Systemic Hypertension and the Eyes Course # 40027

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COURSE DESCRIPTION:

Hypertension is the most common systemic disease for office visits to clinicians. Optometrists play an important role in diagnosing and managing various ocular complications secondary to hypertension. This course focuses to enhance that role.

LEARNING OBJECTIVES:

- Seek interdisciplinary collaboration for optimal management of hypertension
- Classify hypertension based on severity
- Know the prevalence of hypertension in the United States
- Recognize the stages of hypertensive retinopathy
- Identify ocular complications secondary to hypertension
- Provide effective patient education on the ocular effects of hypertension
Good day fellow doctors, thanks for your interest in hypertension and the eyes. The focus of this discussion will be on the effects of hypertension on the eyes and our important roles in managing this common health issue in public health.

Let’s start a short overview of hypertension. It is an indisputable fact that hypertension is the most important health problem with its major complications such as coronary heart disease, stroke, chronic kidney disease, congestive heart failure, and retinopathy.

The treatment of hypertension is the most common reason for office visits of non-pregnant adults to clinicians in the United States and for use of prescription drugs. The national health and nutrition examination survey (NHANES) conducted from 2005 through 2008 estimated that approximately 29 to 31% of adults in the United States have hypertension. (Fig 1) This translates into 58 to 65 million hypertensives in the adult population in the United States. The number of patients with hypertension is likely to grow as the population ages since either isolated systolic hypertension or combined systolic and diastolic hypertension occurs in the majority of persons older than 65 years. The rising incidence of obesity will also increase the number of hypertensive individuals.

### Table 1: Definitions of Hypertensive Stages

<table>
<thead>
<tr>
<th>Classification</th>
<th>SBP</th>
<th>DBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>And &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>Or 80-89</td>
</tr>
<tr>
<td>Stage 1</td>
<td>140-159</td>
<td>Or 90-99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>&gt;160</td>
<td>Or &gt;100</td>
</tr>
<tr>
<td>Severe</td>
<td>&gt;180</td>
<td>&gt;120</td>
</tr>
</tbody>
</table>

The definitions in Table 1 were suggested in 2003 by the seventh report of the Joint National Committee (JNC 7) and reaffirmed in 2013 by JNC 8, and are based upon the average of two or more properly measured readings at each of two or more visits after an initial screening.

- Normal blood pressure: systolic <120 mmHg and diastolic <80 mmHg
- Prehypertension: systolic 120 to 139 mmHg or diastolic 80 to 89 mmHg
- Hypertension:
  - Stage 1: systolic 140 to 159 mmHg or diastolic 90 to 99 mmHg
  - Stage 2: systolic ≥160 or diastolic ≥100 mmHg
  - Severe: systolic >180 or diastolic >120 mmHg

Isolated systolic hypertension is considered to be present when the blood pressure is ≥140/<90 mmHg and isolated diastolic hypertension is considered to be present when the blood pressure is <140/≥90 mmHg. Clinical significance of blood pressure readings appears age dependent. Over age 50 years, systolic blood pressure >140 mmHg predicts mortality regardless of diastolic readings. Under age 50 years, diastolic blood pressure is a better predictor of mortality than systolic readings.

**Hypertensive urgency**—Severe hypertension (as defined by a diastolic blood pressure above 120 mmHg) in asymptomatic patients is referred to as hypertensive urgency. There is no proven benefit from rapid reduction in blood pressure in asymptomatic patients who have no evidence of acute end-organ damage and are at little short-term risk.
As primary eye care providers, we do take BP during a comprehensive eye exam. Some patients have not been diagnosed with HTN, but their pressures are high during the visit, so we attribute it to white coat HTN. Actually, approximately 20 to 25 percent of patients with stage 1 office hypertension have "white coat" or isolated office hypertension in that their blood pressure is repeatedly normal when measured at home, at work, or by ambulatory blood pressure monitoring. This problem is more common in the elderly. One way to minimize the white coat effect is to have the blood pressure measured while seated after five minutes in a quiet, unobserved setting by an automated device that obtains five repeated blood pressure measurements at one- to five-minute intervals. Also, ambulatory blood pressure monitoring (ABPM), which records the blood pressure at preset intervals (usually every 15 to 20 minutes during the day and every 30 to 60 minutes during sleep), can be used to confirm or exclude the presence of white coat hypertension in patients with persistent office hypertension but normal blood pressure readings out of the office.

The target BP is similar to our concept of target IOP for an effective management is <140/90 mmHg, but for patients with comorbidity like diabetes or renal disease the target is lower to <130/80 mmHg. It has been shown that if the BP is under optimal control, the incidence of stroke, myocardial infarction, and heart failure are reduced significantly ranging from 25% to 50%. For instance, the incidence heart failure is reduced by half.

In the absence of a specific indication, there are three main classes of drugs that are used for initial monotherapy: thiazide diuretics, long-acting calcium channel blockers (most often a dihydropyridine, amlodipine, Norvasc), and ACE inhibitors (Lisinopril, Zestril) or angiotensin II receptor blockers (Lorsartan, Cozaar). It is the attained blood pressure, not the specific drug(s) used, which is the major determinant of outcome. In a 2012 study from University of Istanbul, Turkey, the number of antihypertensive drugs used by the patients varied from 0 to 5 with the average of 1.71 ± 1.01 drugs. About 10.1% of the patients (n = 66) have not used any drugs, whereas 33.9% (n = 222) have been using one drug; 35.6% (n = 233) a combination of two drugs, 16.5% (n = 108) three drugs, 3.4% (n = 22) four drugs, and 0.6% (n = 4) five drugs. β-Blockers (metoprolol, Lopressor) were the most commonly used antihypertensive drugs in patients with hypertensive retinopathy (P = 0.025).

Usually patients are not just using medical therapy alone. All patients diagnosed with hypertension should undergo appropriate non-pharmacologic (lifestyle) modification, such as weight reduction aiming for BMI between 18 to 25, eat a diet rich in fruits, vegies and low fat products, reduce intake of salt to less than 6 g, or about 1 teaspoon of salt per day, exercise >30min/d most days of the week, and about a drink of alcohol a day.
Despite the prevalence of hypertension and its associated complications, control of the disease is far from adequate. Data from the 2005-2008 NHANES survey shows that only 46 to 51 percent of persons with hypertension have their blood pressure under control, defined as a level below 140/90 mmHg. There are numerous potential reasons for low rates of blood pressure control, including poor access to health care and medications and lack of adherence with long-term therapy for a condition that is usually asymptomatic. We can relate to this with glaucoma patients, who may not be compliant because they are asymptomatic and when they take their medication, they do not get the immediate relief or reward from spending the money on their medication. The latter may be particularly true when the therapy may interfere with the patient’s quality of life and when its immediate benefits may not be obvious to the patient. Thus, hypertension will likely remain the most common risk factor for heart attack and stroke.

Hypertension is quantitatively the major risk factor for premature cardiovascular disease; being more common than cigarette smoking, dyslipidemia, or diabetes, the other major risk factors. The risk of heart failure increases with the degree of blood pressure elevation. Left ventricular hypertrophy is a common finding in patients with hypertension, and is associated with an enhanced incidence of heart failure, ventricular arrhythmias, death following myocardial infarction, and sudden cardiac death. Hypertension is also the most common and most important risk factor for ischemic stroke, the incidence of which can be markedly reduced by effective antihypertensive therapy. Hypertension is the most important risk factor for the development of intracerebral hemorrhage. Hypertension is a risk factor for chronic kidney disease and end-stage renal disease. It can both directly cause kidney disease, called hypertensive nephrosclerosis, and accelerate the progression of a variety of underlying renal diseases. Marked elevations in blood pressure, as in the case of malignant hypertension or severe hypertension, can be an acute, life-threatening emergency.

A number of ocular abnormalities are directly or indirectly associated with hypertension. These include some that are a direct consequence of elevated blood pressure, including hypertensive retinopathy, choroidopathy, and optic neuropathy. With other abnormalities, hypertension is a significant risk factor, including retinal vein and artery occlusion, retinal artery emboli, and diabetic retinopathy. In addition,
hypertension may accelerate nonvascular eye disease, including age-related macular degeneration and glaucoma.

Now that we have an overview of hypertension, let’s move into details discussing about ocular complications of hypertension.

The underlying pathophysiology of these signs can be divided into stages. The initial response of the retinal circulation to a rise in blood pressure is vasospasm and an increase in vasomotor tone, which is seen clinically as generalized retinal–arteriolar narrowing. Subsequently, chronic arteriosclerotic changes, such as intimal thickening, media-wall hyperplasia, and hyaline degeneration, develop. These changes manifest as diffuse and focal areas of arteriolar narrowing, opacification of arteriolar walls (described as silver or copper wiring), and compression of the venules by arterioles at their common adventitial locations (termed arteriovenous nipping or nicking).

With more pronounced high blood pressure, the blood–retinal barrier breaks down, resulting in exudation of blood (hemorrhages), lipids (hard exudates), and subsequent ischemia of nerve-fiber layers (known as cotton-wool spots).

In the setting of severely high blood pressure, raised intracranial pressure and concomitant optic nerve ischemia can lead to disc swelling (papilloedema), which is sometimes referred to as severe or malignant hypertension or hypertensive optic neuropathy.

Arteriovenous (AV) nicking is seen in Figure 6 with the retinal artery located anterior to the retinal vein at the AV crossings. In the study from 2013 (Table 3), there were 126 occurrences of AV nicking. In these instances, the artery was located anterior to the vein in 96.8% (122/126) of crossings (p<0.001). In the control group, there were four occurrences of AV nicking, all of which were artery anterior. The overall incidence of AV nicking among artery anterior to vein crossings was (122/283 =) 43%, considerably higher than the (4/109=) 4% incidence of AV nicking among artery over vein crossings in the control group (p=<0.001).

Most of the time when we are evaluating hypertensive retinopathy, we only focus on AV nicking. However, this recent study shows that arching of the retinal vein (Fig 7) occurs when the vein is on top of the artery. In the study group, there were 43 occasions of venous arching or cascading of retinal veins over thickened arteries and arterioles.
In these instances, the vein was positioned anterior to the artery in 41 of 43 (95.3%) crossings (p<0.001). There were eight cases of venous arching in the control group, all of which were in the setting of vein anterior anatomy. The overall incidence of venous arching among veins anterior to artery crossings was (41/147) 28% in the study cohort, similar to the (8/31) 26% incidence of venous arching in the younger control cohort of 20 patients without a history of systemic arterial hypertension (p=0.86). So arching of the retinal vein is another interesting retinal sign to look for, but it is not as diagnostic as AV nicking.

The most common ocular diseases directly related to hypertension are progressively increasing retinal microvascular changes, which are known as "hypertensive retinopathy."

Population-based studies that used retinal photographs and standardized assessment methods to define signs of retinopathy detected signs of hypertensive retinopathy in 2–14% of the non-diabetic population aged 40 years and older.

Classically, the features are divided into four degrees and their morphological classification has been widely used. However, a more pathophysiological division has been proposed and seems more logical. This three-degree classification includes mild, moderate, and severe:

**Mild** – Retinal arteriolar narrowing related to vasospasm, arteriolar wall thickening or opacification, and arteriovenous nicking, referred to as nipping.

**Moderate** – Hemorrhages, either flame or dot-shaped, cotton-wool spots, hard exudates, and microaneurysms.

**Severe** – Some or all of the above, plus optic disc edema. The presence of papilledema mandates rapid lowering of the blood pressure.

Fundoscopy should be part of the physical examination on every patient with newly diagnosed hypertension since the retina is the only part of the vasculature that can be visualized non-invasively. Pupillary dilatation with a short-acting mydriatic (ie tropicamide 1%) is almost always useful since the mild changes are hard to quantify,
even with retinal photography. The key message here is that we need to dilate hypertensive patients and look for key signs of early hypertensive retinopathy.

Figure 8 shows some examples of each stage of hypertensive retinopathy for your references when you examine your patients with hypertension. Panel A shows arteriovenous nicking (black arrow) and focal narrowing (white arrow). Panel B shows arteriovenous nicking (black arrows) and widening or accentuation (“copper wiring”) of the central light reflex of the arterioles (white arrows).

In a study performed in 2001 on 800 hypertensive patients, the prevalence of grade mild retinopathies among hypertensive patients was 46%.

In Figure 9, we have examples of moderate hypertensive retinopathy. Panel A shows retinal hemorrhages (black arrows) and a cotton-wool spot (white arrow). Panel B shows cotton-wool spots (white arrows) and arteriovenous nicking (black arrows). In the same study in 2001 on 800 hypertensive patients, the prevalence of moderate hypertensive retinopathies was 32%.

Figure 10 is an example of malignant hypertensive retinopathy. Remember that, in addition to all of the earlier signs, malignant hypertensive retinopathy needs to have the swelling of the optic nerve. In Fig 10, we see multiple cotton-wool spots (white arrows), retinal hemorrhages (black arrows), and swelling of the optic disk are visible. In the same study in 2001 on 800 hypertensive patients, only a few patients (<2%) showed grade 3 and grade 4 abnormalities (2001).

In summary, almost half of the patients in the study have mild or moderate hypertensive retinopathy, but only a few percent of the patients have severe hypertensive retinopathy. Also, it must always be taken into consideration when evaluating retinal changes, as in adult patients, diabetes and hypertension usually coexist in most of the cases. Typical early retinopathy signs of diabetes and hypertension share many morphological and pathophysiological similarities. Thus, when you examine a patient with diabetes and hypertension, you want to keep in mind that some of the early retinopathy signs in diabetes and hypertension share a similar appearance, which can make it more challenging in terms of knowing which ones belong to diabetes and which ones belong to hypertension. However, the management is similar because you want to take care of both hypertension and diabetes.
Hypertensive retinopathy has long been regarded as a marker of systemic vascular disease elsewhere in the body. The hypothesis of a link between hypertensive retinopathy and stroke has been the most consistent, and has been supported by anatomical, physiological, and pathological studies. In a 3-year population-based cohort study of atherosclerosis risk, incident stroke events were more common in participants with signs of hypertensive retinopathy than in participants without retinopathy. In an analysis that controlled for blood pressure, diabetes, lipids, and other risk factors, moderate signs of hypertensive retinopathy, such as cotton-wool spots, retinal hemorrhages, and microaneurysms, were associated with a two-fold to four-fold higher risk of incident stroke. Weaker associations between signs of mild hypertensive retinopathy and risk of stroke were also seen. This study and others have now linked signs of hypertensive retinopathy with cognitive decline, cerebral white-matter lesions identified by cerebral MRI, lacunar infarctions, cerebral atrophy, and stroke mortality. Table 4 shows that cotton wool spots actually have the highest risk, in terms of association with the 3 year cumulative incidence of stroke. Pay attention to those moderate hypertensive retinopathy signs, and manage the patient more closely, accordingly.

Various national guidelines for management of hypertension recommend assessment of retinopathy to enable risk stratification. Patients with mild retinopathy will probably only need routine care, whereas patients with moderate signs might benefit from further assessment of blood-pressure control, such as home or 24-hour blood-pressure monitoring, assessment of other vascular risk, such as cholesterol levels, and, if clinically indicated, appropriate risk reduction therapy, for example, using cholesterol-lowering agents.

In patients with borderline or so-called white coat hypertension, physicians could interpret mild or moderate signs of retinopathy as evidence for end-organ damage, and as an indication that antihypertensive therapy could aid in treatment. Additionally, in patients with established hypertension, signs of retinopathy could suggest a need for close observation of blood pressure, supplementary antihypertensive therapy, or both. Patients with severe retinopathy need urgent antihypertensive management.

Retinal–arteriolar narrowing might also be used to predict subsequent development of hypertension in individuals initially classified as normotensive. Thus, retinal–arteriolar narrowing, possibly indicating more widespread peripheral vasoconstriction, could be an early marker of overt hypertension.

Another important thing is that evidence suggests treatment of hypertension could reverse the changes seen with retinopathy. Thus, it is important to detect the early signs of retinopathy and to treat the
hypertension, to possibly reduce the risk of cardiovascular risks, in terms of stroke or heart attack. Laboratory studies in animals and clinical case series have shown regression of retinopathy signs with control of blood pressure. However, whether regression of hypertensive retinopathy is accompanied by a reduction in cardiovascular risk remains uncertain.

Figure 11: BRVO and AV nicking in a patient with hypertensive retinopathy.

Besides retinal microvascular changes or hypertensive retinopathy, hypertension is highly associated with other ocular complications. For example, it is a major cause of retinal macroaneurysms (80%) and ocular ischemic syndrome (70%). 50% of retinal vascular occlusion and 50% of ischemic optic neuropathy are associated with hypertension. Further, hypertension can exacerbate diabetic retinopathy, for example study has shown that for every 10 mmHg increase in systolic BP the risk of diabetic retinopathy is increased by 10% and the risk of proliferative diabetic retinopathy is increased by 15%.

Let’s talk about some of the associated ocular conditions in more detail, starting off with retinal vein occlusion.

Hypertension predisposes patients to development of retinal vein occlusion, a common, sight-threatening retinal–vascular disorder. Retinal vein occlusion is characterized clinically by dilated and tortuous retinal veins and the presence of retinal hemorrhages, cotton-wool spots, and edema of the macula and optic disc.

Central retinal vein occlusion occurs in both ischemic and non-ischemic forms. Patients with an ischemic central retinal vein occlusion typically present with poor visual acuity and a relative afferent papillary defect. These patients have a poorer visual prognosis and are at risk of secondary neovascular glaucoma.

Population-based surveys generally indicate that central retinal vein occlusions arise in 0.1–0.4% and branch retinal vein occlusions in 0.6–1.1% of adults aged 40 years and older. Almost all relevant studies have recorded a strong and consistent link between hypertension and the risk of a retinal vein occlusion. One investigation showed that participants with hypertension were five times more likely to have a branch retinal vein occlusion than those without hypertension. Moreover, mild hypertensive retinopathy was

Figure 12 [A] CRVO and [B] BRVO in two patients with hypertension
strongly correlated with branch retinal vein occlusion, with an odds ratio of 17:1 for focal arteriolar narrowing, and 23:1 for arteriovenous nicking.

**Retinal Artery Occlusion**

Retinal–arteriolar emboli are discrete plaque-like lesions, lodged in the lumen of retinal arterioles. These emboli are heterogeneous, and can be composed of cholesterol crystals (reflective emboli), fibrin, platelets, calcium, or other materials (non-reflective emboli). Retinal emboli can be single or multiple, and can be seen in one or both eyes.

Epidemiological studies report that asymptomatic retinal emboli are fairly common in adults aged 40 years and older. Two large population-based studies have reported prevalence rates of 1.3% and 1.4% and the 10-year incidence of retinal emboli has been recorded as 2.9%. Asymptomatic retinal emboli are often transient; in one study 90% of retinal emboli detected in baseline photographs were not present 5 years later. The main risk factors for retinal emboli are hypertension, diabetes, and cigarette smoking. In Australia, investigators showed that individuals with hypertension had a two-fold higher risk of prevalent and incident retinal emboli than those without hypertension, but that this risk was increased to six-fold higher in hypertensive people who also smoked cigarettes.

Retinal emboli have two important clinical implications. First, the distal portions of occluded arterioles could be ischemic, and thus, could result in frank retinal artery occlusion. Second, people with retinal emboli have a higher risk of thromboembolic stroke and cardiovascular disease.

Retinal artery occlusion occurs commonly in patients with hypertension. Central retinal artery occlusion presents with a sudden, painless, unilateral loss of vision and typically appears as a cherry red spot.

Occlusion of a branch retinal artery, by contrast, could present with a visual-field defect, and loss of central vision can be slight. In up to 70% of cases of branch retinal artery occlusion retinal emboli is visible in the vessels at the optic disc, or downstream in branch retinal arterioles; these signs are present in about 20% of cases when the occlusion arises centrally.

Retinal artery occlusion is associated with hypertension and other cardiovascular risk factors, with hematological
abnormalities, and with both subclinical and clinical stroke. Nearly half the patients with retinal artery occlusion in one study were reported to have echocardiographic abnormalities, and 10% needed systemic treatment. The disorder has been associated with an increased risk of cardiovascular disease and mortality. In a prospective study of 99 patients with retinal artery occlusions followed-up for a mean duration of 4.2 years, the absolute risk of death was estimated at 8% per year; coronary events caused 60% of the deaths, and stroke only 3%.

Mortality rates might also vary due to the presence of retinal emboli; a study of 86 patients with retinal artery occlusions showed that mortality rates for those without visible retinal emboli were similar to age–sex controls, whereas patients with visible emboli had substantially higher mortality than controls. Thorough cardiovascular and cerebrovascular assessments, including analysis of carotid and cardiac images, are necessary for patients who present with retinal artery occlusions. Interestingly, the presence of retinal emboli has low predictive power for detection of significant carotid-artery stenosis, and thus should not affect decisions to do carotid ultrasonography.

**Retinal Macroaneurysm**

Retinal arterial macroaneurysm, a focal dilation of the retinal arterioles, is an uncommon disorder almost always seen in patients with hypertension. In one hypothesis for the cause of retinal macroaneurysm, the retinal–arterial walls become less elastic with aging, as both the medial muscle fibers and intima are gradually replaced by collagen. This decrease in elasticity renders the arterioles susceptible to dilation caused by raised blood pressure. Hypertensive patients, with impaired auto regulation, are at particular risk. Subsequently, loss of the muscular coat, with thinning and fibrosis of arterial walls could lead to dilation, hyperpermeability, and finally rupture of the macroaneurysm. Data from large case series suggest that about a fifth of macroaneurysms are bilateral, and one in ten are multiple. Macroaneurysm is usually an incidental finding in asymptomatic patients, but can also present acutely, with visual loss secondary to hemorrhage or exudation. Hypertension has been reported in up to 75% of patients with macroaneurysms.

**Ischemic Optic Neuropathy**

Like the retinal circulation, optic nerve circulation is prone to the effects of hypertension and other vascular risk factors. Ischemic optic neuropathy is the most frequent acute optic neuropathy in patients aged over 50 years. Either the anterior or the posterior segment of optic nerve can be affected. Anterior ischemic optic neuropathy accounts for 90% of cases, and typically presents with sudden visual loss and optic-disc edema which is typically absent in posterior ischemic optic neuropathy. Anterior ischemic optic neuropathy can be further subdivided into arteritic and non-arteritic types, of which the arteritic form is typically due to giant-cell temporal arteritis, which is not associated with hypertension.
By contrast, non-arteritic anterior ischemic optic neuropathy has been strongly linked with hypertension and other cardiovascular risk factors. One US study showed that the yearly incidence of non-arteritic anterior ischemic optic neuropathy was 10.3 per 100,000 people aged 50 years and older. Clinical series show that up to 50% of patients with non-arteritic anterior ischemic optic neuropathy might have hypertension and 25% might have diabetes.

In a population based study, raised blood pressure is an independent risk factor for both the initial development of retinopathy and its subsequent progression. Impaired retinal–vascular autoregulation in response to high blood pressure plays a part in this association, since diabetic patients with hypertension seem to be less able to regulate retinal blood flow than non-diabetic patients. In diabetes, hypertension can also result in endothelial damage in the retinal vasculature and increased expression of vascular–endothelial growth factors.

Clinical trial data subsequently provided clear and consistent evidence of the role of hypertension in the development and progression of diabetic retinopathy. In a prospective study in the UK, 1,048 patients with hypertension were randomly assigned to a regimen of tight control (aiming for blood-pressure levels below 150/85 mm Hg with atenolol or captopril) or less tight control (blood pressure below 180/105 mm Hg). Investigators noted that participants under tight blood-pressure control, had reductions of 37% in risk of microvascular disease, 34% in rate of progression of retinopathy, and 47% in deterioration of visual acuity. Importantly, the effects of blood pressure control were independent of glycemia. After 6 years of follow-up, the study estimated that each 10 mmHg reduction in systolic blood pressure, the risk of retinopathy might fall by 10%. Longer term follow-up of patients in this study have lent support to the early results.

**Age-Related Macular Degeneration and Glaucoma**

ARMD and glaucoma are ocular diseases where hypertension is a potential risk factor. Some have suggested that hypertension could increase the potential risk factor for age-related macular degeneration, on the basis of its purported effects on the choroidal circulation. An association between hypertension and risk of age related macular degeneration has been noted in both cross-sectional and prospective data, but has not been shown consistently in all studies.

One study, the Beaver Dam Eye Study, reported that raised systolic blood pressure at baseline was not related to prevalent age-related macular degeneration, but did increase the 10-year risk of the disorder. Another study, the Blue Mountains study in Australia, has shown that focal arteriolar narrowing, was associated with the incidence of some signs of age-related macular degeneration.

Systemic hypertension is suspected to increase the risk of the development and progression of glaucoma. Several pathophysiological mechanisms have been proposed to explain this putative association. First, direct microvascular damage from systemic hypertension could impair blood flow to the anterior optic nerve. Second, hypertension could interfere with autoregulation of the posterior
ciliary circulation, which is already impaired in glaucoma. Third, antihypertensive treatment could induce hypotensive episodes, especially at night, which could reduce blood flow to the optic-nerve head, resulting in additional damage to the optic nerve.

**Hypertensive Retinopathy Associated with Preeclampsia**

Last but not least, here is just a reminder to us to pay attention to pregnant patients because of pregnancy-associated hypertension: this case involved a 25-year-old woman at 36 weeks of gestation presented with a 2-week history of headache, light flashes, and blurred vision in both eyes. Her corrected visual acuity was 20/40 in the right eye and 20/50 in the left eye. Funduscopic examination revealed bilateral grade 4 hypertensive retinopathy, with widespread hemorrhages (black arrows), cotton wool spots (white arrows), hard exudates in a star shape (yellow arrows) in the macular region, and swelling of the optic disks (arrowheads). Physical examination revealed a blood pressure of 220/140 mm Hg and pedal edema. A 24-hour urine specimen showed proteinuria (protein, 7.4 g). Severe preeclampsia was diagnosed.

The patient was referred to the obstetrical department and, after initial stabilization, underwent prompt cesarean delivery, which resulted in the birth of a low-birth-weight boy (1900 g); the 1-minute and 5-minute Apgar scores were 8 and 9, respectively. Three months later, the exudates had spontaneously resolved, and the patient slowly regained visual acuity of 20/25 in both eyes.

**Summary**

Most patients with hypertension have primary (essential) hypertension. The pathogenesis of primary hypertension is poorly understood. Numerous risk factors for developing hypertension have been identified, including black race, a history of hypertension in one or both parents, a high sodium intake, excess alcohol intake, excess weight, and physical inactivity.

The most common ocular diseases directly related to hypertension are progressively increasing retinal microvascular changes, which are subsumed under the name "hypertensive retinopathy." The three-degree classification includes mild, moderate, and severe disease. The presence of papilledema mandates rapid lowering of the blood pressure.

Hypertension increases the risk of a number of ocular diseases, with the most common being diabetic retinopathy. Other ocular diseases wherein hypertension serves as a risk factor include retinal venous and arterial occlusion, retinal emboli, retinal macroaneurysm, and anterior ischemic optic neuropathy. The risk for two of the more common causes of vision loss, age-related macular degeneration and glaucoma, may be increased by the presence of systemic hypertension.
We as primary eye care providers play a critical role in managing one the most common public health issues in the US, so do take blood pressure for all patients and look for the effects of hypertension in the eye and manage them timely and appropriately.

Lastly, you can make a handy reference sheet from the information in this presentation for your office use when you see a patient with hypertension.

Thank you very much for your attention.

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**References**