on their own in this case.

CRVO’s can be ischemic versus non-ischemic. Things that you can look for while the patient is in your chair is looking for things such as the classic definition, which is 10 disc diameters (DD’s) of non-perfusion using fluorescein angiography, but not all of us have fluorescein angiographies in our offices. What you may want to do instead is if the VA is worse (CF vision) it is likely ischemic. If it’s 20/200, probably non-ischemic. A larger APD is more likely to be ischemic. If there is lots of CWS, CWS are an indicator of ischemia, and can be a solid indication that the patient may have an ischemic CRVO.

It is estimated that about 20% of CRVO patients will have ischemic CRVO’s, half of those are going to run into the development of neovascularization, particularly of the iris, which can result in the development of neovascular glaucoma. Neovascular glaucoma is a particularly scary condition because in patients who develop neovascular glaucoma, that eye is pretty much a goner eye because there is nothing we can do to help treat those patients.

Question: What would you recommend to a patient with a non-ischemic CRVO who has decreased vision secondary to macular edema?

1. Monitor as it will resolve on its own
2. Monitor for 5-6 months then macular grid laser if not resolved
3. Macular grid laser immediately
4. Monitor for 5-6 months then initiate anti-VEGF if not resolved
5. Initiate anti-VEGF treatment immediately

(Scroll down to next page for answer)
The majority of polled doctors went with #5, and in my mind that is the most correct answer.

Along with the BRAVO study, they did the CRUISE study, and in the CRUISE study they again did the sham injection for the 1st 5 months, and then in month 6 started anti-VEGF, versus two concentrations of anti-VEGF. The results were very different.

In this case, we can see with the white line (Fig 13 Bottom), patients don’t get better vision, and that is classic for CRVO’s. Then at month number 6, we start giving the injection and we can see a significant increase in the patient’s vision. This is in contrast to the patients who received anti-VEGF from day 1. Now we are talking about a difference of 3-4 lines in vision. So there is really no debate in this case: what we need to do with patients who have a CRVO is they need to be sent for anti-VEGF to get the best vision that they can. Ultimately that means that they are not going to develop the neovascular glaucoma, because we are already giving them the anti-VEGF to prevent that from happening. But really you are sending
them because you want to make sure that they are getting their best central vision, and that is gotten when treated from day 1.

**Artery Occlusions**

The last condition we are going to look at today are artery occlusions. Artery occlusions typically come from emboli that usually come from the carotid area. Ultimately with patients who have artery occlusions, we get very concerned about where the emboli is coming from, and where the next emboli is going. For me, the real emergency in this case is not necessarily what is going on with the eye, but more where the next emboli is going to go, which can end up in the brain and result in a stroke.

With branch retinal artery occlusions (BRAO’s), that occlusion is happening at a bifurcation in the artery. (Fig 14, Top) With central retinal artery occlusions (CRAO’s), it is happening closer to the lamina, which results in this cherry-red macula (Fig 14, Bottom). Remember, this cherry-red macula only lasts for the first couple of days. Eventually the retina is going to re-perfuse and look normal. There will be an APD and VA’s will be count fingers to light perception in this case.

Treatment of CRAO is controversial due to poor prognosis and questionable benefit. Ultimately, if you have a patient that presents within the first 24 hours, you are going to want to recommend digital massage, acetazolamide, topical ocular hypertensive drops such as your beta blockers, ultimately we are recommending these for our patients, but whatever vision they are presenting with is going to be their vision going forward.

Why do you still want to recommend these for your patients? Eventually, your patients will google their condition, and then they will wonder why their doctor didn’t recommend any of these things. Really, no matter what you do, there is not going to be much success – we have roughly 91 minutes to intervene in a patient with a CRAO before the retina is effectively toast. Most patients aren’t going to even notice that change within that short of a period of time. Thus, recommend these things for your patients, but ultimately, probably not much is going to help.

**Quickie**

**Question:** Identify these lesions. (Still visible when we put our red-free filter on.)

1. Choroidal nevus
2. Choroidal melanoma
3. CHRPE
4. Toxoplasmosis

(Scroll down to next page for answer)
The correct answer is 3. This is a CHRPE, or Congenital Hypertrophy of the RPE. A choroidal nevus is something that I would think would be the primary differential for this, but I would remember that all nevi are in the choroid. With the fact that you put the red-free filter on and the lesion is still visible tells you that the lesion is in the RPE. If the lesion were to disappear, then you would know it’s in the choroid. If the lesion is present with the red-free filter, and it’s in the choroid, you have two choices: it’s either hyperplasia, or hypertrophy.

Figure 15 shows the difference between a nevus and CHRPE. CHRPE’s are usually single, very isolated lesions that are defined. You may get lacunae that develop in the middle, which are just RPE window defects. Compare that to your nevus (Fig 15 Right), which is in the choroid.

What you want to do for your nevi is your ABCD’s. You want to look for asymmetry – split the lesion in half and one side should look like the others. B is for borders – we want distinct borders. C is color – we want a nice uniform grey appearance. D is actually three things: disc diameters (or size), depth which shows any elevation, and the final one is duration – how long has the nevi been there? Most nevi are present in patients by the time they are about 10 years of age. They may increase in color, but we don’t tend to get new ones after the age of 40.

31% of choroidal nevi show a slight enlargement over time, so nevi will actually get bigger, but very, very slowly. In contrast to your melanomas, which are going to grow very quickly. The prevalence of choroidal nevi us about 5 to 8% of the Caucasian population in the US. It is assumed that choroidal melanomas arise from preexisting nevi, so it’s really important that we photo document choroidal nevi so that the patient knows it’s there. These need to be monitored.

The risk for metastasis ranges from 16 to 53%, at 5 years of follow-up, depending on the size of the tumor at the time of diagnosis. 80 to 90% of choroidal melanomas metastasize to the liver. This is hugely concerning for patients because the 5 year survival rate for patients who have metastasis to the liver is really nonexistent.
Thus, we really need to make sure that we are monitoring these nevi in patients. If there is the development of a melanoma, it needs to be treated immediately because if there is a metastasis, it tends to go to the liver, and patients don’t survive.

Things that we often want to look for in choroidal nevi is the development of lipofuscin, or that orange pigment. In this case, fundus auto fluorescence is probably the best instrument to help us determine whether or not that lipofuscin is present. It’s also been found that aggressive surveillance of patients who have melanomas has actually resulted in secondary cancers developing, so there is a little bit of a debate as to how aggressively we need to monitor survivors after their treatment.

I believe I am out of my time for this lecture. Thank you very much for your attention, and I hope you took away something to apply in your practice for the benefit of your patients.

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