

Hypertensive Retinopathy Course # 40048

Instructor:

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Section:

Systemic Disease

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COURSE DESCRIPTION:

This course focuses on the clinical features, diagnosis and management of hypertensive retinopathy. Additionally, some background and statistics on systemic hypertension is also presented.

LEARNING OBJECTIVES:

- Know the retinal signs of hypertension
- Understand importance of checking blood pressure and knowing when to refer immediately
- Understand general background and current statistics of systemic hypertension
- Know how to manage and co-manage these types of patients
- Understand early detection and proper referrals can save lives

Hello, I am Dr. Aimee Ho, and welcome to Pacific University's online CE course on Hypertensive Retinopathy. For this course, we will be reviewing systemic hypertension, ocular manifestations of hypertension, and the management of these patients. Without further ado, let's get started.

I would like to start with presenting a case. This is a 45 year old Caucasian male. He presents for his 1st eye exam back in 2005. He complains of blurry vision at near without an Rx. His ocular history and medical history is unremarkable, and he's not on any medications. For his occupation, he reports being an attorney in a very high stress environment. BCVA is 20/20 in the right eye, and 20/20 in the left. Pupils, EOM's and confrontation visual fields were all unremarkable at that visit. The anterior segment was also unremarkable.

Table 1

1 st Exam: 6/12/05
CC: Blurry vision at near without Rx
BCVA 20/20 OD, OS
Pupils: ERRL, (-) APD
EOMs: SAFE
FDT VFs: Full OD, OS
Anterior Segment OU: Unremarkable

Table 2

Posterior Segment OU
Multiple scattered cotton wool spots
Scattered flame hemorrhages
Scattered patches of exudates
Vessels: moderate tortuosity moderate A/V nicking
Macula: flat, even pigment
BP: 185/110 mmHg

After dilation, looking at the posterior segment, the optic nerves looked healthy with pink rims, distinct margins, and a small C/D ratio. In the posterior pole, there were noted multiple scattered cotton wool spots (CWS), a few scattered flame hemes, and a few scattered small patches of exudates. Vessels were noted to have moderate tortuosity and moderate A/V nicking. The macula was flat and had even pigment. Blood pressure was taken that day, and it was at 185/110 mmHg.

The patient was educated on the findings and was advised to seek immediate medical attention. I am not aware of all the details that happened at that visit, but I do believe that the patient was driven to the emergency room right after that eye exam.

That same patient, returning 5 years later, on 6/12/2010, for a routine eye exam. He doesn't have a chief complaint this time – just coming in for a general eye exam. His ocular history is now remarkable for hypertensive retinopathy. His medical history now includes hypertension, and he's now controlled with Lisinopril. He is retired now due to the finding five years ago. He believed that the hypertension was caused by stress, so he retired immediately after that. His BCVA is 20/20 OD and OS. Pupils, EOMs and confrontation visual fields were all unremarkable at that visit. The anterior segment findings include a little bit of beginning cataracts. With dilation, the posterior segment showed that, again, the optic nerves were healthy. The posterior pole looked great this time – there were no hemes, CWS or exudates. The vessels still had that moderate amount of tortuosity and A/V nicking. The macula still looked great with flat & even pigment. The blood pressure was once again taken, and it was 118/80. Thus, a lot better than before.

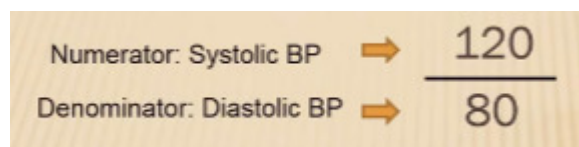
Hypertension – the silent killer

Let's talk a little bit about hypertension (HTN) now. It is often called the silent killer. The reason for that is because often times patients are asymptomatic – they will typically come in with no symptoms,



not knowing that they even have any problems or have hypertension. There is really no way of knowing if someone has hypertension unless you actually get your blood pressure taken. Blood pressure can be measured either at home with an automatic blood pressure instrument, or with a medically trained professional like a nurse at the doctor's office.

Figure 1: A standard blood pressure reading.



Let's do a quick review on blood pressure. Blood pressure is the force of the blood pressing against the blood vessel walls as the heart pumps the blood. High blood pressure, or HTN, is an increase in the amount of this force that the blood places on the blood vessels as it moves through the body. Factors that can increase this force include higher blood volume due to extra fluid in the blood, or blood vessels that are narrow, stiff, or clogged.

Blood pressure readings are normally given as a fraction (Fig 1) with a numerator and a denominator. The numerator will notate the systolic blood pressure, which measures the pressure in the arteries during heart beats. This is during each contraction of the left ventricle, pushing out the blood into the rest of the body. This is where the blood force is actually the highest, where the blood is trying to rush through to the rest of the body and is pushing hard against the blood vessel walls. The systolic blood pressure will always be higher. The denominator, in contrast, the diastolic pressure, measures the pressure in the arteries between heart beats, when the heart is at rest. There is lower pressure here, so there is not as much pressure of the blood against the blood vessel walls, so this pressure will always be lower.

Figure 2: Classification of the stages of hypertension

The way we define HTN is any blood pressure measurement that is equal to or above 140 systolic or 90 diastolic mmHg (140/90). You can see here in Figure 2 that actually there is a normal, which is anything below 120 mmHg for systolic and below 80 mmHg for diastolic. There's a little grey area of "prehypertension". This grey area is abnormal, but it is not yet at the hypertensive stage. Finally, once you get to the 140 systolic or 90 diastolic, then you hit the hypertensive stages, which essentially is divided into four stages, seen here.

BP Classification	Systolic BP	Diastolic BP
Normal	<120 mmHg	and <80 mmHg
Pre-hypertension	120-139mmHg	Or 80-89mmHg
Stage 1 HTN	140-159mmHg	Or 90-99mmHg
Stage 2 HTN	160-179 mmHg	Or 100-109 mmHg
Stage 3 HTN	180-209 mmHg	Or 110-119 mmHg
Stage 4 HTN	>210 mmHg	>120mmHg

Stage 1 and stage 2 are more of the chronic stages. Blood pressure is high, but it is not a hypertensive crisis. Stage 3 or 4, if you ever get anyone in these ranges, are considered to be in hypertensive crisis, and it warrants emergency care. It is very important when you have a patient in your exam room and you get a high blood pressure reading, that you wait a little bit and take the blood pressure again. Sometimes patients may just be rushing into the office, they may be running to get there on time, or maybe they have White Coat Syndrome, so it does take a little while for the patient to calm down and for things to settle down a little bit. Thus, take their blood pressure reading again. If you get a high reading a second time, then it confirms the 1st high reading, and it is considered a confirmed high reading at that point. Otherwise, I would not act on anything if I got a high reading on the very first try.

If you do take the blood pressure again and you do get very high measurements, in Stage 3 or Stage 4 HTN, then this is considered a hypertensive crisis. A hypertensive crisis is essentially divided into two categories: there is urgent, and there is emergency. An urgent hypertensive crisis, the systolic blood pressure is 180 or higher, or 110 or higher for diastolic, but there is no associated organ damage. In an emergency hypertensive crisis, your blood pressure is exceeding 180 mmHg systolic, or 120 mmHg diastolic. At this point, there is actually damage to your organs at this point. In an emergency hypertensive crisis, there can be associated life-threatening complications, and sometimes patients can exhibit signs and symptoms. If the patient does have a really high blood pressure reading over 180 systolic or over 120 diastolic and they are coming in with severe chest pain or a severe headache accompanied by confusion or blurred vision, nausea, vomiting, severe anxiety, shortness of breath, seizures, or they are unresponsive at that point, those are all signs of an emergent, life-threatening condition. Even if they don't have these signs or symptoms, but they are getting extremely high blood pressure readings, it is still considered a crisis and still warrants urgent or emergency care at that point.

Table 3

HTN Risk Factors
Family History
Ethnicity AA > Whites > Mexican > Other
Age > 55 YO
Gender Up to 45 YO: Men > Women 45-64 YO: Men = Women Over 65 YO: Women > Men
Lack of Exercise
Poor diet (too much salt)
Overweight & Obese
Excessive Alcohol Consumption

Who is at risk for hypertension? Certainly family history is a big one. If everybody in their family has HTN, they are more at risk for developing it. Race is actually a risk for developing HTN, as African-Americans have a two-fold risk factor for developing HTN. Then the next most common is Caucasians, then Mexicans, then all other races. There was a study that found that more than 40% of non-Hispanic African Americans have HTN. These are some very alarming statistics.

Aging is definitely a risk factor. Anyone over 55 years old is automatically at a higher risk.

Gender is an interesting risk factor. If you are younger than 45 years old, men are at a higher risk for developing HTN than women. Somewhere between 45 and 64 years of age, women and men's risk factor is about the same. Once you are over 65 years old, women are actually at a higher risk than men for developing HTN. There is a very interesting dynamic here as the risk factor switches from men to women as your age increases.

Lack of exercise and poor diet go hand-in-hand in causing possibly overweight and obesity, which are all risk factors for developing HTN. Poor diet, especially, if you consume too much salt, causes you to retain fluid, which gets filtered into your vascular system giving you an excess amount of fluid in the vascular system, increasing your blood pressure.

Excessive alcohol consumption also puts you at a higher risk for developing HTN.

Other contributing risk factors can include smoking or second-hand smoking, stress (as in my patient), and obstructive sleep apnea (OSA). These are all contributing risk factors linked to developing HTN.

Some alarming statistics that I've found is that actually 1 in 3 US adults, or about 80 million Americans have hypertension. Of these 80 million Americans, only 54% have the condition under control. This is definitely very alarming. HTN was either the primary or a contributing cause of death for more than

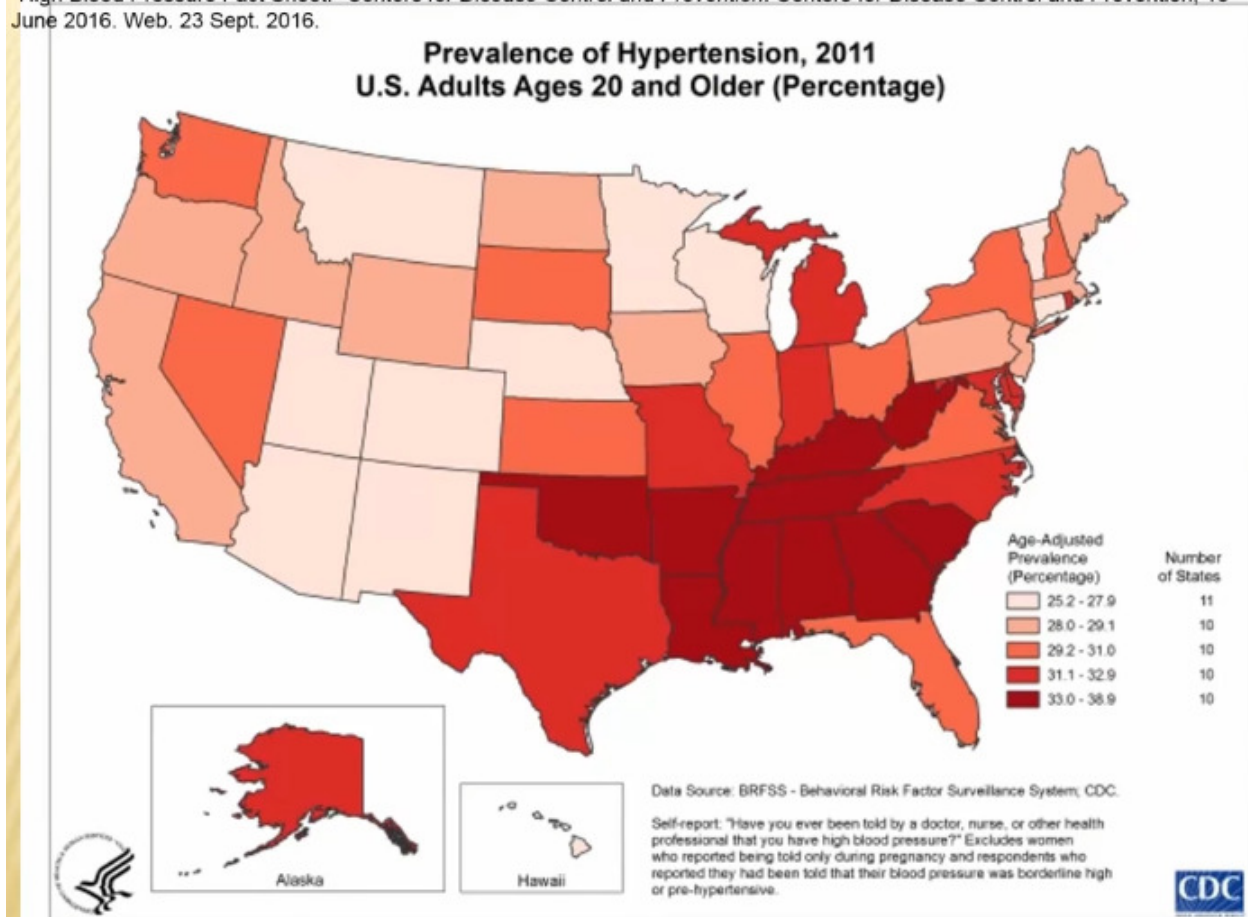


Figure 3: A demographic map showing the prevalence of hypertension in the US population, broken down by state.

410,000 Americans in 2014. I apologize that the data is a little bit dated, but I assume that the numbers are even more alarming or even higher today if we had more update statistics on it.

Figure 3 is a demographic map of the United States when we look at the prevalence of HTN. You can see that the darker red colors are actually states that have the highest prevalence of hypertension. As you go the lighter gradient of red, as in some of these Midwestern states, it looks like they are least prevalent in HTN. Even these states that are at the least-prevalent, their hypertensive rates are still at 25 to 28%, which is still pretty high.

Where does hypertension come from? HTN is divided into two groups: Primary Hypertension and Secondary Hypertension. With Primary HTN there is really no known underlying cause, the patient just has HTN for whatever reason. With Secondary HTN, there is usually a cause that you can link to causing their increased blood pressure.

Table 4 is a list of some of the usual suspects for causing

Secondary HTN. Preeclampsia and eclampsia are associated with pregnancy, and are actually very dangerous for both the mother and the baby. A pheochromocytoma is a neuro-endocrine tumor of the adrenal glands, which causes high amounts of secretion of catecholamines, which are mostly

Table 4

Secondary HTN causes
Preeclampsia/eclampsia
Pheochromocytoma
Kidney disease
Adrenal disease
Coarctation or abnormality of the aorta

norepinephrine and a little bit of epinephrine. This increased secretion causes things like palpitations, anxiety, elevated heart rate, and elevated blood pressure.

Kidney disease is something else that is pretty common in causing secondary HTN. Kidney disease that causes HTN is also known as renal hypertension. This essentially happens because the arteries that are delivering blood to the kidneys actually narrow, and once these arteries narrow, so less blood is going to the kidneys and the kidneys are thus receiving low blood flow. The kidneys perceive this low amount of blood flow as the body being dehydrated, so they respond by releasing a bunch of hormones to stimulate the body to retain sodium and water. In the end, blood vessels fill up with this additional fluid, and blood pressure goes up.

Adrenal disease is actually very similar to pheochromocytoma in that it releases a high amount of catecholamines that end up spiking the blood pressure.

The last reason that I have on here as causing secondary HTN is coarctation or abnormality of the aorta. Coarctation in itself means a narrowing, so a coarctation of the aorta is essentially a narrowing of the aorta at the aortic arch. Since the aorta is narrowed, the left ventricle of your heart has to generate a higher pressure than normal in order to force enough blood out to get it to the rest of the body, which causes that spike in the blood pressure.

Hypertensive Retinopathy

Now that we've reviewed a little bit about systemic

hypertension, let's discuss hypertensive retinopathy. The definition of hypertensive retinopathy is any retinal vascular changes secondary to chronic or acutely elevated systemic blood pressure. Essentially, a person has high blood pressure systemically, and it's caused vasculature changes in the back of the eye.

In hypertensive subjects, people who do have HTN, the prevalence of retinopathy has estimated to be well over 70% in these patients, so definitely something to be looking for in our patients. In addition, hypertensive retinopathy is a marker for subclinical cerebral disease. It is associated with a risk of stroke and with a higher mortality. Again, this gives us more motivation to look for these retinopathy changes due to hypertension.

In general, the patient is going to come in asymptomatic – they will not have any visual symptoms. In very, very rare instances, the patient may come in complaining of decreased vision. If the patient does come in with decreased vision and it truly is due to HTN, then it is a very, very severe case of hypertensive retinopathy. We will talk more about why that is, later. Usually, most cases are asymptomatic. Often times, the eye exam will be the first clue to systemic hypertension, so it is very important to look for these signs in the back of the eye.

In hypertensive subjects, the prevalence of retinopathy has been estimated at well over 70%

Table 5

Signs of Hypertensive Retinopathy
Almost always bilateral and symmetrical
Arteriolar attenuation
Arteriolar light reflex
Arteriovenous crossing changes
Retinal hemorrhages
Cotton wool spots
Exudates
Vessel sheathing
Optic nerve swelling

So what exactly are you looking for? Table 5 lists some of the common signs of hypertensive retinopathy there. Something to always note is that hypertensive retinopathy is almost always bilateral and symmetrical. A lot of things that happen systemically, including hypertension, will affect both eyes

equally. On that same note, if you're really convinced that the changes in the back of the eye are due to hypertension, but what if you find all this hypertensive retinopathy, but it's unilateral instead of bilateral. What are you thinking in this case? If that ever does happen, and you're super convinced that the findings are truly hypertensive retinopathy, and it's not another co-morbidity – which is something you always have to differentiate, as a lot of hypertensive patients also have diabetes – in those cases you really have to differentiate if it's a diabetic change or a hypertensive change, either-or. Maybe there are vein occlusions or different things going on with the patients. If you are really sure it is a hypertensive change and it's showing up unilaterally, then you really have to think about your carotid artery stenosis. If there is any carotid artery blockage, then it will cause a unilateral presentation of hypertensive retinopathy. The eye that looks more normal is the side that has the carotid artery blockage, because the blockage is preventing the high pressure from causing damage to the retinal vessels, which is why the normal eye is the unaffected side. Something to keep in mind.

Moving on with the signs. I really think the top three listed in Table 5, arteriolar attenuation, arteriolar light reflex, and arteriovenous crossing changes, are more specific for finding hypertensive retinopathy. The rest of what I've listed here, hemorrhages, CWS, exudates, vessel sheathing and optic nerve swelling can actually occur if there are other systemic conditions going on with the patient, so they are not as specific to hypertensive retinopathy, so I would encourage you as a practitioner to look for the 1st three as more specific for hypertensive retinopathy. If any of the others show up, that's great, but the top 3 are what we are looking for, as they are more specific.

You may be wondering why I didn't list vessel tortuosity. Isn't that a sign? Vessel tortuosity by itself is actually not a sign of hypertensive retinopathy. If you do find segmental tortuosity, that is a little more of an indication that could be a sign of hypertensive retinopathy, and that most commonly occurs in the nasal retina. A larger reason why we don't really pay too much attention to vessel tortuosity is that 80% of patients with hypertension do not present with vascular tortuosity. Thus, it is not as helpful for us to look for vessel tortuosity. A lot of times, when you see vessel tortuosity, and you don't see any other signs of hypertensive retinopathy, that vessel tortuosity is just congenital. However, if you do see segmental tortuosity, and you do believe it is due to HTN, you can record it as vascular tortuosity, recording the severity and the location that you find it at.

Attenuation

Let's start with the first sign I have listed above: arteriolar attenuation. This is looking at the caliber of your arteries compared to your veins. The normal A/V ratio is 2/3 or 3/4. Essentially, the caliber of the arteries should almost be the same as the caliber of the vein. Anything below those ratios are abnormal. You can see in Figure 4 [Left] with large, possibly glaucomatous cupping here, if you ignore that and focus on the vessels, you can see how the superior artery is pretty narrow compared to the vein that is right next to it. You can see that also with the artery exiting the nerve at about 4:00, how attenuated it is, how narrow it is compared to the veins. This is very concerning and indicative of hypertensive changes here.

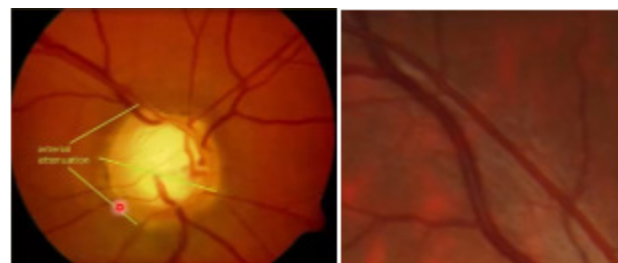


Figure 4 [Left] A photo showing attenuated arteries.

[Right] Another example of arteriolar attenuation.

Something to note is that attenuation will show up differently if you have a younger patient compared to an older patient. In younger patients, these patients have autoregulation of the arteries. As a result,

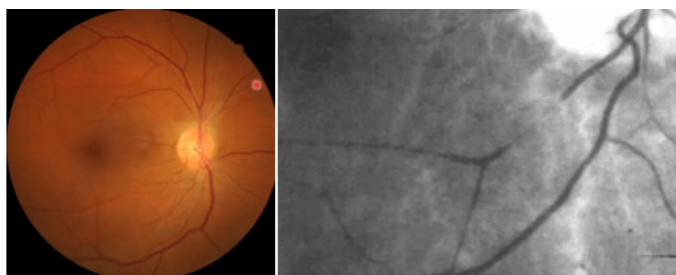


Figure 5 [Left] General attenuation in a younger patient with hypertensive retinopathy. [Right] Focal attenuation in an older patient with hypertensive retinopathy.

when you have that high blood pressure, the autoregulation combined with the malleability of the arteries causes a uniform narrowing of the arterioles. With older patients who are undergoing arteriosclerosis, some parts of their arteries are more stiff than others, and there is still some form of autoregulation going on, so a lot of time that leads to focal narrowing of the arterioles.

Figure 5 is an example of general or uniform

attenuation compared to focal attenuation. In Figure 5 [Right], the parts of the artery that have not narrowed, that is most likely due to the fact that it is stiff or has arteriosclerosis and thus is not responding to autoregulation. The attenuated portion is a little bit more malleable, and as such is responding to the autoregulation by narrowing in that area.

Arteriolar Light Reflex

Arteriolar light reflex (ALR) is the next sign I want to talk about here.

Essentially with this you are just looking for the light reflex that is bouncing off the surface of the blood column. As you can see in Figure 6 [Left], there is a light reflex that is bouncing off of the artery.

The normal width is $\frac{1}{5}$ or maybe even $\frac{1}{4}$ of the width of the blood vessel. Anything that is wider than that is abnormal; any widening or increase in brightness. If you do notice a light reflex, it is probably abnormal at that point. Sometimes the ALR can span the entire width of the blood column, and that is very severe in the stage of hypertensive retinopathy, and at that point you can start calling it “copper wiring” or “silver wiring”.

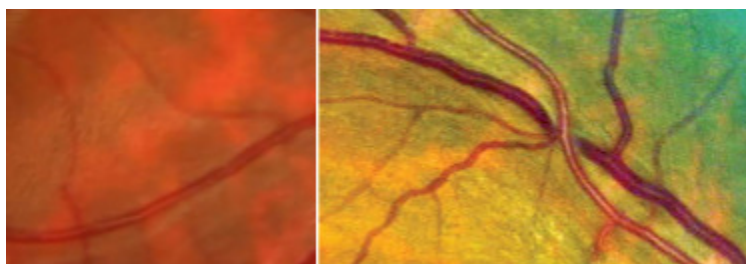


Figure 6: Two examples of an increased arteriolar light reflex, indicating arteriolar attenuation.

Arteriovenous Crossing Changes

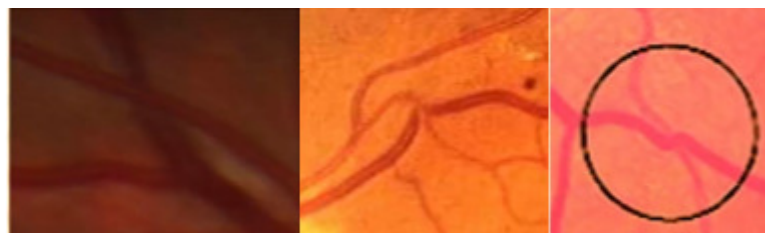


Figure 7 [Left] Generalized nicking. [Center] Right-angle deflection. [Right] S-shaped deflection. All caused by hardening of the arteries due to damage from hypertension.

Arteriovenous (A/V) crossing changes are basically crossing changes from arteries crossing over the veins. It can manifest in any way, shape or form, including generalized nicking, tapering, compression, S-shaped bends, right angle deflections or banking of a venule. First of all, it's very important to understand that the

arteries cross over the veins. In high blood pressure, chronically, the arteries are getting damaged, so the walls of the arteries are stiffening up and, as a result, it causes the artery to push down on the vein. Normally the arteries are nice and malleable, and crossing over the veins are no big deal. Once it stiffens up, however, it pushes down on the vein, and can cause these S-shaped deflections, as you can see in Figure 7 [Right], so the vein just looks like it is being compressed. Figure 7 [Center] shows the

artery crossing the vein and the vein is getting compressed and tapering a little bit. Think of the vein as a very malleable garden hose, and if you have an artery that is like a stiff pole made out of metal, and you are just pushing down on the hose. That is essentially what is happening when you have these A/V crossing changes.

Retinal Hemorrhages



Figure 8: Flame hemorrhages.

With retinal hemorrhages, patients can present with this as a sign of hypertensive retinopathy, but when you see these, you need to start thinking about other co-morbidities. Co-morbidities must also be considered with the remaining signs I am going to discuss today.

Retinal hemorrhages are most likely going to show up in hypertensive retinopathy as flame hemorrhages. Sometimes you might get dot-blot hemorrhages, but if you have too many dot-blot hemes, you need to start thinking of a different co-morbidity that

could be causing those. We will discuss that more later on in the lecture.

Cotton Wool Spots

Cotton Wool Spots (CWS) are something else that can show up with hypertensive retinopathy, and this indicates acute inner retinal ischemia. Because the inner retina is ischemic, it fluffs up a little bit and looks like a cute cotton wool spot in the back of your eye. (Fig 9 Top)

Exudates

Exudates certainly can happen. They are a little bit more rare, but they can happen. Exudates are basically lipids that have deposited in the outer plexiform layer. A lot of times, it is associated with edema. (Fig 9 Middle Top)

Vessel Sheathing

With time, chronicity, and high blood pressure damaging the vessel walls, a lot of times there is glial proliferation that happens along the vessel walls in response to the vascular damage. (Fig 9 Middle Bottom)

Optic Nerve Swelling

Optic nerve head swelling is not that common with hypertensive retinopathy. Optic nerve head swelling is not seen in your typical run-of-the-mill cases; instead, it is seen with accelerated or malignant hypertension. This indicates that something has acutely spiked the blood pressure, and has spiked it pretty high – enough to cause the optic nerve head to swell. This is nothing typical you would see in the normal cases, but you should know that this can appear in rare cases.



Figure 9

Possible complications

There are definitely a lot of complications that can happen with hypertension and hypertensive retinopathy. If you leave the patient untreated systemically, the hypertension gets out of control, and they develop hypertensive retinopathy, they can suffer from any of the complications listed in Table 6.

The vein occlusions happen because, of course, the arteries are getting more stiff, compressing the veins, and once you pass a certain point and compress too much of the vein, you may end up with a vein occlusion in any form; central or branch.

Retinal artery occlusions also make sense because with the stenosis of the artery, and the artery becoming more and more attenuated, it can get to a certain point where the artery is occluded, and at that point you either have a CRAO or a BRAO.

Retinal artery macroaneurysms certainly can happen. You are getting a lot of retinal vascular changes. Anterior ischemic optic neuropathy (NAION) can also happen because you are getting a lot of vascular changes, which can easily cause ischemia to the optic nerve. Any of the cranial nerve palsies, again, can be another possible complication as we expect a lot of vascular changes that can lead to ischemia of the nerves, leading to a palsy. A choroidal infarction, reducing the blood supply going to your choroid, which is very critical to allow you to continue to supply the choroid with blood. When we have the vessel changes, it can cause choroidal damage, too.

Something interesting to note is that if the patient does have diabetes, and their hypertension is out of control causing hypertensive retinopathy, their diabetic retinopathy can also be adversely affected. If your hypertension is out of control, it will cause your diabetes to be out of control. It is a very interesting dynamic, and a lot of these patients that have hypertension also have diabetes. That is something to be aware about.

Our last listed complication is glaucoma. Again, vessel changes can cause damage to the nerve.

There is a hypertensive retinopathy grading system that was developed by Keith-Wagner-Barker (KWB). This grading system is commonly used in the literature. It includes Grade 1 through 4. Grade 1 is very mild, generalized arteriolar narrowing and an A/V ratio of

Table 6

Possible Complications
Retinal Vein Occlusion (CRVO, BRVO)
Retinal Artery Occlusion (CRAO, BRAO)
Retinal Artery Macroaneurysm
Anterior Ischemic Optic Neuropathy (NAION)
Cranial Nerve Palsies
Choroidal Infarction
Adversely Affect Diabetic Retinopathy
Glaucoma

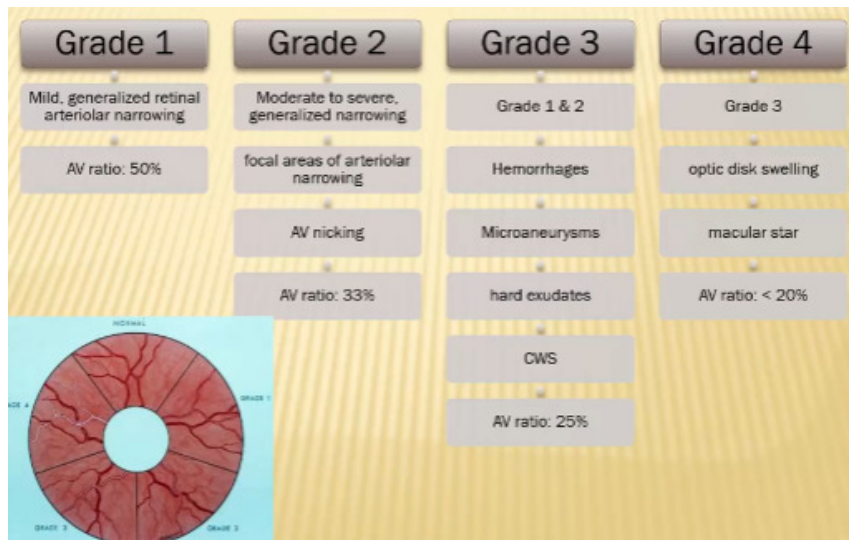


Figure 10: KWB Hypertensive Retinopathy Complications Grading Scale

50% or so. Grade 2 moves onto moderate or severe generalized narrowing, with possible focal areas of narrowing. You may find A/V nicking, in addition. An A/V ratio may be 33% now. Grade 3 is everything you see listed in Grade 1 & 2, and also including hemorrhages, microaneurysms, exudates, CWS and an A/V ratio of 25%, which is a little bit worse. Once you go on to Grade 4, it includes everything in Grade 3 and also optic disc swelling, and/or a macular scar, meaning the optic nerve or the macula now may be involved. Your A/V ratio is now less than 20% in these patients.

In general, the KWB grading of hypertensive retinopathy scale, Grades 1 and 2 are typically chronic cases, your run-of-the-mill chronic hypertensive cases. Grades 3 and 4 are typically more acute cases and definitely include more cases of the malignant hypertension or some sort of accelerated hypertension that has caused so much of the more wet retinopathy, and maybe optic nerve head swelling or macular edema. Grade 3 tends to have a diastolic blood pressure that is greater than or equal to 110 mmHg. Anybody with a diastolic blood pressure greater than or equal to 130 mmHg correlates to Grade 4. It is important to pay attention to the diastolic blood pressures in these cases, as it correlates more to the grading system that has been established by KWB.

As I mentioned before, the KWB hypertensive retinopathy scale is widely cited. However, there has been some criticism with the KWB scale because it is not adequately sensitive to differentiate between Grade 1 and Grade 2 in clinical practice, so it is pretty hard to use, and really, Grade 1 and Grade 2 are most of the hypertensive retinopathy cases that you are going to see, as more of the run-of-the-mill chronic hypertensive cases.

Figure 11: Mitchell-Wong Classification of Hypertensive Retinopathy (2004)

In addition, the lower grades of retinopathy have not been closely shown to correlate to blood pressure levels as well as validated markers of target organ damage. Because of that, in 2004, Mitchell and Wong developed another classification system (Figure 11) for hypertensive retinopathy. Instead of dividing it into 4 categories, they divided it into 3 categories of mild, moderate and accelerated.

Retinopathy	Description	Systemic associations
Mild	One or more of the following signs: generalized arteriolar narrowing, focal arteriolar narrowing, AV nicking, arteriolar wall opacity (silver-wiring)	Weak association with stroke, coronary heart disease and cardiovascular mortality
Moderate	Mild retinopathy with one or more of the following signs: retinal hemes, microaneurysms, CWS, hard exudates	Strong association with stroke, congestive heart failure, renal dysfunction and cardiovascular mortality
Accelerated	Moderate retinopathy signs plus optic disk swelling; may be associated with visual loss	Associated with mortality and renal failure

Mild is essentially defined as “One or more of the following signs: generalized arteriolar narrowing, focal arteriolar narrowing, AV nicking, arteriolar wall opacity (silver or copper wiring).” If you see any of those 3 signs that are more specific to hypertensive retinopathy, those vascular or vessel changes, then we are in the mild category. There are very weak association with stroke or coronary artery disease or cardiovascular mortality at that point.

If you are in the more moderate retinopathy category, which is defined as “mild retinopathy with one or more of the following signs: retinal hemes, microaneurysms, CWS, or hard exudates.” This is where we start to get a little more involvement outside of the retinal vasculature changes, and we get a little more

retinopathy showing up. Moderate cases have a strong association with stroke, congestive heart failure, renal dysfunction and cardiovascular mortality.

The last grade is accelerated, which is where we start to see moderate retinopathy signs plus optic disc swelling, and it may be associated with visual loss. The systemic associations with that are very much associated with mortality and renal failure. Ever since 2004, when Mitchell-Wong came up with this classification, it has picked up more and more, with more people using this category. There are still people who still use the KWB, and they go hand-in-hand, but just noting what type of retinopathy you see is very helpful, whether you use a classification system or not.

Differential Diagnosis

It is important to differentiate whether those retinopathy signs you are seeing are actually hypertensive retinopathy or if they might lead you to another co-morbidity. As I mentioned before, a lot of times these hypertensive patients are also diabetic, so it is important to know if these changes are also diabetic, or if it's hypertensive. I've already gone over the 3 signs that are a little more specific for hypertensive retinopathy: the attenuation of the arterials, the arterial light reflex, and the A/V crossing changes. If your patient has a little bit of those, but also exhibits signs of diabetic retinopathy, (some things to look for include more hemorrhages which are usually dot-blot in fashion rather than flame hemes that you would see in hypertensive retinopathy; the diabetic retinopathy cases also have more microaneurysms, and as I mentioned before, they have less of the vessel attenuation) it is important to differentiate those there.

Some differential diagnosis is to differentiate from any type of a vein occlusion, whether it be a CRVO, where the entire retina is involved, or a BRVO where just one branch is involved, or a hemi, which is where half of the retina is involved. Some ways to differentiate it is that most of these vein occlusions are going to be unilateral – it is very rare for vein occlusions to be bilateral. It can happen, but it is super-rare. If you have a vein occlusion, you are looking at multiple, as in “tons and tons” of hemorrhages. You will most likely have venous dilation and tortuosity because the vein is occluded. When you are looking at the arteries, there will be no arteriolar narrowing, which makes the arteriolar narrowing all the more important to look for in regards to hypertensive retinopathy.



Figure 12: Retinopathy in a patient with collagen-vascular disease

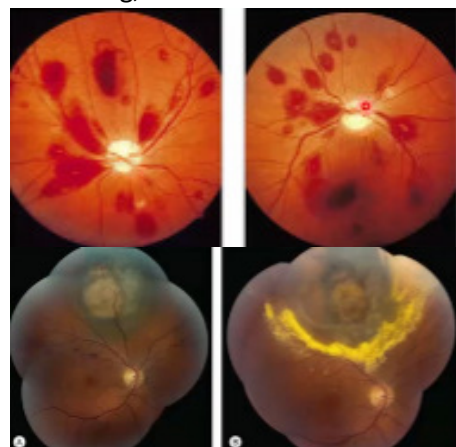


Figure 13 [Top] Anemic Retinopathy [Bottom Left] Radiation to the eye. [Bottom Right] The same area a few years later with radiation retinopathy.

More differential diagnosis include collagen-vascular disease. With collagen-vascular disease, you are going to get multiple cotton wool spots. In Figure 12, you see that it is mainly cotton wool spots that are presenting, and you are not seeing as much of the arteriolar attenuation.

Anemic retinopathy is more likely to give us more hemorrhages, again, with no arteriolar changes. (Fig 13 top) Radiation retinopathy, if your patient has a history of radiation adjacent to the eye (Fig 13 bottom left), you can see the area of the eye that had radiation, and, as a result, a few years later (Fig 13 bottom right) you can get a presentation that looks somewhat like this. However, again, you will not get that arteriolar attenuation.

Most commonly, it's hard for clinicians to differentiate between diabetes and hypertension because, like I said, most of the time these patients have both co-morbidities and they come hand-in-hand. So how can you tell between the two? I've written out in Table a few of the differences between the two.

Hypertension in general is more of a dry retina, and diabetes is more of a wet retina.

What I mean by that is with hypertension, you get fewer hemorrhages. Edema is more rare, as are exudates, in the dry retina (hypertension) than the wet retina (diabetes). Exudates and edema go hand-in-hand. CWS are not really considered "wet", it's just that the inner retinal layer is ischemic, thus fluffed up a little bit – that's not really "wet", so it can occur in hypertensive retinopathy. Flame-shaped hemes, if you get any hemorrhages, are going to show up as flame-shaped as opposed to dot-blot shaped. Again, with hypertension, you will get abnormal arteriolar changes – attenuation, light reflex and A/V crossing changes.

In contrast, with Diabetes, it is more of a "wet" retinopathy, so things are just leaky in the retina. We have multiple hemorrhages, extensive edema, multiple exudates. You get fewer CWS. It is more rare to get flame-shaped hemorrhages – you can get them, but they are less common than dot-blot hemes. A lot of times, with diabetes, the retinal veins and capillaries are more abnormal than the arteries.

This is not to say that it is always clear-cut and dry, if you see dry retinopathy it is hypertension compared to if it is wet, it is always diabetes. Sometimes a patient has both of these conditions, and when you see retinopathy in the back of the eye, you can have both hypertension and diabetes working in the back of the eyes at the same time. Thus, it is not out of the question that a patient could have both hypertensive and diabetic retinopathy. At least, looking for what type of signs you see in the back of the eyes will help you differentiate between the two, but keep in mind that both can present at the same time. Sometimes what also might help is just asking the patient themselves how they are controlling their diabetes and how well they are controlling their hypertension; finding out which is more out of control. That can lead you more towards the answer as to if it is more of a hypertensive or diabetic retinopathy. And, as I said, sometimes it is just both.

Work-Up

What is a work-up on these patients? Taking a very good history, asking about any systemic conditions. Particularly ask about hypertension – has anybody ever told them they had hypertension, are they being treated for hypertension, is it well-controlled? Have they ever had a high blood pressure reading, things of that nature. Asking about diabetes, again, has anybody ever checked their blood sugar level? Have they ever been told they have high blood sugar? Do they have a history of adnexal radiation, as well, too. After taking a good history, we need to do a good eye exam, including a dilated fundus exam, making sure you really scrutinize the back of the eye, all the vessel vasculature, and looking at everything in the retina to make sure it all looks healthy.

Table 7

Hypertension vs	Diabetes
Dry retina	Wet retina
Few hemorrhages	Multiple hemorrhages
Rare edema	Extensive edema
Rare exudates	Multiple exudates
Multiple CWS	Few CWS
Flame-shaped hemes	Rare flame-shaped hemes
Visibly abnormal retinal arteries	Visibly abnormal retinal veins and capillaries

Checking blood pressure – I cannot emphasize how important this is, to check every patient's blood pressure that comes in. And not just checking their blood pressure, you need to make sure that you measure it accurately by using the correct cuff size. For a pediatric patient, you have to use a pediatric cuff size. For an adult patient you need an adult size. There are also extra-large cuff sizes for your overweight or obese patients. Ensuring that you use the correct cuff size and correct placement of the cuff will help ensure that you get a good reading. It is good to now allow the patient to smoke or consume caffeine right before the blood pressure reading, because it can affect the blood pressure reading, but that is not always preventable. It is, however, good to ask if they've smoked or consumed caffeine right before they came into your office.

The following workup is definitely optional, but if you see a very bad/severe case, maybe even an accelerated case of hypertensive retinopathy, of course you should refer to their PCP, but you can also refer to ophthalmology for a fluorescein angiogram (FA). An FA can help check for more arteriolar narrowing or straightening that is not particularly easy to see with just a DFE. It can help you see more MA's, capillary non-perfusion, and maybe a very subtle case of macular edema, as well. This is definitely optional, and maybe just for the more severe cases that you would refer to ophthalmology for.

Of course, we need to refer our patients appropriately. The patient really needs a medical consultation if they have an unremarkable systemic history. If they've never known that they have high blood pressure, and you check their blood pressure, it is elevated and they have hypertensive retinopathy, you need to refer them to a primary care doctor so they get treated and it gets caught early. You can truly save lives if you are the first person to see this, or the first person to mention it to them. Especially refer if the patient is visually symptomatic – if they have blurred vision, or transient dimming of vision like a TIA or Amaurosis Fugax. Why would they have blurred vision or the transient dimming of vision? It is because they are probably in that Grade 4 or Accelerated grade of hypertensive retinopathy where their macula is involved, with macular edema or they have optic nerve head swelling due to those accelerated cases. If patients come in, and they do have those accelerated complaints that are definitely due to hypertension, that is a very severe case and they need to be referred immediately. Especially if your patient has a diastolic pressure over 110 to 120 mmHg, refer immediately! Call an ambulance if you have to, call them a cab to drive them to the ER, because these are the patients who are truly at high risk for developing a stroke or having a heart attack, and you do not want to be the last person to see them, and send them off doing nothing about that, then they have a stroke or heart attack on their way home, or as they are driving to the ER, causing a major accident. That will not only be on your conscience, but will also go back on you medico-legally. That is something to think about and pay attention to.

If a blood pressure is less than or equal to 179/109, it is a non-urgent referral.

If you have blood

pressure that is 180/110 to 209/119, that is a more urgent referral. Anything that is above 210/120 is a medical crisis and warrants an immediate referral. These are just general guidelines, but use your clinical judgement in terms of when you are going to immediately refer. Remember that a lot of these patients are asymptomatic, and they are going to come in with no symptoms systemically or visually. It is so important to convey the seriousness of the condition to the patient, because if you do not, they

+ BP \leq 179/109: non-urgent referral

+ BP 180/110 - 209/119: more urgent referral

+ BP $>$ 210/ $>$ 120: medical crisis, immediate referral

might not think it is that serious of an issue, and they may not go to their PCP, or they might not seek the medical care that they need to get treatment.

Treatment/Management

When you refer the patient out and they get treated, you are mainly co-managing with a primary care physician (PCP) to control the underlying hypertension. Suggesting lifestyle changes to the patient, including weight reduction, exercising more, decreasing their salt and cholesterol intake, relaxing more – in the case of my patient the attorney, having them retire so they are stress-free now or undertaking stress management courses – smoking cessation, all of those are very helpful in controlling and managing the underlying hypertension. Of course, with the PCP actually prescribing hypertension medications to help control the hypertension. In general, if the hypertension is well controlled, then the prognosis is usually very, very good.

HTN meds:

× Diuretics, beta-blockers, ACE inhibitors, Angiotensin-receptor blockers, Calcium-channel blockers, vasodilators

Follow-Up

If it is a more severe case of hypertension, you may want to follow-up every two to three months at first, then every 6-12 months as you get more comfortable with the patient and their hypertension is more under control. If it is a mild or more moderate case, following up every 3-6 months or 6-12 months is appropriate, as you see fit. These are just general guidelines, so use your clinical judgment in terms of how well the patient is controlling their hypertension and how comfortable you are with releasing them to a longer follow-up.

More Statistics

I want to talk a little bit about an interesting paper that came out in 2005 looking at hypertension and hypertensive retinopathy as indicators of cardiovascular morbidity and mortality. In this paper, the statistics that they found are very interesting. They found with Grade 1 hypertensive retinopathy, these patients have a 70% survival rate in the next 3 years. That's a pretty good survival rate, but still, 30% of these patients do not have a survival rate. If you find Grade 4 hypertensive retinopathy, these patients only have a 6% survival rate in the next 3 years, which is a very alarming drop in the survival rate from Grade 1 to Grade 4.

If you find any hemorrhages, microaneurysms or CWS in the fundus, those patients are two to four times more likely to develop an incident of clinical stroke within three years, even with control of blood pressure, lipids, smoking, etc. They are two times more likely, as well, to develop congestive heart failure (CHF), and a three-fold increased risk of heart failure events. If in addition to the hemorrhages, aneurysms and CWS, you also find A/V nicking, these patients are more likely to develop renal dysfunction.

More indications that they found with this paper include if you find arteriolar narrowing but the blood pressure of the patient is normal at this time, then this patient is 60% more likely to be diagnosed with hypertension within the next three years. This is really big – this means that sometimes their blood pressure may be normal, but you are noticing arteriolar narrowing, which is the first indication that they may actually be moving towards hypertension. Really, as an eyecare provider, we can be the first people that see the signs of hypertension – even three years before hypertension shows up

systemically! That is HUGE!! In addition, if you see arteriolar narrowing, and blood pressure is normal, it helps predict the incidence of Type II diabetes. This patient may develop diabetes as well, too.

Any retinal microaneurysms or hemorrhages that you see in a patient, that patient is two times more likely to die from a cardiovascular event. Treatment of any microcirculation in hypertensive patients may further reduce cardiovascular morbidity and mortality. We need to be very diligent in looking for these signs of hypertensive retinopathy – you could potentially save lives and be the first person to detect hypertension in these patients.

Summary

In summary, patients are going to walk into your office asymptomatic if they have hypertension, even if they have hypertensive retinopathy. There will be no visual systems. It is important that we be diligent in checking blood pressure and looking for signs of hypertensive retinopathy. Not just checking the blood pressure – after we check the blood pressure, it is important to know when to refer immediately and get these patients in for the care that they need.

Also, know the retinal signs of hypertension. The three that are the most common and specific to hypertension, I would emphasize:

1. Arteriolar attenuation
2. Increased ALR
3. AV crossing changes

Really look for those as the first presenting signs of hypertension.

It is so important to co-manage with a PCP for hypertension. If these patients are managed appropriately with early diagnosis, they have great prognosis. Really, having good communication with their PCP is key.

Thank you so much for your attention and for taking this online CE course. I watch my email, listed below, if you have any questions, comments or concerns. Please feel free to email me. Below is the list of my references.

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