So we’re going to switch gears a little bit right now and talk a little bit about macular degeneration, or in this case actually things that can sort of look like macular degeneration but aren’t.

So there’s a couple of goals here in mind, one is to help remind you to keep an open mind when you are evaluating patients that possibly may have AMD because sometimes it can be confusing. And it helps a lot to think about these different diagnoses that I’m going to go through. This also helps in clearing up patient confusion. I’m sure we’ve all seen patients where they may have been seen somewhere else and given a diagnosis and they come in for a second opinion, or they just wanted to have more information about what’s going on and they think that keeping this list of things in mind will help you with that.

And the ultimate goal here is to deliver appropriate care. It’s hard to treat somebody if you don’t have the diagnosis right, so I think that’s also very important. And also, especially now with the advent of anti vegf therapy, you know these intravitreal injections are done more and more often and fortunately they are quite safe but there is a small risk of having a severe infection, so I think that before you commit patients to having these injections you are pretty confident in your diagnosis.

So I’m not a huge fan of lists, so the whole point of this is to sort of give you visual reminders of different things and hopefully some of these will stick in and help you when you are seeing these patients in the office. All right, so just really basic information, things to keep in mind, always think
about the patient’s age, family history and also the symptoms you know in terms of duration and the severity.

So the first part of my talk here I’m going to review a couple of different things that can sort of look like nonexudative AMD. The first one is chronic central serous chorioretinopathy. These patients – let me go back a second here – they may or may not actively be leaking when you see them in the office but when they are not leaking they can develop these RPE changes which can sometimes be confused with changes related to AMD.

So this was a 39 year old gentleman and he had complained of waxing and waning vision, blurry vision over the last year and a half and this was his fluorescein angiogram which showed hyperfluorescence in the area of those pigmentary changes that you could see on the color photograph with late staining in the end. I didn’t include an OCT here but it at that time did not show any leakage. There is two flavors, there is the acute and the chronic forms, which I’m sure you are all familiar with.

Something else that we can not too infrequently see in the office is familial drusen. This was a 51 year old gentleman and he had no visual complaints, his vision was 20/20 OU and he had had a history of having these drusen type appearance in his fundus for years. So this is an inherited disorder, autosomal dominant with varying degrees of expression and these patients get
accumulations of hylan deposits in Bruch’s membrane, sometimes in the RPE cells as well, and this is thought to be an inborn errant metabolism.

So the deposits typically appear younger, at a younger age than you would expect for age related macular degeneration, usually 3\(^{\text{rd}}\) and 4\(^{\text{th}}\) decades, and they gradually accumulate over time. And if you are fortunate enough to examine other people in the family you may find similar findings, which can help to sort of clue you in on that diagnosis. These patients can develop metamorphopsia, and eventually they can sometimes have decreases in visual acuity.

Something else that we don’t see that frequently but I thought it would be interesting to include is the membranoproliferative glomerulonephritis type II. This is a systemic condition, we just happen to be able to see it pretty well inside the eye. These patients get build-up of deposits in basement membranes in various organs of the body and in Bruch’s membrane which is where we see it, and these can resemble drusen. Late in the course of this it can be complicated by choroidal devascularization which also can make the diagnosis a little more confusing except you already have the systemic diagnosis most likely.

This is sort of a hodgepodge of various dystrophies, I just want to include some pictures here just to get your mind going a little bit. This is somebody with a pattern dystrophy, you can see pigmentary changes as well as little spots of atrophy, this is someone in their, I believe they were in their late 30’s or 40’s, pretty much asymptomatic.
This is somebody with Stargardt’s that has the more classical appearance. This is somebody who had come in and I believe he was in his 40’s, he had been diagnosed with AMD but you can see there’s pretty extensive atrophy there in the macula with basically a foveal island of vision remaining.

And on a similar note here was someone else who came in, also a little bit younger than you what you would expect for someone with AMD but you can see here these patches of atrophy with a small area of foveal sparing.

I’m going to spend a little bit more time talking about this a little bit later in my talk but this is someone with adult vitelliform dystrophy or pseudo vitelliform dystrophy and this can take on various appearances so here’s an example of one patient where the vision is about 22-100 and this is sort of a later stage in the process. This is someone else in a younger age group who also has this and earlier stage where you can see pigmentary changes and this sort of orange-ish lesion in the foveal area and this has the classic appearance on OCT, have this nice dome shape subfoveal lesion here and usually the vision is pretty good in these patients. Sometimes they don’t even notice that they have this.
Something else that can sort of fall into that category of things that look like dry AMD are drug toxicities. The main one we see the most often is ______ toxicity, this can cause RPE changes in the macula so here’s an example of that. And here’s another patient also with ______ toxicity.

So I’m going to switch gears here a little bit and talk a little bit more about exudate of AMD and things that can cause that. You know we see a lot of patients that tend to fall into the older age groups and like a lot of things, a lot of these problems start to happen when you get older, so you may have been following the patient with drusen and one day they come in and now they have a hemorrhage in their macula and you’re sort of left wondering if this exudate of AMD or is this something else.

So I’m going to review a couple of different things here that can sort of take on that kind of an appearance. The first one is macular aneurysm. So these are usually unilateral and more common in older age groups especially a hypertensive, long history of hypertension and these can cause hemorrhages in the subretinal, intraretinal and preretinal spaces. And in the more chronic forms you can get more exudate then hemorrhage on exam.

So this is another photograph of somebody with macular aneurysm and a preretinal hemorrhage that’s actually been there long enough to start layering. If you’re fortunate you can actually see the macular aneurysm, they tend to originate over, they do originate over a retinal artery, that’s where the
aneurysm is. Sometimes fluorescein can be helpful with this especially if you it’s not quite clear where the hemorrhage is coming from. And these can be treated with laser.

So this is a more chronic macular aneurysm where you don’t see a lot of hemorrhage, it’s mainly exudate. But on fluorescein it lights up pretty nicely. And this is another patient with a macular aneurysm and there it is on the fluorescein. I just mentioned this before but this sort of overlaps and can fall into both categories of wet and dry AMD.

So this has an onset in adulthood and you get these yellow-orange pigmentary changes in the macula and they can sometimes have an elevated appearance which can lead to confusion because then you’re wondering if this is a choroid neovascular membrane hiding out there. This is something else. And these can also be associated with choroidal neovascular membranes. So this is the same patient but showing both eyes so this person had adult vitelliform and they happen to develop later on a peripapillary choroid on the neovascular membrane and the patient had noticed this as a new blind spot in their vision and they were treated with ANTI-VEGF therapy.

This is just the fluorescein showing you that there’s a substantial subretinal hemorrhage there and in the later phases you can sort of see there’s this ill-defined hypofluorescence underneath the hemorrhage sort of telling you that that’s where the membrane is at. Whereas pseudo-vitelliform regions is just sort of staining.
Some more photograph from before, this is just someone that doesn’t have that sort of end stage appearance, this is more just pigmentary changes and a little raised lesion on exam and pretty good vision.

There’s the OCT again. Okay, so this is something we don’t see quite as common but I think it’s important to think about. This is juxtafoveal telangiectasis, this is a retinal vascular disorder and it’s caused by irregular capillary dilation and incompetence of the vessels near the fovea. It’s thought to occur near the fovea because this is sort of a watershed zone in terms of the circulation. It does have an association with diabetes and it can be congenital or acquired.

It’s divided into three groups, groups 1, 2 and 3 and I won’t spend too much time differentiating them except that group 2 seems to be the most common type. This is a patient with this and you can see next to the fovea there there’s an area of hyperpigmentation which you can see sometimes with this especially in Type 2. And here it is on the fluorescein early stage, a little bit of hyperfluorescence in that area, some blockage from the pigment and then later in the fluorescein you can see the edema present.

Here’s another patient, you can see it a temporal to the fovea, there’s all of these mall microvascular abnormalities and quite a bit of exudate. On the fluorescein you can see that’s the vascular disturbance actually involves a larger area then you would think just looking at the color photograph
that surrounds the fovea and extends temporally and in the late stages pretty significant cystoid edema.

So in terms of treatment there really isn’t an effective treatment for this, photocoagulation doesn’t seem to work very well, I know there’s trials going on looking at anti-VEGF therapy for this. The visual acuity can vary greatly. Some patients hardly have any difficulty from this and others have substantial vision loss. Some types lead to atrophy of the fovea and permanent vision loss. And this can also be complicated by choroidal neovascularization. Something that I just wanted to throw in there as a reminder is the BRVO, has a pretty typical appearance but in some cases it can be a little bit hard to figure out, maybe if you catch it at an earlier stage or if you see a patient a few months after its happened and the hemorrhages have started to go away, then sometimes it’s a little bit harder to see what’s causing that hemorrhage that you see on exam.

You know in the classic definition you’ll see hemorrhages, cotton wool spots and microvascular abnormalities along the distribution of one of the branch retinal veins. Here’s another disorder that can sometimes pass through your clinic, this is a patient with Coats Disease. They can get hemorrhages and exudates either on the periphery or in some cases closer to the macula which can lead to some confusion sometimes. This is a patient that had been treated with laser, you can see that there’s various laser spots already present there in the temporal aspect of the macula.
This is an idiopathic condition and these patients have, can have multiple vascular abnormalities, aneurysms, telangiectatic vessels, intraretinal and subretinal exudates. I have been seeing a patient with this now for a couple of years and from what I can tell the best diagnosis I can give her is Coats Disease. But her one macular looks totally normal and the other one all the vessels around the macula have been treated for aneurysms in the past and she’s had multiple sessions of laser in other places and edema and substantial loss of vision in that eye.

I believe she had also been treated with anti-VEGF therapy which it didn’t really respond to. So these can present either in juvenile or adult and it’s thought to be, to have some sort of genetic basis. So these patients can have severe vision loss, retinal detachment and the treatment of choice is laser and these can also be associated with choroidal neovascular membranes in some patients and if that occurs then anti-VEGF therapy would be recommended.

This is one of these things that can sort of overlap into the wet or dry categories. Once again our good friend central serous, this is somebody who comes in with the classic serous detachment in the macula. I’m going to wrap this up then with a case, a patient of mine that I’ve been seeing now for about a year. She came in complaining of several months of decreased vision in her right eye and she’s actually been treated with anti-VEGF previously with no response. On her exam you can see that there are some pigmentary changes in the macula in both eyes and you don’t really see an obvious hemorrhage. The thing that gives this away that this wasn’t just your typical AMD, in the left eye you can see she has pigmentary changes that form what we call a gutter basically. This is
someone who’s had central serous in the fluid sort of tracks down with gravity and leads to these sort of like a track of RPE changes.

This is what her OCT looked like when she came in. You can see in the right eye which is on the left, there’s subretinal as well intraretinal fluid and it seems to be emanating from an area just superior to the optic disk and in her left eye you can see that there’s fluid there as well and it seems to be coming from the area just temporal to the disk. And this is with the fluorescein angiogram looked like, you can see some areas of hyperfluorescence in both eyes that go along with these areas of pigmentary changes.

And on ICG angiography it’s a little bit hard to see in these photos but in the right eye which is on the left, there’s like a sort of ill-defined area of hyperfluorescence just above and temporal to the disk and the left eye, you can’t really see it that well here but there was a similarity of hyperfluorescence just temporal to the disk, both of these areas sort of corresponded with the areas that you saw where there was fluid on the OCT. So that was very helpful.

So this patient actually underwent photodynamic therapy and this was taken one month after PDT was done and on the right eye even though there’s still some fluid there still, it does look improved compared to the pretreatment OCT. The left eye had completely dried even one month out. No fluid is there now.
So I just, I sort of followed her because her vision had improved and she was symptomatically doing much better. So three months after the treatment this was the right eye, fluid is still sort of there but it’s going away. Six months later you can see there’s still a little bit there, just near the optic disk and this was more recently, I just saw her ten months out and there’s just a tiny trace near the optic disk but her vision is 20-25 OU and the left eye has stayed dry the whole time.

So, you know, given the time constraints and it’s just a little bit overwhelming to go through all these things, there’s other things here that I didn’t really talk a lot about. There’s various inflammatory conditions which can also resemble exudative AMD such as choroiditis, VKH or posterior scleritis. You have to look at the type of symptoms the patient is having with these type of diagnoses and obviously also choroidal tumors which can depending on where they’re located can resemble exudative AMD as well.

So on conclusion I hope this helped refresh your memory and remind you that there’s a variety of conditions that can resemble nonexudative and exudative AMD and sometimes or in all cases actually, examining the macula and the periphery can help sort of clue you in on what’s going on and keeping these diagnoses in mind can help you find out what the correct diagnosis is and it may help you in cases where the diagnosis of AMD doesn’t quite fit. And this would help direct you towards the right treatment.

Thanks a lot.