

Hypertensive Choroidopathy: a Teaching Case Report | 1

Abstract

Approximately 1 in 3 adults in the United States has hypertension.²³ Blood pressure is under control in only approximately half of those adults.²⁴ Because the retina is the only place in the body where the vasculature can be observed directly, eyecare providers are uniquely positioned to evaluate the effects of hypertension. This case-based report discusses fundus signs in a patient with accelerated hypertension. A review is given of ocular blood flow, the pathophysiology of fundus signs, diagnostic testing, staging of hypertension and hypertensive retinopathy. The discussion should lead to an understanding of the optometrist's role in the management of patients with elevated blood pressure.

Key Words: hypertensive retinopathy, hypertensive choroidopathy, malignant hypertension, hypertensive crisis

Introduction

Patients with hypertension often present with ocular findings of hypertensive retinopathy. In 1898, Marcus Gunn first described hypertensive retinopathy to include generalized and focal arteriolar narrowing, arteriovenous crossing changes, retinal hemorrhages, cotton wool spots and disc edema. Later, fundus findings in hypertensive choroidopathy would be described to include Siegrist streaks²⁵ and Elschnig spots.²⁶ Three distinct entities have been hypothesized: hypertensive retinopathy, hypertensive choroidopathy and hypertensive optic neuropathy.¹ This teaching case report describes a noncompliant patient with chronic hypertension in a hypertensive urgent crisis. It highlights the findings associated with hypertensive retinopathy, choroidopathy and optic neuropathy. This paper is intended for third- and fourth-year optometry students and all eyecare providers in clinical care.

Case Report

A 57-year-old white male presented to the Veterans Affairs (VA) healthcare clinic on Dec. 5, 2014 for the initial visit with his primary care provider. His blood pressure was 251/136 mmHg, and he had a 7-year history of severe hypertension managed outside the VA. He was taking amlodipine 10 mg daily, clonidine 0.1 mg bid and metoprolol 50 mg bid. He reported no focal neurological symptoms and no vision problems, and he denied palpitations. After examination, the patient was diagnosed with acute renal failure in addition to chronic renal disease due to malignant hypertension from improper medication management and noncompliance. Clonidine 0.2 mg was given in-office and the patient was referred to the emergency room.

The patient received additional medication in the emergency room, but his blood pressure remained elevated at 191/110 mmHg. He was transferred to a private nephrologist. After a review of records on file, the nephrologist placed the patient on the following medications to be taken once daily: carvedilol 12.5 mg, doxazosin 2 mg, clonidine 0.1 mg patch, 1,000 mg Vitamin D2 and calcium acetate. The nephrologist discharged the patient back to the VA with a poor prognosis given the history of non-adherence to medical care. On Dec. 12, 2014, the patient returned to his VA primary care provider for follow-up, and his blood pressure was improved at 114/72 mmHg. He reported seeing "sun spots" or "afterimages" in both eyes after starting the new hypertensive medications and was referred to the eye clinic for same-day evaluation.

Further history during the eye exam included a severe (10/10 on the pain scale) left temple headache that started one week prior. At the exam, the patient felt the severity of the headache was 5/10. Entering visual acuity and best-corrected visual acuity were 20/20 in each eye. Intraocular pressure was 10 mmHg OD and 11 mmHg OS at 2:09 p.m. Dilated fundus exam showed cup to disc (C/D) ratio of 0.4 OD and 0.5 OS. Macular findings were unremarkable. Vessels showed marked arteriolar attenuation and crossing changes. Some small, scattered hemorrhages were present in the posterior pole, OS>OD, as were a few areas of flat greyish spots OS (**Figures 1 and 2**). Optical coherence tomography (OCT) of the optic nerve and macula were performed. OCT of the optic nerve showed diffuse retinal nerve fiber layer thinning in both eyes (**Figure 3**). Macular scans showed a nasal area of thinning OD and inferior-nasal thinning in both eyes. The patient was diagnosed with moderate hypertensive retinopathy, and it was recommended that he return to the eye clinic for follow-up in 6 months.

The patient continued to be seen by nephrology and primary care at regular intervals over the next 2 years during which time medication non-compliance issues continued. His in-office blood pressure ranged from 105/71 mmHg to 190/109 mmHg. In February 2016, he reported poor home blood pressure control, fatigue, decreased appetite and weight loss. Consequently, dialysis was initiated and the patient was referred as a candidate for a kidney transplant.

In October 2016, approximately 2 years after his initial visit, the patient returned to the eye clinic for follow-up. His chief complaints at this visit were diminished distance vision while wearing glasses and the need to remove his glasses to read.

Hypertensive Choroidopathy: a Teaching Case Report | 2

Refraction yielded visual acuities of 20/20 OD and 20/20-2 OS with -1.75-1.25X070 OD and -2.00-0.75X140 OS. Intraocular pressure was 12 mmHg OD and 14 mmHg OS at 1:04 p.m. Slit lamp examination findings were remarkable for 1+ superficial punctate keratitis OU. Dilated fundus exam revealed a C/D ratio of 0.55 round (rd) OD, 0.6 rd OS with pallor and the appearance of thinning inferiorly. A linear hypopigmented choroidal lesion inferior to the optic nerve OS, which was not present on prior photos, was noted (**Figures 4 and 5**). OCT scans of the optic nerve and macula were obtained, and both eyes showed diffuse progressive thinning of the retinal nerve fiber layer (**Figure 6**) and macula compared to prior scans. The patient’s blood pressure at this visit was 146/87 mmHg. He was diagnosed with moderate hypertensive retinopathy with secondary extensive progressive atrophy of the optic nerve and macula. The patient was also diagnosed with a possible Siegrist streak OS, indicating hypertensive choroidopathy, and referred to ophthalmology for evaluation.



Figures 1 and 2. Fundus imaging at the patient’s initial visit in 2014 shows arteriovenous crossing changes and arteriolar attenuation OU secondary to hypertension, scattered retinal hemorrhages (OS>OD), and a few areas of flat greyish spots OS.

[Click to enlarge](#)



[Click to enlarge](#)

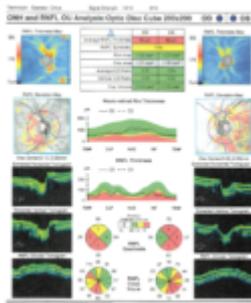
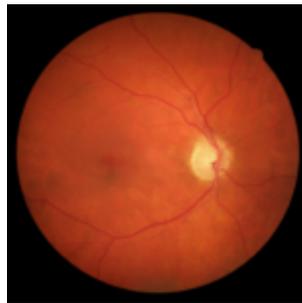


Figure 3. Initial OCT scans in 2014 reveal diffuse thinning of the retinal nerve fiber layer OU.

[Click to enlarge](#)



Figures 4 and 5. In October 2016, dilated fundus exam reveals C/D ratios of 0.55 rd OD and 0.6 rd OS with pallor and the appearance of thinning inferiorly. A linear hypopigmented choroidal lesion inferior to the optic nerve OS, which was not present on prior photos, was observed.

[Click to enlarge](#)



[Click to enlarge](#)

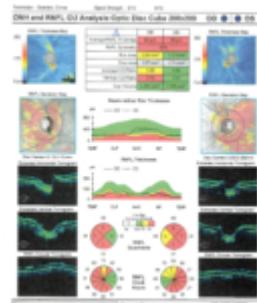


Figure 6. OCT scans obtained in October 2016, show diffuse, progressive thinning of the retinal nerve fiber layer (and macula).

[Click to enlarge](#)

In December 2016, the patient was evaluated by ophthalmology. Examination findings were consistent with the October 2016 optometric findings. The ophthalmologist recorded a C/D ratio of 0.55 rd OD, 0.6 rd OS with pallor and appearance of thinning inferiorly OU and superiorly OD. The hypopigmented choroidal lesion extending inferior to the optic nerve OS was confirmed to be consistent with Siegrist streak. The final diagnosis was malignant hypertension with temporal optic nerve pallor OU and hypertensive choroidopathy with Siegrist streak OS. The recommended follow-up was with optometry in 6 months.

In June 2017, after retinal consultation, the patient returned to the eyecare clinic for the 6-month follow-up. He had no ocular complaints. Visual acuity was 20/25-2 OD and 20/30-2 OS. Blood pressure was 174/84 mmHg. Intraocular pressure was 15 mmHg OD and 16 mmHg OS at 9:56 a.m. Slit lamp examination was unremarkable. Dilated fundus exam showed 1+ nuclear sclerotic cataracts OU, stable C/D ratios of 0.55 rd OD and 0.6 rd OS with pallor OU, Siegrist streak inferior to the optic nerve OS, macular mottling OU, attenuated vessels and unremarkable retinal periphery OU. OCT of the optic nerve and macula were obtained again and showed stable thinning of the retinal nerve fiber layer and macula (**Figure 7**)

Hypertensive Choroidopathy: a Teaching Case Report | 3

compared to scans from the October 2016 visit. The patient was assessed as having malignant hypertension with improved control of blood pressure, stable optic nerve atrophy OU and Siegrist streak OS. He returned in October 2017 to undergo baseline visual field testing (**Figures 8 and 9**). The results were unreliable OU; however, the test showed defects inferiorly OU and superiorly OS, which correlated with the Siegrist streak OS.

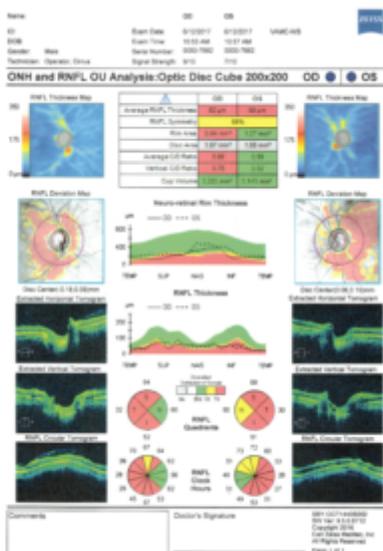


Figure 7. OCT in June 2017 shows stable thinning of the retinal nerve fiber layer (and macula) OU.
[Click to enlarge](#)



Figure 8. Baseline visual field testing in October 2017 shows defects inferiorly OU.
[Click to enlarge](#)

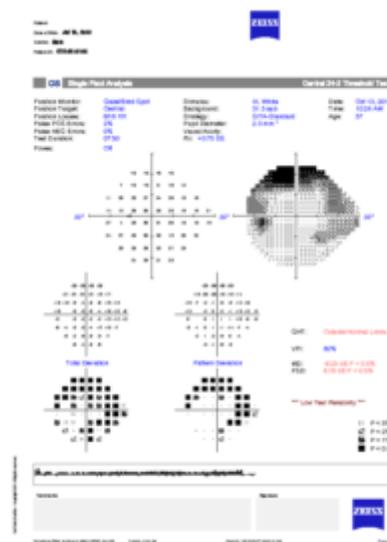


Figure 9. Baseline visual field testing in October 2017 shows defects inferiorly OU and superiorly OS, which correlates with the Siegrist streak OS.
[Click to enlarge](#)

Educator's Guide

Key concepts

1. Hypertension affects millions of Americans, and dilated fundus exams are essential in the management of the condition
2. Recognizing signs and symptoms of hypertensive retinopathy and choroidopathy may help providers to understand and manage these conditions
3. There are key differences in the vascular flow between the retina and choroid and these differences play a role in the development of hypertensive retinopathy vs. choroidopathy
4. Ancillary testing, such as fundus photography, OCT, fundus autofluorescence and fluorescein angiography may be useful in the management of hypertensive retinopathy and choroidopathy

Learning objectives

After this case discussion, participants should be able to:

1. Classify hypertensive retinopathy
2. Categorize hypertensive crises into emergencies and urgencies
3. Recognize the fundus findings associated with hypertensive retinopathy
4. Identify common findings associated with hypertensive choroidopathy
5. Understand the difference between retinal and choroidal blood flow
6. Understand how ancillary testing can be used to recognize and manage hypertensive retinopathy and choroidopathy

Hypertensive Choroidopathy: a Teaching Case Report | 4

7. Understand the optometrist's role in managing uncontrolled hypertension

Discussion points

1. Categorize hypertensive crises into emergencies and urgencies
2. Describe the blood flow from the ophthalmic artery to the retina and choroid
3. What retinal findings are associated with hypertensive retinopathy?
4. What are some ocular findings associated with hypertensive choroidopathy?
5. What are the differential diagnoses of hypertensive choroidopathy?
6. What is the pathophysiology behind optic nerve head and nerve fiber layer changes in chronic hypertension?
7. Describe the role of fluorescein angiography and fundus autofluorescence in hypertensive retinopathy/choroidopathy diagnosis and management
8. What are some end organs that are damaged by hypertension?

Discussion

Hypertension is defined by the American Heart Association as systolic pressure greater than 139 mmHg or diastolic pressure greater than 89 mmHg. Approximately 75 million Americans have hypertension and are at increased risk for myocardial infarction, heart failure, stroke, renal disease and early death.⁶ An accelerated hypertension or hypertensive crisis affects approximately 1% of people with high blood pressure and is defined as extremely high blood pressure that develops rapidly and causes some type of organ damage. It is most common in younger adults, especially African Americans. Blood pressure is typically 180/120 mmHg or greater in these cases, which should be treated as medical emergencies.

Changes in vasculature from hypertension are visible in the retina and are indicative of vessel changes elsewhere in the body. The retina receives a dual blood supply from branches of the ophthalmic artery. The inner retina is supplied by the central retinal artery. The outer retina and choroid receive their blood supply from the long and short posterior ciliary arteries.⁶ The posterior ciliary artery is the main arterial blood supply for the optic nerve.²⁹

The retinal and choroidal vascular beds have fundamentally different properties. The choroidal bed lacks an autoregulatory mechanism for blood flow, has no blood-ocular barrier, and has a sympathetic nerve supply.¹ The retinal vascular bed has efficient autoregulation, a blood-retinal barrier, and no sympathetic nerve supply. Because of these properties, the choroidal and retinal vascular beds respond differently to elevated blood pressure.

In hypertensive retinopathy, the initial response to elevated blood pressure is vasospasm and vasoconstriction of the retinal arterioles leading to arteriole narrowing called the vasoconstrictive phase.² Eventually, elevated blood pressure may lead to endothelial damage, intimal thickening and vessel narrowing called the sclerotic phase. This results in copper wiring, i.e., moderate vascular wall changes, and arteriovenous nicking. If hypertension remains uncontrolled, an exudative phase may develop. In this stage, the blood-retina barrier is disrupted leading to blood and fluid accumulation within the retina. Ischemic findings during this phase may include cotton wool spots, microaneurysms, retinal hemorrhages and macular edema.^{2,3,12} It is important to note though that the stages may not be sequential. The exudative signs may be seen without the sclerotic phase. This occurs because the sclerotic phase is a result of chronic hypertension and the exudative phase is indicative of recent uncontrolled hypertension.¹³

Accelerated hypertension can lead to choroidal ischemia and hypertensive choroidopathy, as in the patient in this case report. In reno-vascular malignant hypertension, excessive angiotensin and norepinephrine in the choroidal fluid cause choroidal vasoconstriction that leads to the ischemia. Hypertensive choroidopathy is less commonly seen than hypertensive retinopathy. It can manifest as Elschnig spots, Siegrist streaks and rarely serous retinal detachments.⁵ Elschnig spots are changes to the retinal pigment epithelium (RPE) that overlies infarcted choriocapillaries. These appear as pale, yellow, well-demarcated lesions often found in the perimacular region followed by the peripapillary region.¹ Fluorescein angiography at this stage often shows generalized delayed patchy filling of the choroidal vascular bed and in the macular region marked delay and staining of the lesions during the late phase.¹ Over time, the RPE becomes hyperpigmented with a margin of hypopigmentation. Healed Elschnig spots do not leak fluorescein, but transmission defects may appear through the hypopigmented halo.⁹ In cases of chronic or severe hypertension, Siegrist streaks can be seen as linear hyperpigmented streaks along the course of choroidal arteries.⁹ The presence of Siegrist streaks may signify advanced

Hypertensive Choroidopathy: a Teaching Case Report | 5

vascular sclerosis.¹ Persistent chronic choroidal ischemia may lead to progressive late RPE degenerative lesions that are widely scattered with heavy distribution in the temporal aspect of the macula and periphery.¹ The lesions can be composed of polymorphic RPE atrophic lesions as well as diffuse pigmentary changes and may be focal to confluent.¹ The macular changes often look like those seen in senile macular degeneration, and the peripheral fundus changes may resemble late-stage birdshot retinopathy.¹ These late degenerative lesions are typically more extensive than the acute lesions, and fluorescein angiography often shows even more extensive lesions than seen clinically.

In rare cases, serous retinal detachment occurs because of global choroidal dysfunction and decompensation of the RPE.⁶ These are usually bullous in nature, and the macular region is often the area affected followed by the periphery. The detachments are usually shallow and well-circumscribed. Once resolved, extensive RPE changes are often more evident.

Hypertensive optic neuropathy usually presents as bilateral disc swelling. This finding has the strongest association with death; therefore, it is a true hypertensive emergency. The pathogenesis of the papilledema is unclear but may be due to ischemia, raised intracranial pressure secondary to hypertensive encephalopathy or obstruction of axoplasmic flow from ischemia and choroidal nonperfusion.^{1-3,12} Other causes of papilledema, such as space-occupying lesions and benign idiopathic intracranial hypertension must be ruled out, so imaging may be required. Optic disc swelling secondary to hypertension usually resolves with improved blood pressure control. Resolution of the swelling often results in optic nerve pallor and retinal nerve fiber layer loss. Longstanding chronic elevated hypertension may result in nerve fiber layer loss as well, as seen in this patient.¹¹ The nerve fiber layer defects are localized and have not been shown to change the size or shape of the neuroretinal rim or peripapillary atrophy as seen in glaucomatous optic neuropathy.¹¹ Chronic hypertension can lead to vasoconstriction of peripapillary choroidal vessels and posterior ciliary arteries that supply the optic nerve. This chronic hypoperfusion can result in optic nerve atrophy and pallor.^{27,28} These changes are reflected on an OCT as thinning in the nerve fiber layer.

Differential diagnosis

There are several conditions that may mimic hypertensive retinopathy including diabetic retinopathy, anemic retinopathy, venous occlusive disease, carotid occlusive disease, radiation retinopathy, perifoveal telangiectasia and collagen vascular diseases.^{14,18,19} Because the RPE and other tissues supplied by choroidal vasculature respond in limited ways to decreased perfusion, hypertensive choroidopathy may be difficult to distinguish from other clinical conditions that cause choroidal ischemia including vascular, inflammatory and degenerative diseases. These conditions include toxemia of pregnancy, disseminated intravascular coagulopathy, multifocal acute ischemic choroidopathy, collagen vascular disorders, thrombotic thrombocytopenic purpura, leukemia, Goodpasture syndrome, hemolytic diseases, cardiac lesions, giant cell arteritis, and other local vascular and systemic disease.^{1,15} Furthermore, in the late stages of hypertensive choroidopathy, the macular region is often indistinguishable from senile macular degeneration as drusen develop. This may indicate that chronic choroidal ischemia may play a role in development of drusen.⁴ Choroidal ischemia can also result from iatrogenic sources. Laser thermocoagulation and photodynamic therapy have been shown to create segmental choroidal ischemia and vascular occlusion.¹⁷

Role of fluorescein angiography and fundus autofluorescence

Fluorescein angiography may show varied findings. With hypertensive retinopathy, dilation of capillaries, telangiectasia, capillary nonperfusion and/or leakage from retinal vessels may be seen.^{14,18} Leakage may be present at the optic nerve if edema is present.^{14,18} In hypertensive choroidopathy, choroidal bed abnormalities can be seen and RPE degeneration is visualized more clearly compared to visualization with funduscopy alone. This is due to unmasking of choroidal fluorescence in degenerated areas, which demonstrate choroidal circulatory insufficiency.²¹ In early hypertensive choroidopathy, a mild to marked delayed or patchy filling of the choroidal bed can be seen and is especially noticeable in the macular area. Acute focal RPE lesions are associated with this delayed and patchy choroidal bed fluorescein filling and will stain during the late phase.^{21,22} Older degenerative choroidal lesions will begin to show unmasking of the choroidal fluorescence during the transit stage, but they do not stain late.²¹

By highlighting lipofuscin distribution in the RPE, fundus autofluorescence may also play a role in identifying retinal changes due to hypertension. In a small study by Ramezani, et al., fundus autofluorescence revealed some pathological changes at the level of the RPE.⁴ These changes included a hyper-autofluorescent ring surrounding a small area of hypo-autofluorescence at the fovea. This was evident in patients with chronic hypertension significantly more often than in normotensive patients. It was also noted that hyper-autofluorescent patches of RPE outside of the fovea were found more often in patients with chronic hypertension, but this was not statistically significant.⁴

Hypertensive Choroidopathy: a Teaching Case Report | 6

Changes in the foveal RPE are thought to be the result of possible damage caused by chronic hypertension resulting in lipofuscin accumulation in the RPE at the fovea, a reduction of macular pigment, or both. It is also possible that patches of mixed autofluorescent patterns outside the fovea are secondary to past episodes of acute elevated blood pressure; however, a larger study would be needed to find statistical evidence of this. It is possible that fundus autofluorescence may be used to detect retinal problems such as hypertensive retinopathy or choroidopathy in early stages because the retinal changes found were not visible by routine imaging or funduscopy.⁴

Target organ damage

Hypertensive end organ damage occurs when the major organs (i.e., heart, brain, kidneys, eyes) nourished by the circulatory system undergo damage or impairment from uncontrolled hypertension. This can manifest in a variety of ways including stroke, myocardial infarction, heart failure, albuminuria, proteinuria, renal failure, encephalopathy, subarachnoid hemorrhage, dissecting aortic aneurysm, pulmonary edema and renal failure.^{10,12-14}

Ocular target organ damage includes the ocular findings discussed above as well as retinal artery or vein occlusions, cranial nerve palsies, nonarteritic anterior ischemic optic neuropathy and macroaneurysms.^{12,13}

Identifying end organ damage is important in the clinical decision-making process for managing the patient’s overall cardiovascular risk. If end organ damage is present, such as in the case of this patient, more intense treatment may be required as the goal should be to reduce the blood pressure to values of 130/80 mmHg or lower.¹⁰

Classification and management of hypertensive patients

TABLE 1
Classification of Hypertensive Retinopathy

Grade of retinopathy	Retinal findings	Diastolic BP (mmHg)	Systemic associations
Mild	generalized and focal arteriolar narrowing, A/V crossing changes	> 90 and < 110	modest association with risk of stroke, heart disease
Moderate	hemorrhages, retinal aneurysms, cotton wool spots, hard exudates	≥ 110 to 120	strong association with stroke, death, cardiovascular disease
Severe	moderate retinal findings plus optic nerve swelling	≥ 120	strong association with death

From Wong et al¹³
A/V = arterio/venous

Table 1. [Click to enlarge](#)

Various staging systems for hypertensive retinopathy have been developed. One that has been widely used in more recent years was presented by Wong et al¹³ (**Table 1**). Optometrists play an important role in the treatment of patients with hypertension and knowing how to effectively manage these patients is crucial (**Table 2**). The information outlined in Tables 1 and 2 can aid the eyecare provider in grading retinopathy, educating the patient on the likely target organ damage based on ocular findings, and directing appropriate timely referrals and follow-up visits. For example, when mild retinopathy is present, it indicates chronic hypertension and does not require as urgent of a referral as moderate hypertensive retinopathy, which represents an acute rise in blood pressure and similar vascular damage occurring in the brain, heart and kidneys.^{13,14,18} Optic nerve swelling is most associated with mortality and renal failure and therefore is a true medical emergency.^{13,15}

Hypertensive Choroidopathy: a Teaching Case Report | 7

Treatment of hypertensive retinopathy is typically controlling the blood pressure with medication. If blood pressure has not been elevated for a prolonged period and is controlled promptly, retinal blood vessels may return to a normal state with no permanent changes.^{14,20} If blood pressure remains elevated and hypertension is chronic, retinal findings may be irreversible even if blood pressure is subsequently brought under control.^{14,20}

Assessment of learning objectives

The assessment of the learning objectives for this teaching case report can be accomplished in several ways. Students presented with fundus photos should be able to describe the photos, including normal and abnormal findings of the retinal vasculature, optic nerve and macula as well as grade any hypertensive changes. PowerPoint slide quizzes are an option for testing students' knowledge. Once abnormal findings are identified, students should be queried on differential diagnosis along with what additional testing should be performed to determine the underlying disease and any associated conditions. Students should also be assessed on their ability to describe and recognize a hypertensive crisis and what organs can be damaged from hypertension. Case-based discussions are a good platform for helping students work through the differential diagnoses and management. In this report, fluorescein angiography and fundus autofluorescence were addressed as having a role in the diagnosis and management of hypertensive retinopathy; students can be evaluated for their knowledge of each of these tests and should be able to describe images in detail and describe normal and abnormal retinal and choroidal circulation. Role-playing simulations can help students meet patient education and management expectations.

Conclusion

Optometrists play an important role in the management of patients with systemic arterial hypertension. Ocular manifestations vary, and prompt recognition and accurate diagnosis are crucial for optimizing both the ocular and systemic health of the individual.

References

1. Hayreh SS, Servais GE, Viridi PS. Fundus lesions in malignant hypertension. VI. Hypertensive choroidopathy. *Ophthalmology*. 1986;93(11):1383-1400.
2. Bhargava M, Ikram MK, Wong TY. How does hypertension affect your eyes? *J Hum Hypertens*. 2012;26(2):71-83.
3. Hammond S, Wells JR, Marcus DM, Prisant LM. Ophthalmoscopic findings in malignant hypertension. *J Clin Hypertens*. 2006;8(3):221-223.
4. Ramezani A, Saberian P, Soheilian M, et al. Fundus autofluorescence in chronic essential hypertension. *J Ophthalmic Vis Res*. 2014 Jul-Sep;9(3):334-8.
5. Bourke K, Patel R, Pisant M, Marcus DM. Hypertensive choroidopathy. *J Clin Hypertens*. 2004 Aug;6(8):471-2.
6. Fraser-Bell S, Symes R, Vaze A. Hypertensive eye disease: a review. *Clin Exp Ophthalmol*. 2017;45(1):45-53.
7. Schechtman DL, Falco LA. Hypertension: more than meets the eye. *Review of Optometry*. 2007 Sept 15;144(9).
8. Moser M, Papademetriou V, Pickering TG, Sica DA. Hypertension treatment guidelines. *J Clin Hypertens*. 2004 Aug;6(8):452-457.
9. Ugarte M, Horgan S, Rassam S, Leong T, Kon CH. Hypertensive choroidopathy: recognizing clinically significant end-organ damage. *Acta Ophthalmol*. 2008 Mar;86(2):227-228.

TABLE 2
Hypertensive Crisis Management by Optometrists

	Severe Hypertension	Hypertensive Urgency	Hypertensive Emergency
	Urgent	Urgent	Emergency
Blood pressure	> 180/110 mmHg	> 180/120 mmHg	> 180/120 mmHg
Clinical findings	no acute TOD, + mild HTN, + HTN-associated ocular findings, may have headaches	+ moderate HTN retinopathy, ± headaches and/or dyspnea, peripheral edema	papilledema w/ moderate HTN retinopathy, chest pain, severe dyspnea, headache, altered mental status, ± focal neurological deficit
Referral	patient's PCP consultation	patient's PCP consultation	emergency department or call 911
Physician setting	outpatient, long-acting oral medical treatment needed	usually outpatient, may advise sending to PCP's office or ED, oral medication with rapid onset needed	inpatient, ICU, intravenous medication needed
Treatment timeline	BP control w/in 3-7 days	BP control w/in 24-72 hours	immediate BP reduction, titrated and decreased by 15-25% w/in 2 hours, ICU release once 140/90 mmHg
Recommended optometric follow-up	1-3 months	1 month	1 month after release from hospital

From Meez et al.¹¹ Wong et al.¹² Wong et al.¹³
HTN = hypertension; BP = blood pressure; TOD = target organ damage; PCP = primary care provider; ED = emergency department; ICU = intensive care unit

Table 2. Click to enlarge

Hypertensive Choroidopathy: a Teaching Case Report | 8

10. Schmieder RE. End organ damage in hypertension. *Dtsch Arztebl Int.* 2010 Dec;107(49): 866-73.
11. Hayreh SS, Jonas JB. Appearance of the optic disk and retinal nerve fiber layer in atherosclerosis and arterial hypertension: an experimental study in rhesus monkeys. *Am J Ophthalmol.* 2000 Jul;130(1):91-96.
12. Meetz R, Harris T. The optometrist's role in the management of hypertensive crises. *Optometry.* 2011;82(2):108-116.
13. Wong TY, Mitchell P. Hypertensive retinopathy. *N Engl J Med.* 2004 Nov 25;351(22):2310-7.
14. Luo BP, Brown GC. Update on the ocular manifestations of systemic arterial hypertension. *Curr Opin Ophthalmol.* 2004 Jun;15(3):203-210.
15. Wong TY, McIntosh R. Hypertensive retinopathy signs as risk indicators of cardiovascular morbidity and mortality. *Br Med Bull.* 2005 Sep 7;73-74:57-70.
16. Hayreh SS. *Ocular vascular occlusive disorders.* 1st ed. Switzerland: Springer International Publishing; 2015:409-410.
17. Choroidal perfusion abnormalities [Internet]. San Francisco: American Academy of Ophthalmology; c2017 [cited 2017 August 14]. Available from: <https://www.aao.org/bcscsnippetdetail.aspx?id=bb9a0c9a-4a9c-488a-b28d-52cab794fc30>.
18. Mandava N, Yannuzzi LA. *Vitreoretinal disease: the essentials.* New York, NY: Thieme; 1999:193-209.
19. Gerstenblith AT, Rabinowitz MP. *The Wills eye manual,* 6th ed. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2012:308.
20. Tso MO, Jampol LM. Pathophysiology of hypertensive retinopathy. *Ophthalmology.* 1982 Oct;89(10):1132-1145.
21. Hayreh SS, Servais GE, Viridi PS. Fundus lesions in malignant hypertension. VI. Hypertensive choroidopathy. *Ophthalmology.* 1986 Nov;93(11):1383-400.
22. Wolffsohn JS, Hurcomb PG. Hypertension and the eye. *Curr Hypertens Rep.* 2002 Dec;4(6):471-6.
23. Nwankwo T, Yoon SS, Burt V, Gu Q. Hypertension among adults in the United States: National Health and Nutrition Examination Survey, 2011-2012. *NCHS Data Brief, No. 133.* Hyattsville, MD: National Center for Health Statistics, Centers for Disease Control and Prevention, US Dept of Health and Human Services; 2013.
24. Farley TA, Dalal MA, Mostashari F, Frieden TR. Deaths preventable in the U.S. by improvements in use of clinical preventive services. *AM J Prev Med.* 2010;38(6):600-9.
25. Siegrist A. Beitrag zur Kenntnis der Arteriosklerose der Augengefasse. In: IXth International Congress on Ophthalmology. 1899:131-9.
26. Klien BA. Ischemic infarcts of the choroid (Elschnig spots). A cause of retinal separation in hypertensive disease with renal insufficiency. A clinical and histopathologic study. *Am J Ophthalmol.* 1968 Dec;66(6):1069-74.
27. Brown M. Ocular manifestations of systemic hypertension. *Optometry Times.* 2013 June 19.
28. Kovach JL. Hypertensive optic neuropathy and choroidopathy in an 18-year-old woman with renal failure. *Retin Cases Brief Rep.* 2010 Spring;4(2):187-9.
29. Hayreh SS. Blood supply of the optic nerve head. *Ophthalmologica.* 1996;210(5):285-95.



Dr. Richwine, a graduate of Indiana University School of Optometry, is an optometrist in Charlotte, N.C. She is an Associate Professor and educates optometry externs and optometry residents.

Dr. Mah [diana.mah@va.gov], a graduate of the Pennsylvania College of Optometry, is an optometrist in Winston Salem, N.C.

Dr. Mercado, a graduate of the Illinois College of Optometry, is an optometrist in Hickory, N.C.