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Illuminating Patient Selection for PBM in AMD: A Case-Based Discussion

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Chapter 1: Welcome

Dr. Do:
Welcome. It's great to be in New Orleans together. I'm Dr. Diana Do, and I'm joined by 2 friends and colleagues, Dr. David Boyer and Dr. Nora Lad. Welcome, David and Nora.

Dr. Boyer:
Thank you.

Dr. Lad:
Lovely to be here.

Dr. Do:
We have a very exciting topic today, photobiomodulation, which is the newest technology available for patients with dry age-related macular degeneration. And we're going to talk about how you can use it in clinical practice. What's the clinical evidence, and what type of patients are ideal for this non-invasive therapy?

Chapter 2: Dry AMD Care in 2026

Dr. Lad:
So here's where we are currently. Current guidelines for reducing the risk of AMD progression of patients in clinic. So the first thing we tell them: we advise them to quit smoking. So current and former smoking are strong risk factors for AMD, the strongest environmental risk factor. So current smokers should be counseled on quitting, and that's important. I always document it in my notes. Then we should pay attention to modifiable risk factors, such as high blood pressure, excess weight, and maintaining a healthy lifestyle can improve long-term health. And that's just generally good for the eyes, but specifically for AMD.

The AREDS supplement: so supplementation should be considered in patients with intermediate and more advanced forms of AMD,

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such as GA and neovascular AMD. Mediterranean diet: This one's my favorite. So a healthy diet consisting— they're going to ask, “What is a Mediterranean diet, Dr. Lad?” So I tell them it's fruits, vegetables, olive oil—especially so—healthy fats, whole grain, nuts, beans, seafood. So the ratio between the healthy and less healthy fats should be as high as possible. That was one of the factors that was particularly researched in the 10-year studies that NEI conducted. Limiting amounts of red meat and butter—as tasty as they are—as much as we can, sugar and processed foods, and this all reduces the risk of AMD longitudinally.

And interestingly, and not related to this, but somewhat related, NIH did a 10-year study of prevention of neurocognitive declines or dementia, and they had the same results. So I think if there's one thing we can counsel our AMD patients to do is use a Mediterranean diet.

The other 2 things that were helpful in it, anecdotally, was an intake of whole fruit, so fruits with a peel, I'm thinking berries and others. And the third, and my favorite, was moderate alcohol intake. I have teenage boys, so they all love that, but a little bit. And there's guidelines for that, so just a bit, if they already drink some wine or such.

Regular monitoring is very important for our AMD patients, so regular dilated eye exams and self-monitoring at home. This allows for early detection and early treatment. And timely treatment of neovascular AMD is particularly important within a week or 2, I would say most of us would advise, and that can be done with Amsler grids or the ForeseeHome device.

So last but not least, and that's why we're all here tonight, photobiomodulation. It decreases the onset of new geographic atrophy. You'll see the data tonight and is approved for the treatment of patients with non-neovascular AMD.

So a few words on the AREDS supplement and the evolution of these vitamins over time. So the original formulation was shown to decrease progression to advance AMD by about 20%—a 20% reduction. However, the studies at the time showed that beta-carotene increases the risk of lung cancer in smokers, in current or former smokers, and increased mortality.

So as a result, the AREDS2 formulation was developed. Instead of the beta-carotene, the formulation contained lutein and zeaxanthin, which are protective pigments in the macula, and that provided a 10% reduction in progression to late AMD as compared to the original formulation. And the addition of omega-3 fatty acids as present in fatty fish had no benefit or DHA. You can see there, the hazard ratio that was researched for this, and the prevention of progression to neovascular AMD accomplished by AREDS2 vitamins.

Well, your patients might ask you, and they often do, what's going on with the AREDS3 formulation that's now on the shelves in the pharmacies. So this is the AREDS2 plus the B vitamin complex. And the addition of the B vitamins is based on prior research. They've been implicated in retinal health and AMD progression, so they've been added. The trial hasn't been done yet, but it is in the planning stages.

Okay, so a little bit on patient education. So there's some really helpful materials that AAO provides. They're videos, short clinical videos to reinforce the overviews on disease and what the patients should expect when they receive treatment. There's good animations to visualize the disease progression with graphics over time and the treatment mechanics, pathobiology of disease, and there's some really nice handouts that AAO puts out.

But more importantly, and you can scan that QR code while you have the opportunity—I'd recommend you do that—that is our monograph that we did on photobiomodulation, and that provides really helpful information from the experts on the biology of photobiomodulation, all the research studies that have been done in various diseases, and lately in dry AMD as well.

Chapter 3: Mechanisms Made Practical

Dr. Lad:
So I will keep going forward. The next section is on mechanisms of age-related macular degeneration made practical as they relate to photobiomodulation. So this is a busy slide, but it's very well done because I think it illustrates the progression of disease across the stages, and this is what we're trying to prevent with treatment with supplementation, Mediterranean diet, and photobiomodulation.

So all our patients and all of us start with a normal case scenario, which is on the left-hand side, and so basically what happens, the

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photoreceptors have this material that has to be discarded through Bruch's membrane, and then also the photoreceptors and RPE cells obtain the nutrients and oxygen from choriocapillaris and choroid. With time, with age, and other risk factors from no AMD, we're having a problem with transport of these nutrients and oxygens from the vasculature into the retina. And there's also Bruch's membrane changes. That is the barrier, if you will, between the retina and the blood, the blood-retinal barrier. And Bruch's membrane starts to become clogged with age, kind of like you know the hair in the shower filter, and that's no good because over time, those materials that should have been discarded become accumulated.

At the same time, over time, within age and early to intermediate macular degeneration, the photoreceptors become more impaired due to inflammation. They go through an energetic crisis, they undergo oxidative stress, and they have more of these materials that should be discarded. However, instead of doing so, it becomes deposited as drusen. And within drusen and under drusen, there's complement activation that is not helpful in the disease process. So over time, these drusen accumulate in large, from small to intermediate to large, and over time, we have the late dry AMD stage that leads to RPE and photoreceptor loss, and that defines geographic atrophy. It's very debilitating when it involves the foveal center, especially. About 15% of patients convert to the wet form of late AMD with vessels that come from the underside of the retinas. What I explain to the patients, they seem to understand that. So choroidal neovascular membranes that occur and lead to leakage and loss of vision acutely, and that's why we want to institute treatment as soon as possible.

But AMD as a whole, the whole spectrum of disease turns out to be caused by a vicious cycle, and that's a vicious cycle of mitochondrial dysfunction. The mitochondria is the energy-producing organelles of the cells. It's the battery that powers the cell, if you will. So initially, we have those early insults. Aging, which none of us can help. The alternative is no good—I was told by a dermatologist. Think about that. Genetics, smoking, and over time, it leads to impaired mitochondrial dynamics. The mitochondria became fragmented, and they fail to have healthy mitophagy or the normal process that mitochondria uses to self-clean and get rid of debris. So over time, the cell undergoes a bioenergetic crisis with oxidative stress, and there's less ATP or energy being produced and increased in reactive oxygen species. Over time, there's mitochondrial DNA damage and DAMP release with inflammation and complement activation. And then again, due to this, we have RPE and photoreceptor dysfunction and cell death, and more drusen production, and then geographic atrophy, which is then further exacerbated by the initial insults, and then the cycle goes on and on and on, and the disease keeps progressing.

So what photobiomodulation does, it breaks this vicious cycle, and you will find out more about photobiomodulation. Again, this is our monograph, so please scan this QR code as well. And this has a lot of important information and all the publications on that. So what it does, this is near-infrared and far-red irradiation that restores mitochondrial function. It increases the production of helpful cytoprotective factors. It reduces that inflammation in the cell and prevents cellular death. So when we use the irradiation, we have increased cell survival. It creates this stable environment by activating transcription factors, altering gene transcription and protein synthesis over time. So instead of that unhelpful cycle, we have a positive cycle that is stimulated by irradiation with far-red and near-infrared.

Complement has been shown to play an important role in the progression of age-related macular degeneration, and so complement inhibition has been attempted, and successfully so, in decreasing the progression of geographic atrophy. So what complement inhibition does, it decreases the formation of the membrane attack complex in the cascade, in turn protecting the RPE-photoreceptor complex.

And you can see this complex diagram that illustrates the complement cascade with the 3 pathways: classical, lectin, and alternative. And most of the targets that have been used in clinical trials and recently approved work on the alternative pathways, and you can see them listed.

In orange, we have the 2 approved therapies: the pegcetacoplan, which inhibits C3, and avacincaptad pegol inhibited C5. The other agents in red are currently in clinical trials. There's one that addresses the classical pathway, that's ANX007, and there's a number of others on the right that inhibit the alternative pathway.

So the whole goal with complement inhibition is to decrease the recruitment of inflammatory cells. These are macrophages, microglia, perhaps even T and B cells, and promoting the survival of VEGF-A-producing cells, which drive the cases of new-onset CNV. That's in a way good and bad because you can protect some of the cells that would die due to GA, but you can increase the risk of choroidal neovascular formation. So we counsel the patients when we use these approved 2 therapies in orange, that they're at higher risk for developing wet AMD, and that treatments can be started for that.

Lastly, there's another technology called transpalpebral microcurrent device that is placed on closed eyelids of the patients and on the

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back of the neck, and this is a low-level alternating pulse microcurrent to the upper eyelid that stimulates the retina. It's thought to restore the transepithelial potential of the aging RPE, because just like aging photoreceptors, the retinal pigment epithelium also age, and they don't have a normal potential, and so their cellular morphology and function is impaired. So this is the design of the study, and the microcurrent device demonstrated both functional and anatomic improvements at month 3 and 6 in the treated patients.

What that's aimed to do is to restore the electrical balance of the retina, just like photobiomodulation does, but through a different mechanism. So vision is actually an electrical process. It's basic electrophysiology. We rely on electricity in the back of the eye, which I think is really cool. But what happens over time due to age and oxidative stress, the RPE falters and you don't have this normal TEP potential anymore, so we jolt it, kind of like doing CPR on a patient that needs the stimulation to come back to life. So we bring back the RPE through this microcurrent delivered to the retina, and the TEP then is normalized, and also because the photoreceptors are better supported by the RPE, the function improves.

So we've seen the regular treatments improve best-corrected visual acuity over 12 months, and this is a study called i-SIGHT2, and it was done in intermediate to advanced non-exudative AMD, and that's ongoing. So excited to see results on those.

Dr. Do:
So we're just going to briefly discuss right now, and then David's going to present the data. But which eyes would be good candidates for photobiomodulation? Nora mentioned that the aging changes that occur in dry AMD contribute to mitochondrial dysfunction, and that photobiomodulation may be helpful in restoring the normal energy-producing organelles of the eye.

Chapter 4: Reading the PBM Evidence

Dr. Do:
Now we're going to have Dr. David Boyer take a deeper dive into the clinical evidence and show us why the FDA authorized PBM.

Dr. Boyer:
Thanks, Diana. And Dr. Lad did an outstanding job of explaining to you the thought process behind. And I will go through the results of the LIGHTSITE III pivotal trial. It was a 24-month trial. Basically, patients received a total of 9 treatments, usually 3 a week for 3 weeks, and then this is repeated every 4 months. So in the course of a year, they get approximately 3 cycles of treatment. This was carried on for a total of 24 months.

The patients were >50 years of age, which you'd expect. They had vision between 50 and 75 letters, which is 20/32 to 20/100, and dry AMD, which was defined in the study as having 1 druse larger than 125 µm, or 3 drusen between 63 and 150 µm. The GA was not included. You could not have GA that involved the center 1-mm diameter of the ETDRS. So this was not set up to look at patients with geographic atrophy to see if there was an improvement.

The exclusion criteria were center-involving GA within 1-mm diameter of the ETDRS subfield, they couldn't have neovascular AMD, no history of epilepsy because it is a light therapy that perhaps could have precipitated a problem, or any other significant retinal diseases.

The baseline characteristics: there was approximately 148 eyes, 100 eyes which received active treatment, 48 eyes which received treatment at a much-reduced level. So it's important to understand that in the course of doing studies, you really want to continue masking. You couldn't have someone sit in front of a machine that was supposedly giving light therapy and have no light coming, so the light therapy in the sham group was reduced tremendously, just to make sure that they still felt they were receiving treatment. It was the usual 99% Caucasian, 68% women, average age about 75, and these were eyes that had AREDS3. These were eyes that actually were not small amounts of change, but these were eyes that actually had fair amounts of drusen or signs of atrophy elsewhere.

The primary endpoint was the best-corrected visual acuity, and the efficacy endpoint was met. You can see that the treatment after 2 series of treatments, by the third treatment series, the visions had improved over 5 letters and continued at that level all the way through to the end of the study.

You can see also that the sham group had some improvement, and we can argue what caused that. But I've done a lot of clinical trials, and many times patients learn how to take the test better, and they do very well for a short period of time. But then, over a long period of time, you can see that the vision actually decreased. You could also argue that even low levels of the light therapy that was

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administered was enough to cause a good reaction in the mitochondria, as was pointed out.

Let's see what happened during the trial. You can see at month 13, 21, and 24 that the patients who received active PBM treatment had a much larger gain of 5 letters, 10 letters, or 15 letters all the way throughout the study. And you always are concerned when you do a study, was there a certain amount of patients who did phenomenally well and they carried the entire study, so we should look at the patients who lost vision. So if you're treating with an active treatment and you're hoping that the vision improves, did it make a difference? Did it reduce the number of patients who actually physically lost vision? And you can see here at month 13, 21, and 24, there was an increased risk of losing at least 5 letters of vision in the sham-treated group as compared to the PBM-treated group.

I think this, to me, was the most important slide, and it helps me identify who I feel is a real good candidate for treatment. Overall, the entire group did improve overall >5 letters of visual acuity. However, if you really look at the patients who had vision between 20/40 Snellen and 20/63, the amount of improvement was very, very large compared to the sham. If you look at the group of patients that had 20/40 or better vision, 20/32 in this case, there was an improvement overall, but the improvement wasn't as great.

Here is a Kaplan–Meier Cox proportional hazard model, and you can see that it reduced the incidence of best-corrected visual loss in the treated group as compared to the untreated group all the way through the study.

This is probably a slide that most of us in clinical practice don't really understand very much. We don't do it, but this is a quality of life improvement. This is asking the patients basically: Did this improve your quality of life? And I think we're all sitting here and we look at visions and we look at the OCTs and we look at how many drusen they have, but this is a patient basically telling you that, “Hey, my quality of life was improved or remained the same” as compared to the group that was in sham that continued to lose vision, especially if you look at the composite score, there was an improvement compared to a detriment.

Macular drusen volume was evaluated, and certainly the patients who received active treatment had a reduction in drusen volume. I will tell you this is a small study. The numbers were very small, and it was not set to look at basically if drusen volume was going to decrease. The important thing about the reduction in drusen volume that I found when I reviewed some of the cases is that drusen volume did decrease, and those of us who treat retinal diseases, we know that drusen will come and go. You can show patients, “Whoa, your drusen are getting better,” and then they come back, and they have some more drusen in a different area. But in those cases, when the drusen come and go, many times the drusen leave you with geographic atrophy. Here, when the drusen went away, they really didn't leave us with geographic atrophy, which I think is an important point. And this shows just in this one case the reduction in volume.

Here's the reduction of incident geographic atrophy. Again, a post hoc Cox proportional hazard model showing a reduction in the incidence of GA formation. And here is a conversion. Patients’ incidence of new GA in patients who only had incomplete retinal outer atrophy, and conversion to complete retinal outer atrophy. And you can see in the sham group this was extremely high. So again, this may be something that we can look at prospectively in a randomized trial to see if we're able to actually reduce drusen geographic atrophy formation.

Every study that we do, the most important thing that we look at is safety, and this was a very safe drug—or a safe treatment, not a drug. But neovascular AMD did stand out, and it was at 3.8% in the sham group versus 7.5% in the PBM-treated group. And I think those of you who have seen the complement studies will realize that this is a lower incidence than we were seeing at 24 months in the complement study. So this may be part of the natural history of the disease. We may not be able to stop it.

And, also, I think that we're going to be looking very carefully in the future at these patients who may have occult CNV, either doing OCTA or looking for different signs that may indicate that they may go on to developing a problem.

EUROLIGHT is a registry. We've only had this since November of what, 24, I guess it is, that the approval from allowing us to be able to treat patients based upon not only the efficacy but the excellent safety. The EUROLIGHT registry in Europe, this has been going on for a long time. They've had a CE Mark for a considerable period of time. And they treat patients who have neovascular AMD. They treat patients for very different conditions, and they have excellent results. And the purpose of the EUROLIGHT registry is to gather enough patients to be able to see the experience because they're well ahead of us, and they can give us 2- and 3- and 4-year follow-up. There are some patients that have been treated for central serous in Europe with good results, and these are patients who have not responded to observation over a long period of time.

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Chapter 5: PBM in Practice

Dr. Boyer:
So I'm going to go on PBM in practice. This is important. This is a light delivery system. This is not lasers. So these are diode-emitting lights that are at certain wavelengths. And as Dr. Lad intimated, these lights actually will improve the mitochondrial function. They are vasodilation, reducing or liberating nitric oxide, and also are anti-inflammatory. And this combination seems to be what's going on here. This is not red-light therapy alone. You're not going to get the same effect from a red light; 590, 660, and 850 all are working together at different parts of the retinal pigment epithelium.

As I indicated, they get 3 treatments a week for 3 weeks, and then if they need to, and because of hurricanes that come in, and they can't come in—in California, we don't have that, but if it's too hot, they won't come in—so you can extend it out to 4 or 5 weeks and spread it out over a period of time. But every 4 months, the treatment is repeated, and you do not need to dilate the patient so they can drive safely home.

In practice, who can administer the PBM is state-dependent. Every state has their own way of interpreting it. Some require a nurse, some require a doctor, and in California, it can be a technician.

Clinic integration: How is PBM integrated in the clinic? I think, Diana, you want to?

Dr. Do:
Yeah, so for me, we have separate appointments because when a patient comes in for PBM only, they do not have to have their vision checked. They do not require dilation, no topical anesthesia, so we set separate appointments just for PBM. And then after they finished a cycle, before they go on to their second cycle, I'll see them in the clinic, check their vision, do OCT fundus photos.

How about you, Nora? How do you integrate it?

Dr. Lad:
Well, we have more experience in clinical trials now. So, but yeah, I agree with you. I think the flow is very important to consider.

Dr. Boyer:
So information sharing. What information? The patient comes in to see you've been involved in the clinical trials; you've done a lot of clinical trials, you have a lot of things that you can offer the patient, what's your discussion like? I mean, what do you tell them basically?

Dr. Lad:
I give them the cliff notes of the clinical trial results, and then I go into potential side effects. And I say they require monitoring still, standard of care monitoring with me every 6 months, ideally, and they still should do home monitoring and all the AREDS vitamins and healthy diet and all the other, but in parallel to their treatments. And should wet AMD occur, then they'll get treated for that concomitantly, and we have to decide whether to keep treating the other eye that hasn't converted, if they're comfortable, or if not, we take a break and then resume treatment when the eye is quiet.

Dr. Do:
David, we had some questions from our audience. A doctor asked: What if a patient misses a PBM cycle or is lost to follow-up, can we just pick up from where we left off, or what do you tell them?

Dr. Boyer:
I think it's reasonable. If somebody gets sick, is unable to come in, and they go 5 months, I would just start all over again and just start the treatment and start the 4-month cycle again. So I don't think it's a problem. LIGHTSITE III had a 20-month period in which no treatment was administered for a variety of reasons. But then, when the treatment was re-administered, there had been a decrease in vision in the group of patients that were being treated. But once you started retreatment, the visions popped up again, and they regained the vision they had. So I think that just starting it all over again is fine.

Dr. Do:

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Another good question from our audience is: During the LIGHTSITE III study, could patients have undergone cataract surgery? And if so, would that have caused the vision improvement?

Dr. Boyer:
Well, in the study, nobody was undergoing cataract surgery. Part of when you do a clinical trial, you want to try to eliminate all the variables that you possibly can. So, certainly, if you're treating a patient and you think they need cataract surgery, there's no problem at all of having it done and resuming the treatment. Treatment would be additive to whatever the patient has.

Dr. Do:
We had another question, Nora: How durable is photobiomodulation? We know in the clinical trial it was administered quarterly, but in clinical practice, could we deliver it less often, every 6 months? Do we have evidence for a different interval?

Dr. Lad:
That's a great question, very thoughtful, and I think it's great for future inquiry. We have data where, in between the LIGHTSITE III and IIIb, there was a good period which the patients did not receive treatment and the gains were maintained.

So there is potential for more durability. We just haven't had a study protocol to address this.

I know from the experience outside the US that's been done in other countries who have had the device for longer, and the patients have been extended successfully while maintaining the visual benefits, I think that that should be a great study to do.

Dr. Boyer:
Well, I think that also I agree with Dr. Lad, and I think that there has been a LIGHTSITE I, a LIGHTSITE II that have given us some additional information of having this regimen. I've always said retinal specialists are dyslexic because we never read labels or do the way that study's been designed. But Dr. Lad brings up a good point that there is evidence that this is a good treatment. After 2 years, perhaps you can spread it out more. Perhaps you might get a better result if you do it more frequently. But I think at this time, the FDA is only allowing us to do it 3 times a year. And obviously in the future, as we gain more experience and we look at the EUROLIGHT, perhaps we'll change what we do in year 3, 4, and 5.

Chapter 6: Case-Based Panel Discussion

Dr. Do:
We wanted to look at some clinical cases. So this is a patient who came into my clinic, 77-year-old woman, history of dry AMD. She complains of blurry vision and distortion, and she asks if there are any treatments besides the AREDS vitamins. Her vision has fluctuated between 20/40 and 20/30. Today, she's 20/30 in her office visit, and we can see on OCT she does have large drusen in both eyes.

Nora, would this be a good candidate for PBM?

Dr. Lad:
I think this would be a great candidate. It's at the better range of vision per label. However, if I look closely, I can see that, especially in the left eye, the ellipsoid zone, the mitochondria layer on OCT, is missing on top of those large drusen, so that distortion and the symptoms the patient is having are making sense to me. And probably that's the case if I were to scan through the whole OCT in the right eye. So blurry vision in this range of vision is towards the earlier stage of disease, but it's not unreasonable if that blurriness is maintained. If only occasionally, you'd have to document and see if it's dry eye component to it, and maybe that can be addressed by something else. But I've got to wonder.

Dr. Do:
What if the patient had 20/20 vision and was relatively asymptomatic, but you did notice on examination medium- and large-size drusen, David, would you recommend PBM in a 20/20 visual acuity eye?

Dr. Boyer:

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Well, I think we all would like to treat earlier, but we don't have the evidence to be able to really document that we should be doing that. The study was well done and gave us a lot of information. I think all of us that see patients with AMD have patients that are 20/20 for 4 or 5 years, and they may have drusen. But obviously, if the patient's vision decreases, I would certainly institute it. I'm hoping that in the future— nobody wants to do a 5-year study. I mean, if you have a 20/20 eye, you'd have to do a 5-year study to find out if there's really any benefit from treatment. I would just say I'd watch the 20/20 eye.

Dr. Do:
Let's look at another case right now. So we discussed the inclusion criteria for the LIGHTSITE III, so they did not include eyes that had 20/20 vision. But in clinical practice, we may treat according to the patient's symptoms. And if we go on to this next case, this is a 72-year-old woman. She has more blurry vision in the right eye than the left eye, but she heard about photobiomodulation because her friend is receiving it, and she wonders if she is a candidate.

Nora, tell us about what you see on the OCT, and is she a good candidate for PBM?

Dr. Lad:
That's an interesting case right here because the right eye has this lamellar hole or pseudohole that is distorting the inner retina. However, the patient does have these large drusen—intermediate to large drusen—vision is 20/60, probably partially due to dry AMD and perhaps a spot of GA too, because I see a hypertransmission defect there. So I think this is early GA. There are very few patients in the LIGHTSITE studies that had geographic atrophy, those more like this were not really well studied in the context of that study. So probably multifactorial vision loss in that eye, so that's a tough decision. But the left eye is asymptomatic. I'm assuming 20/32 is that early limit of vision. So this is a tough one.

David, what do you think? I would discuss risks, benefits, and alternatives. If she really wants it, we would try to see if we get a vision benefit for the dry AMD component. I would really carefully set expectations then, because she has other pathology, including GA and the lamellar hole, she might not improve as much.

Dr. Boyer:
Nora, I think, summarized it very, very well. This patient has early GA that will progress. There may be some benefit to the photoreceptors in that area, but the patient has to be counseled that they have several things going on, and I think Nora really summarized my feelings looking at this case.

Dr. Do:
Yes. And we mentioned that sometimes we can still after we have this good discussion with the patient, if she elected to go ahead with photobiomodulation in both eyes, would you treat both eyes on the same day? How do we administer PBM to both eyes on the same day, David?

Dr. Boyer:
Same day.

Dr. Do:
Yes.

Dr. Boyer:
The procedure itself the patients tolerate extremely well. Very, very few complaints from patients that have undergone this, and we've treated a large number of patients now. So we treat both eyes, and they can drive home because after a few minutes, their vision will return to normal. The only thing that some people complain of a little heat, which is really transient and goes away.

So, yeah, we treat both eyes rather than having them come back. It's very tedious to come back 3 times a week in my practice for 3 weeks. So we try to make it as convenient, and the patients don't need to be dilated.

People argue, "Oh, that's voodoo" if you don't dilate the patient, but there's actually studies that have shown the amount of light that gets to the retina is the same whether you have your eye closed or open, for those times when you have your eye closed.

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Dr. Do:
How many physicians in the audience already have a photobiomodulation machine? A few of you. Yes, great. And maybe you could share with us some of your experience if you want to. How have you been incorporated in your clinical practice?

[Inaudible audience member]

Okay, so maybe other office locations are utilizing it. Yes, and maybe I'll ask the physician in the middle there.

Audience Member 1:
The experience is so short but really is very short the time to explain something. But the patient tried to say me this happy or increase the vision, but I have to wait.

Dr. Do:
Yes. So for—

Audience Member 1:
So my question in the real practice is, how do you schedule the patient in your office? Three times in a week? Okay, but continual days? Monday, Wednesday, Friday? Or daily continually? Because I receive patient from far place, and she came to do the treatment, and pay the hotel, pay the stay, and it's so difficult, this.

Dr. Do:
Yes, you could do it 3 consecutive days, Monday, Tuesday, Wednesday. Yes. So it's flexibility. We don't do 2 sessions in 1 day, but yes, the 3 times, 2 to 3 times a week is flexible.

How about you, David?

Dr. Boyer:
I think the EUROLIGHT is going to give us more information about that. I think you can treat– I've had patients that had gone to Europe and received treatment and continued to go back, and they were getting 5 treatments, 1 treatment a day for 1 week, and then a few extra treatments the next week, because it's long distance and hotel rooms. But I think we're going to find out the ideal for that group of patients, but I try— in Los Angeles, there's probably where the number of PBM machines is equivalent to Starbucks. There are a lot of us, and so it's not a transportation problem. So I think we'll answer that question in a longer study.

Dr. Do:
Great. Well, let's move to this case. This is an 83-year-old gentleman who has a history of wet AMD in the left eye, and he is undergoing anti-VEGF treatment for the left eye. He has some mild distortion in the right eye and was wondering if there was any new therapy for that eye.

Nora, what would you recommend in this clinical case? He asks, “Can I treat the right eye, and is there another treatment also for the left eye?”

Dr. Lad:
Okay, so undergoing treatment, assuming anti-VEGF injections for wet AMD and wet control, since I don't see any exudation, although it appears there's a fibrovascular PED, so I would continue treatment for that eye, first of all, for the left eye.

The right has equal vision, but I think it's less because the patient has a subfoveal deposit there that's probably blurring the vision, and it sounds like the patient is concerned that he might develop advanced, especially wet AMD in that eye. So I would discuss photobiomodulation and the study and risk benefits and alternatives, and so I'd recommend it in both eyes or just the left eye. I wouldn't do it in the left eye because that's already exudating and that's an exclusion criterion in the trial, so we don't have data. Although it sounds like in Europe they are using it for wet AMD, but we don't have the evidence in the United States for this or the approval. And I would recommend photobiomodulation in the right eye, especially if the patient is concerned about progressing to the late stage, the GA wet AMD, in he's going to say his good eye. Although they're equal in vision.

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Dr. Do:
What are your thoughts, David?

Dr. Boyer:
No, I agree with Dr. Lad. Nora, I think there's a pachychoroid here to some degree, and that 20/40 eye may have a cataract or some other or something else going on there. My concern is they already have wet in 1 eye; I have to explain to them that the risk in the other eye is not insignificant. So they have about a 7%— Now, the natural history tells me it's going to be at 3.5% to 4% risk over a 2-year period. So I'd be a little reluctant because I don't want them to come back and say, “Oh, you've caused my neovascularization,” because they do read online that it can cause it. So I'd be concerned about this patient, try to figure out why it's really 20/40. Because the EZ zone looks intact, but maybe it's broken up there. I don't know.

Dr. Do:
David, in your experience, or anybody in the audience, if you're treating a patient who has dry AMD with PBM, but then maybe a year later develops wet AMD in that eye that has been receiving PBM, do you stop PBM completely while you're continuing the anti-VEGF? We don't have any evidence to combine the 2, but that could be a scenario.

Dr. Boyer:
Yeah, no, that's a great question. I can tell you what I do, but if you ask me for the science behind it that's kind of weak. I continue the treatment. I have one person who insisted he was treated elsewhere and developed an RPE rip. If I treated him, he would have developed an RPE rip, but I didn't treat him. And I said, “You may need anti-VEGF occasionally,” but he was doing well. He demanded to do it. He said, “I'll pay out of pocket to do it.” He swears that his vision gets better with the treatment.

Dr. Do:
With PBM?

Dr. Boyer:
With PBM. Absolutely swears. He said, “I want to do it 4 times a year, and I'm going to pay extra for it.” And I said I have no evidence that it's a benefit. He says, “I know it's helping me.” So, you know, my N of 1 would indicate that you probably can treat these patients, and maybe it'll be beneficial.

Dr. Do:
What about, Nora, eyes with foveal-involving geographic atrophy? We have patients who are desperate for treatment; they may or may not want intravitreal complement inhibitors, and they ask, “Is PBM beneficial?” Can they try that for the geographic atrophy? What are your thoughts?

Dr. Lad:
It's a great idea, but we don't have evidence for this. It hasn't been studied. We had very few patients in the trial that had non-subfoveal GA, so we don't have a lot of information, and we can't glean conclusions from that subpopulation, so I wouldn't recommend it. I'd explain that we don't have evidence at this time. The study hasn't been done yet. Even if they have large drusen, and I think the visual benefit would probably not be significant for them to notice the difference.

Dr. Do:
How about you, David? Let's say they have extrafoveal GA, because in the LIGHTSITE III study, eyes with extrafoveal GA were allowed if they also had drusen, and that post hoc analysis seemed to show it decreased the incidence of foveal-involving GA. Would you treat a patient who was really concerned about developing progression of their GA?

Dr. Boyer:
I totally agree with Dr. Lad. We don't have the evidence not to do it, but we do have evidence that complement inhibitors are somewhat beneficial. Certainly, it's a question of whether we would get reimbursed if we did both. That's the other question. If you use a complement inhibitor in someone who had extrafoveal and did this, is it additive? And I don't have the answer to that.

Dr. Do:
Great. Any questions from our participants in the audience?

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We discussed a lot today. Dr. Lad went over the complex pathophysiology of AMD. She discussed evidence-based nutrition and the rationale for photobiomodulation. Dr. Boyer did an excellent job reviewing the LIGHTSITE III clinical trial and all the secondary endpoints that were also evaluated in that randomized clinical trial. And then we discussed some real-world cases that we see every day in our busy clinics.

We hope this session has been educational for all of you.

Chapter 7: Audience Q&A

Dr. Boyer:
I have a real quick question that I'd like to ask the audience.

Dr. Do:
Yes.

Dr. Boyer:
How many of you have considered photobiomodulation? And if you have, what is your hesitation? Does anybody want to answer?

Dr. Do:
Consider buying it for your office and to treat patients.

Dr. Boyer:
We're sending patients in to be treated.

Dr. Do:
Yes. Yes.

Audience Member 2:
We're going to have it eventually at the university. But we are waiting to learn more about the mechanism, as has been explained today, and what other people with practice are doing. But yes, certainly we're waiting on the instrument.

Dr. Boyer:
Any other comments? I'm just curious.

Audience Member 3:
Now, is photobiomodulation covered by Medicare?

Dr. Boyer:
It is in California.

Dr. Lad:
What are the 5 Medicare regions I just learned recently? North Carolina, where I practice, unfortunately, doesn't cover it yet, so it's out of pocket still.

Audience Member 3:
So, state by state.

Dr. Lad:
State by state.

Audience Member 3:
Because area by area, that introduces an element of inequity because there may be patients who can't afford it.

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Dr. Boyer:
Absolutely.

Dr. Do:
I agree. I think there was another comment here. No, okay. Great.

Well, thank you so much for joining us tonight. Really enjoyed discussing this with David and Nora, and answering the questions that people presented. We'll be here to enjoy some refreshments. Feel free to come and talk to us. Thank you so much.

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