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# Hypertensive Retinopathy

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## REFERRAL REFINEMENT PART 9: C-16903 O/D

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The prevalence of hypertension increases with age and therefore it is a growing public health problem in the Western world. Approximately 1.56 billion people are estimated to be affected with hypertension worldwide by 2025.<sup>1</sup> The prevalence of hypertension in England is thought to be in the order of 32% in men and 30% in women.<sup>2</sup> Hypertension is the single most important modifiable risk factor for stroke. Even milder degrees of blood pressure elevation pose increased risk for cardiovascular events. It is an underlying factor in the development of peripheral vascular disease and hypertension is associated with vascular events in the brain, heart, kidneys and eyes. This article describes the ocular features of hypertension and aims to provide referral advice to practitioners for different stages of the disease.

### Classification of systemic hypertension

Essential hypertension is of unknown aetiology and yet is responsible for up to 95% of cases.<sup>3</sup> Risk factors include increasing age, family history, obesity, smoking and being of African-Caribbean race. Essential hypertension is diagnosed when the average blood pressure measures greater than 140mmHg systolic or 90mmHg diastolic on at least two subsequent visits. Malignant hypertension is rare and occurs when the systolic blood pressure is over 200mmHg or the diastolic blood pressure is greater than 140mmHg. As essential hypertension is an asymptomatic condition, many patients remain undiagnosed to this silent killer. The retina provides a window to study the human circulation. Retinal arterioles can be visualised both easily and non-invasively. They share similar anatomical and physiological

properties with the cerebral and coronary microcirculations. Therefore it may be at a routine examination that the diagnosis of hypertension is made by the attending optometrist. Recent research in the USA found that optical professionals detected signs of certain chronic conditions before any other healthcare provider recorded the condition, including 65% of the time for high cholesterol and 30% of the time for hypertension.<sup>4</sup>

### Hypertensive Retinopathy

Hypertensive retinopathy represents the ophthalmic findings of end-organ damage secondary to systemic arterial hypertension. As well as retinal changes, hypertension can also damage the choroidal circulation and is responsible for optic and cranial neuropathies. Hypertension may also present in the form of subconjunctival haemorrhages. The

differential diagnosis of chronic hypertensive retinopathy includes diabetic retinopathy, hyperviscosity syndromes, radiation retinopathy and the ocular ischaemic syndrome.

The arteriolar changes of hypertension are thought to result primarily from vasospasm, whereas the arteriolosclerotic changes are considered to occur secondary to thickening of the arteriolar wall. Because hypertension accelerates arteriolosclerotic change it is impossible to completely separate these processes. Diffuse arteriolar narrowing is characteristic of hypertensive retinopathy. The normal arteriole to venule ratio is 2:3 and this is reduced in hypertension. Focal arteriolar narrowing is attributed to localized areas of spasm of the arteriolar wall and may be reversible.

Hypertensive arteriolosclerosis refers to the progressive increase in the elastic and muscular components of the wall of the arteriole induced by hypertension. The changes in the walls of the arterioles induce a change in the character of the light reflex from the vessels.

### Classification

In 1953, Scheie classified the changes of hypertension and arteriolosclerosis separately into five stages ranging from normal to the most severe changes in the retina (Table 1). Normally the arteriolar wall is invisible and only the column of red blood cells in the lumen is visible. There is a thin line of reflected light in the middle of the blood column – the normal light reflex. As the wall becomes thickened the light reflex loses its brightness and becomes somewhat broader, duller and more diffuse in appearance. With increasing thickening of the arteriolar wall and decreasing lumen, there is further diffusion of the light from the arteriole,



Stage	Description
<b>Hypertensive changes</b>	
0	Patient has diagnosed hypertension There are no visible retinal vascular abnormalities
I	Diffuse arteriolar narrowing is seen, especially in the smaller vessels. Arteriolar calibre is uniform, with no focal constriction
II	Arteriolar narrowing is more pronounced, and there can be focal areas of arteriolar constriction
III	Focal and diffuse arteriolar narrowing is more obvious and severe Retinal haemorrhages may be present
IV	All of the previously listed abnormalities may be present, along with retinal oedema, hard exudates, and optic disc oedema
<b>Arteriolosclerotic changes</b>	
0	Normal
1	There is broadening of the light reflex from the arteriole with minimal or no arteriovenous compression
2	Light reflex changes and crossing changes are more prominent
3	The arterioles have a "copper wire" appearance, and there is more arteriovenous compression
4	The arterioles have a "silver wire" appearance, and the arteriovenous crossing changes are most severe

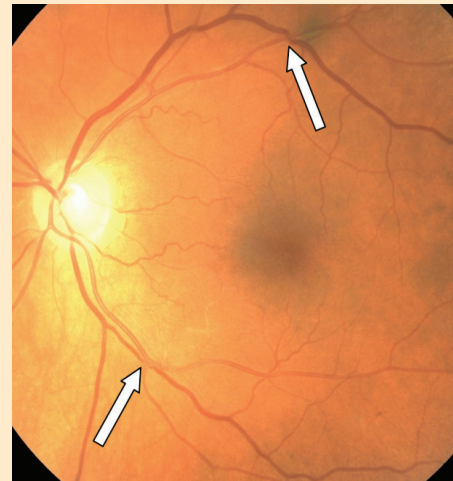
**Table 1**

Scheie classification of hypertensive retinopathy

Stage	Description
1	Mild to moderate narrowing or sclerosis of the arterioles
2	Moderate to marked narrowing of the arterioles Local and/or generalized narrowing of arterioles Exaggeration of the light reflex Arteriovenous crossing changes
3	Retinal arteriolar narrowing and focal constriction Retinal oedema Cotton-wool patches Retinal haemorrhages Hard exudates
4	As for Group 3 plus optic disc swelling

**Table 2**

The Keith-Wagener-Barker classification of hypertensive retinopathy



**Figure 1**

A/V nipping and arteriolar sclerosis

and the light reflex adopts a reddish-brown hue or "copper-wire" reflex. When the column of blood can no longer be visualised it is termed "silver-wire".

The Keith-Wagener-Barker classification is commonly used to classify hypertensive retinopathy. It can divide retinopathy into acute and chronic phases (Table 2). In grades 1 and 2 there is hyalinization and thickening of the retinal arterial walls leading to the straightened vessels in grade 1 and arteriovenous nipping (see next section) in grade 2. In grade 3 hypertensive retinopathy, the systemic diastolic blood pressure is typically at least 110 to 115mmHg. At this point the retinal arteries lose their ability to autoregulate their blood flow and the high pressure is passed distally to the retinal arterioles and capillary bed. In grade 4 hypertensive retinopathy, the systemic diastolic blood pressure is usually at least 130 to 140 mmHg. With both grades 3 and 4 hypertensive retinopathy, the increased blood pressure can damage the blood vessel wall, leading to fibrinoid necrosis (the presence of fibrin thrombi within the vascular lumina). Grades 1 and 2 are chronic whereas grades 3 and 4 indicate acute retinal vascular

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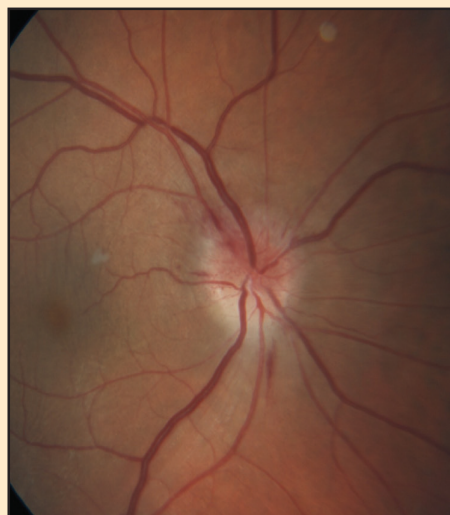
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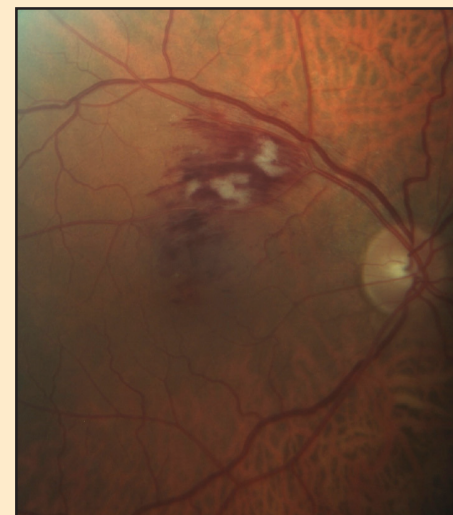
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**Figure 2**  
Retinal macroaneurysm



**Figure 3**  
Swelling of the superior hemi-disc in non-arteritic  
ischaemic optic neuropathy



**Figure 4**  
Branch retinal vein occlusion (BRVO)

decompensation and are seen in malignant hypertension (see later).

## A/V Nipping

Arteriolosclerotic thickening and perivascular glial cell proliferation contribute to compression of venules at arteriovenous crossing, termed arteriovenous (a/v) nipping (Gunn's sign) (Figure 1). A/v nipping is a highly specific finding and the hallmark of chronic hypertensive retinopathy. If there is impedance to flow, the segment of the vein distal to the constriction appears larger, darker and more tortuous. Additional signs to impedance of blood flow are retinal haemorrhages, macular oedema and cotton wool spots. Secondary ocular complications of chronic systemic arterial hypertension include retinal vascular occlusive disease, macroaneurysm formation (Figure 2), and non-arteritic ischaemic optic neuropathy (Figure 3).

Branch retinal vein occlusions (BRVOs) (Figure 4) almost always occur at an arteriovenous crossing. The artery is nearly always anterior to the vein. Most BRVOs occur supero-temporally, probably because this is

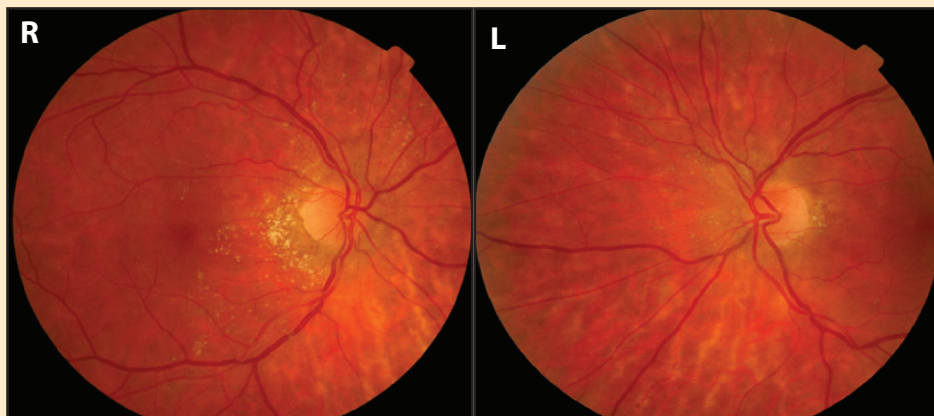
where the highest concentration of arteriovenous crossings lie. The Eye Disease Case Control Study clearly demonstrated the important association of hypertension with vein obstructions. In this study, more than 50% of BRVOs were associated with hypertension.<sup>5</sup> The Framingham Study reported an association of Age-related Macular Degeneration (AMD) with systemic hypertension, a relation that increased with the duration of the hypertension.<sup>6</sup>

## Malignant Hypertension

Malignant hypertension causes systemic complications including ocular, cardiac, renal and cerebral injury. Persistently elevated malignant hypertension can lead to a rapidly fatal course with heart failure, myocardial infarction, stroke or renal failure. Visual disturbances are common in malignant hypertension. Symptoms include headaches, scotomata, diplopia, dimness in vision and photopsia. Retinal haemorrhages tend to be linear and occur in the nerve fibre layer in the peripapillary region. Fibrinoid necrosis of the choroidal vessels can cause patchy non-perfusion

of areas of the choriocapillaris. Patches of retinal pigment epithelium (RPE) overlying occluded choriocapillaris appear yellow and profusely leak on fluorescein angiography. As these heal, the RPE becomes hyper-pigmented directly over the occluded choriocapillaris with a margin of hypopigmentation. Localised bullous detachments of the neurosensory retina or RPE are occasionally observed. Some of these are attributed to a breakdown of the inner blood-retinal barrier with retinal endothelial cell decompensation. However, most are considered to result from RPE decompensation due to fibrinoid necrosis of choroidal arteries with occlusion of the choriocapillaris. The outer retina and sub-retinal space in these cases contain a protein-rich exudate. Siegrist's streaks are linear configurations of hyper-pigmentation that develop over sclerotic choroidal arteries in chronic hypertension. As a result of chronic swelling of the optic disc, hard exudates can precipitate around the disc (Figure 5) or can form a stellate pattern or macular star. Hypertensive optic neuropathy





**Figure 5**

History of malignant hypertension secondary to renal stenosis in a 30-year-old male. The disc swelling has resolved but peripapillary exudates remain

occurs secondary to vasoconstriction of the posterior ciliary arteries supplying the optic nerve head.

The differential diagnosis of malignant or accelerated hypertensive retinopathy includes bilateral bullous central serous chorioretinopathy, bilateral central retinal vein occlusion (CRVO) (Figure 6), collagen vascular diseases and diabetic retinopathy complicated by diabetic papillopathy.

## Referral Guidelines

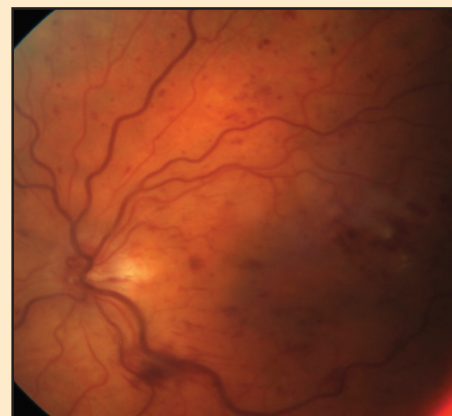
Any patient displaying retinal signs of hypertensive changes with undiagnosed systemic hypertension should be referred to their general practitioner for investigation, diagnosis and management. The degree of urgency will naturally vary depending upon the degree of retinopathy, with later stages of retinopathy requiring greater urgency, and malignant hypertensive retinopathy requiring immediate referral to the A&E department.

Perhaps the most clinically relevant association between findings of hypertensive retinopathy and systemic disease comes from Wong and Mitchell.<sup>7</sup> It has been shown that there is a modest association with increased risk of clinical stroke, sub-clinical

stroke, coronary heart disease, and mortality if a patient exhibits one or more of the following arteriolar signs: generalised arteriolar narrowing, focal arteriolar narrowing, arteriovenous nipping or arteriolar wall opacity (silver wiring). There is a strong association with risk of clinical stroke, sub-clinical stroke, cognitive decline, and cardiovascular mortality where there is moderate retinopathy and one or more of the following clinical signs are present: haemorrhages (blot, dot, or flame shaped), microaneurysms, cotton wool spots or hard exudates. It is important therefore that the optometrist refers an at-risk patient to their general practitioner.

## Association with diabetes

Diabetes and hypertension are both vascular risk factors and may share similar pathophysiological mechanisms. The prevalence of diabetes among patients with hypertension is high, and Type 2 diabetes may remain unrecognised for years before being diagnosed.<sup>8-9</sup> When diabetes is associated with hypertension, cardiovascular risk rises exponentially and retinopathy becomes more severe and rapidly progressive. In turn, tighter control



**Figure 6**

Central retinal vein occlusion (CRVO)

of blood pressure in people with hypertension and diabetes has been shown to prevent cardiovascular events as well as halting the deterioration of both retinopathy and visual acuity (VA).<sup>10-12</sup> Among the various pathophysiological mechanisms, endothelial dysfunction has been implicated in the pathogenesis of the metabolic syndrome and points to a link between diabetes and hypertension.<sup>13-14</sup>

## Treatment

The treatment for hypertensive retinopathy is to correct the underlying condition by normalizing the blood pressure. This causes resolution of the fundus abnormalities over a period of weeks to months in eyes with grade 3 and 4 changes (Figure 5), but often does not affect the changes seen with grades 1 and 2 hypertensive retinopathy. Treatment of malignant hypertensive retinopathy, choroidopathy and optic neuropathy consists of lowering blood pressure in a controlled manner. If the decline is too rapid there is impairment of autoregulation and this can lead to ischaemia of the optic nerve head, brain and other vital organs. The management of malignant hypertension is considered a medical emergency. Untreated, the mortality rate is 50%

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at 2 months and 90% at one year.<sup>15</sup>

## Pre-eclampsia

Pre-eclampsia is defined as the development of proteinuria in a woman who has developed hypertension during her pregnancy. Pre-eclampsia has an incidence of approximately 5% and typically occurs after 20 weeks gestation.<sup>16</sup> Eclampsia is heralded by the onset of seizures in the setting of pre-eclampsia. Pre-eclampsia-eclampsia syndrome is a multi-system disorder that can include cardiovascular changes, haematological abnormalities, hepatic and renal impairment, and neurologic or cerebral manifestations.

Ocular sequelae are observed in 30% to 100% of patients with pre-eclampsia-eclampsia syndrome.<sup>17</sup> Blurred vision is the most common visual complaint, and focal or generalized arteriolar narrowing is the most common ocular finding in pre-eclampsia-eclampsia syndrome. Areas of non-perfusion or arterial and venous occlusive disease may also develop.<sup>18-19</sup>

Pre-eclampsia and eclampsia have

been associated with severe retinopathy similar to hypertensive retinopathy, with serous retinal detachments, yellow, opaque RPE lesions, and cortical blindness.<sup>20</sup> Choroidal dysfunction, primarily choriocapillaris ischaemia, is the underlying mechanism that leads to the serous retinal detachments and yellow RPE plaques.<sup>21</sup> While most patients recover normal vision within a few weeks of delivery, some have residual RPE changes in the macula that appear as Elschnig spots or which mimic macular dystrophy or tapetoretinal degeneration (a group of inherited abnormalities in the retina characterized by night blindness, retinal atrophy, weakening of the retinal vessels, pigment clumping, and contraction of the visual field).<sup>8</sup> Although rare, optic atrophy may develop if chorioretinal atrophy is widespread. Permanent blindness from retinal vascular changes is rare, and cortical blindness is generally reversible.

## Conclusion

This article has described the

retinal changes that are associated with systemic hypertension, and the possible consequences of this disease if left undetected and unmanaged. Optometrists are well-placed to detect such retinal changes and therefore should be fully conversant with appropriate detection and referral protocols.

## About the Author

Louise O'Toole is a consultant medical ophthalmologist in the Mater Private Hospital, Eccles Street, Dublin. She is also a lecturer to undergraduate optometrists in the Dublin Institute of Technology. She has been involved in teaching on the MSc in Clinical Optometry at City University, London, particularly in the area of Ocular Therapeutics, and she has written several articles in the field for Optometry Today.

## References

See <http://www.optometry.co.uk/clinical/index>. Click on the article title and then download "references".

## Module questions

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### 1. The diagnosis of malignant hypertension is made when:

- a) The diastolic blood pressure is greater than 60mmHg
- b) The diastolic blood pressure is greater than 120mmHg
- c) The systolic blood pressure is greater than 200mmHg
- d) The systolic blood pressure is greater in one arm compared to the other

### 2. Which of the following statements about arteriovenous nipping is FALSE?

- a) It is a feature of hypertension
- b) It is more commonly seen in the inferotemporal arcades
- c) It is termed Gunn's sign
- d) It refers to compression of a venule

### 3. Which of the following statements about malignant hypertension is FALSE?

- a) It may present with headaches
- b) It may present with visual disturbance
- c) It should be rapidly reversed
- d) It can result in renal failure

### 4. Which of the following is NOT part of the differential diagnosis of hypertensive retinopathy?

- a) Diabetic retinopathy
- b) Hyperviscosity syndromes
- c) Radiation retinopathy
- d) Central retinal vein occlusion

### 5. Which of the following is NOT an ocular feature of hypertension?

- a) Macroaneurysm
- b) Arteritic ischaemic optic neuropathy
- c) Cotton wool spots
- d) Retinal haemorrhages

### 6. Which of the following is NOT a feature of pre-eclampsia and eclampsia?

- a) Serous retinal detachments
- b) Elschnig spot
- c) Cortical blindness
- d) Step defects on visual perimetry