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Review Paper

# Insight into Hypertensive Retinopathy and Choroidopathy, The Unveiling of Silent Ocular Threat Prompted By Systemic Hypertension

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**ABSTRACT:**
This article details the impact on eye health of hypertension retinopathy and choroidopathy as a result of hypertension, its long-term effects on vision and mortality by the defilement of the retinal blood vessels along with the other multiple adverse effects. Hypertensive patients have concerns for these entities as the raised systolic pressure affects overall mortality and the raised diastolic pressure causes aortic disease. Therefore retinal vasculature named branch retinal artery and vein, central retinal artery and vein gets thickened and circumscribed followed by the tortoisity and aneurysm and on rupture and occlusion visual threat develops. The obstruction of the major retinal vessels; like branch retinal and central retinal artery or vein, is likewise dependent on hypertension. Retinal haemorrhages are the reaction of the necrotic vessels and their specific characteristics are defined under different levels. Lipids accumulate to form exudates at the area of haemorrhage while in malignant hypertension; papilloedema takes place due to haemorrhage and arteriolar ischemia over the optic disc and finally undergoes fibrinous necrosis. If the fluid accumulates under subretinal layers due to the leaks from the retinal pigment epithelium and choroid is commonly named choroidopathy or central serous chorioretinopathy, assumed fourth most ubiquitous retinopathy followed by age-related macular degeneration, diabetic retinopathy and branch retinal vein occlusion with the common complaints of metamorphopsia, hyperopia, central scotoma, reduced contrast sensitivity and colour saturation. For the diagnosis of disease, fluorescein angiography, and optical coherence tomography imaging are the best option. The first line of treatment prefers control over the blood pressure with the drugs angiotensin-converting enzyme inhibitors, calcium channel blockers, and diuretics. For the ophthalmic treatment low-dose aspirin, anti-VEGF, traditional argon laser, and photodynamic therapy are used.

***Keywords: hypertension, systolic, diastolic, hypertension retinopathy, choroidopathy, CSCR, FFA, OCT, ICG, VEGF.***

## INTRODUCTION:

Uncontrolled hypertension (HTN) affects various systems of the body including cerebrovascular, cardiovascular, retina of the eye and renal system, such damage is known as target-organ damage (TOD).[1] Chronic patients with raised systemic blood pressure extensively have three ocular entities hypertensive retinopathy, choroidopathy and optic neuropathy, further leading to significant unpropitious impact on the visual potential of the eye.[2] Among the various concerns of the blood, oxygen and nutrients are transferred to the organs through the circulatory system by the pumping of the heart. This pump creates a force to move in the vascular system called blood pressure which is not solely based on the heart but the lumen of the vessels too.[3] Two known subtypes of raised blood pressure, Primary type (essential) includes no reason for raised blood pressure and settled gradually over the years. Plaque formation (atherosclerosis) increases the chances of such hypertension. The secondary type of raised blood pressure has an underlying condition and appears suddenly, either due to Adrenal gland tumours, congenital heart diseases, medicines for cough and cold, pain relievers, contraceptive pills, cocaine, amphetamine, obstructive sleep apnea, kidney and thyroid issues. Sometimes during health checkups blood pressure increases with a temporary effect named white-coat hypertension.[4]

Some of the patients have headaches, shortness of breath and nasal bleeding, however, these are not considered distinct symptoms but if hypertension reaches an extensive level then they become life-threatening. The measurement of blood pressure is millimetres of mercury (mm Hg) and is divided into four categories by the American Heart Association and the American College of Cardiology. [4]

1. Normal B.P. range – 120/80 mm Hg.,
2. High B.P. range – 120 - 129/80 mm Hg.,
3. Stage 1 hypertension – 130 - 139/80 to 89 mm Hg.,
4. Stage 2 hypertension – 140 or higher/90 mm Hg or higher.

The health of the heart is based on blood pressure, characterized by systolic B.P. (SBP) and diastolic B.P. (DBP) components. SBP is the amount of blood pushed into the arteries by the heart during a contraction, the effort phase of the heart. Between two beats of SBP, the rest phase of the heart allows blood flow in arteries known as the DBP. Therefore Systolic pressure is the higher value (Artery Pressure) and Diastolic pressure is the lower value.[4] SBP and DBP provide a comprehensive assessment of the health of the heart. SBP indicates artery stiffness or blockages and DBP represents the narrowing of arteries, therefore, both pressures concern the heart and kidney disorder at different intensities (raised SBP - overall mortality, raised DBP - aortic disease).[4] SBP tends to increase with age and DBP may decrease with the age of 50 years. The applied Antihypertensive medicines simultaneously act over both pressures.[5] Untreated B.P. increases the risk of hypertensive retinopathy, chorioretinopathy and optic neuropathy, because of blood vessels that have swollen, constricted, brittle and bulged to form an aneurysm which further burst to give rise to visual threat [4] Due to hypertension, The major retinal vessels such as the branch retinal vein, central retinal vein, branch retinal artery, and central retinal artery become occluded.[2, 6] The collection of fluid under the retina due to the leaks from the retinal pigment epithelium (RPE) and choroid is commonly named choroidopathy, was initially introduced by Albrecht von Graefe (1866) and named central recurrent retinitis, thereafter a variety of names applied as idiopathic flat detachment of the macula (Walsh et al), central angiospastic retinopathy (Gifford et al), Idiopathic central serous chorioretinopathy (Gass et al-1967) and central serous retinopathy (Straatsma et al).[7] Central serous chorioretinopathy is assumed fourth most common retinopathy followed by ARMD (age-related macular degeneration), DR (diabetic retinopathy) and BRVO (branch retinal vein occlusion) with common complaints of metamorphopsia (micropsia/macropsia), commonly hyperopia and sometimes myopia, central scotoma, decreased contrast sensitivity and colour saturation.[8, 9] The disease is considered as the hyper-permeability of the choroidal capillaries incorporated with retinal pigment dysfunction and finally introduces serous detachment of the neurosensory retina. The possibility of recurrence of the disease is a major issue.[10]

## EPIDEMIOLOGY:

Once the changes in the retina take place next to malignant hypertension, the treatment is unable to improve AV changes, arteriolar narrowing, optic nerve destruction, macular changes, and finally, visual acuity gets diminished. The severe hypertensive retinopathy and arteriosclerotic changes are indicated for coronary disease, peripheral vascular disease, and stroke. The mortality rate with unattended malignant hypertension is 50% in 2 months and 90% in 1 year.[11] The chronic hypertension of stage 2, (SBP > 140 mmHg and DBP > 90 mmHg) is the cause of hypertensive retinopathy with arteriosclerotic changes.[12] According to a study, hypertensive retinopathy is prevalent in African, Americans and Chinese descent and their incidence of rising blood pressure issues increases with age. Men are found more affected in the age group < 45 years old and women are get affected in age groups > 65 years old.[6] In nondiabetic patients, hypertensive retinopathy ranges from 2-17% although this range varies demographically.[13, 14] Pontremoli et al. measured the role of genetic factors and the action of the angiotensin-converting enzyme for hypertensive retinopathy.[15] According to Chatterjee et al., hypertensive retinopathy is more common among Afro-Caribbean people than in European people, and it is more common in women. Genetic factors with certain genotypes also have an important role in the higher risk of hypertensive retinopathy.[16] Smoking has a connection to the seriousness of the malignant hypertensive retinopathy observed by Poulter et al.[17] Renal dysfunction with end-organ damage is a justified reason for hypertensive retinopathy.[18] The increased plasma leptin level makes vascular endothelium damage the property of the individuals having hypertensive retinopathy.[19]

Erden et al.'s research indicate a strong correlation between the severity and duration of hypertension and the occurrence of hypertensive retinopathy[20]. According to Kabedi et al., 83.6% of all hypertension patients had hypertensive retinopathy, and the association between this condition and chronic renal disease was significant.[21]

The age group of 20 to 50 years of males tend to be affected by central serous chorioretinopathy with the symptom of acute or sub-acute central vision loss. The use of endogenous and exogenous (intravenous, cutaneous and nasal route) steroids has the strongest association with CSR. [22-26] The inflammatory choroiditis,

does not prove linkage with infection and is precipitated by glucocorticoids.

Most patients integrate with CSCR, with the age group of 28-68 years. The odds of bilateral illness with RPE loss and choroidal neo-vascularization are higher in individuals having the age of about 50 years or over. [27, 28]

The incidence of CSCR is approximately six times higher in males (9.9/100,000) compared to females (1.7/100,000) with the prevalence in Asian, African American, and Caucasian populations.[29, 30]

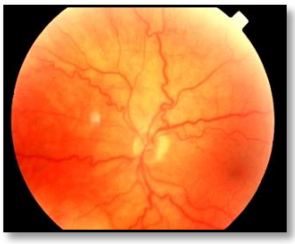
CSCR-affected individuals may have hyperopia type of error or emmetropia as well. Alcohol addiction, pregnancy, prolonged use of antibiotics, untreated hypertension, obstructive sleep apnea and the recurrence of the disease, commonly counted in depression, Cushing syndrome, organ transplantation, systemic lupus erythematosus (SLE), and end-stage renal disease etc. are the common risk factors.[31, 32]

## PATHOPHYSIOLOGY:

Retinal blood vessels have some specific characteristics as they don't have a sympathetic nerve supply, have autoregulation of blood flow and blood-retinal barrier.[33]

1. **HYPERTENSIVE RETINOPATHY**:
   Hypertensive retinopathy has the following levels-

- The vasoconstrictive Phase starts with elevated pressure in the retinal arteriole thus autoregulatory mechanisms give rise to narrowing (vasospasm) of the retinal arteriole for the decrease of blood flow.
- The sclerotic Phase develops due to the continuous increase of blood pressure to give change in retinal vessels, the inner wall gets thickened with hyaline degeneration and the middle layer has hyperplasia, therefore, arteriolar narrowing, arterio-venous crossing changes - thickened arteriole crosses over a venule and the vein turn to, appear dilated and torturous (fig-1) distal to the AV crossing(fig-2), widening and emphasized light reflex (silver and copper wiring) takes place.[1]
- The exudative phase is concerned with intracranial hypertension concludes optic nerve ischemia, papilledema (fig-3) and fibrinoid necrosis of the choroidal arterioles with specific signs of Elschnig's spots - RPE appears yellow, Siegrist's streak - RPE hyperplasia (fig-4) and detachment of neurosensory RPE (these three signs have concern to the choroidopathy) arrises with retinal haemorrhages, hard exudates, retinal ischemia, and necrosis of smooth muscle.[1]



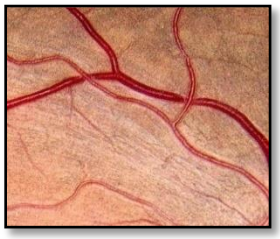


**Fig 1 Dilated& tortuous retinal veins** **Fig 2 AV Crossing**

Retinal haemorrhages are the reaction of the necrotic vessels, if they bleed superficially named flame-shaped haemorrhages and in the deep retina named dot and blot haemorrhages. The cotton wool patches develop due to the ischemia of the nerve fibre layer resulting in fibrinous necrosis and blood vessel luminal constriction (fig-5).

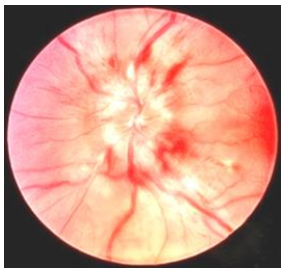


**Fig 3 pappliedema**

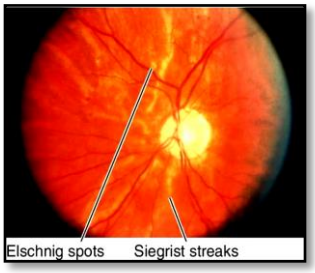



**Fig 4 Elsching spot & Siegrist streak**

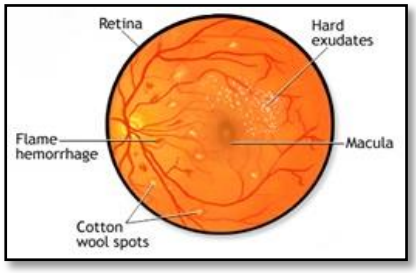



**Fig 5 exudates & haemorrhage**

At the site of haemorrhage, exudates develop due to the lipids deposition. In malignant hypertension, papilledema is the result of haemorrhage and arteriolar ischemia around the optic disc and finally fibrinous necrosis takes place.[34]

## CENTRAL SEROUS CHORIO RETINOPATHY:

In the beginning, RPE dysfunction was assumed for the pathophysiological cause of CSCR (central serous chorio retinopathy) but later on, it was considered a sequel of CSCR, (fig-6) therefore epidemiology, hormonal studies, fluorescein angiography (FA), and optical coherence tomography (OCT) imaging are suggested for confirmed manifestation. The capacity of indocyanine green angiography to stain choroidal tissue allows it to rule out conditions that are crucial for the development of CSCR, such as abnormal dilatation of choroidal veins, increased capillary permeability, and choroidal lobular ischemia.[35-37] The depth imaging through OCT, CSCR patient shows choroid thickening due to the foveal or perifoveal leakage, It points to increased hydrostatic pressure and vascular congestion.[38]

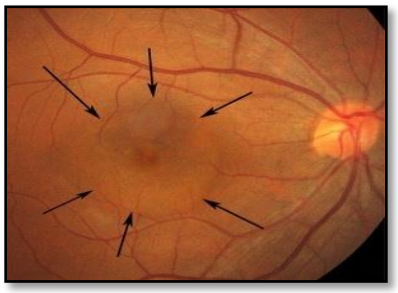


**Fig 6 central serous chorioretinopathy**

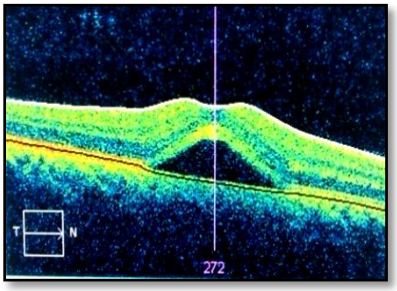



**Fig 7 OCT of central serous chorioretinopathy**

In parts of the choroid affected by CSCR, Prunte and Flammer showed the existence of delayed arterial filling and ischemia followed by capillary and venous congestion. Fifty per cent of eyes may have foveal or perifoveal leakage and any or both of these conditions may be the cause of the increased permeability.[39] When there is acute CSCR, pooling of auto-fluorescence suggests increased metabolic activity of the RPE[40] otherwise in chronic CSCR, secondary RPE damage shows low or absence of auto-fluorescence.

## CLASSIFICATION:

Various classifications are used for the severity of hypertensive retinopathy.

| SCHEIE CLASSIFICATION FOR HYPERTENSION RETINOPATHY (malignant hypertension)[1, 12] | |
|---|---|
| Stage 0 | Without alteration |
| Stage 1 | Undetectable narrowing of an artery |
| Stage 2 | Apparent narrowing of an artery with focal differences |
| Stage 3 | Grade 2 symptoms with retinal haemorrhages/exudates/cotton wool spots/retinal oedema |
| Stage 4 | Grade 3 plus papilleodema |
| **SCHEIE CLASSIFICATION FOR HYPERTENSION RETINOPATHY (chronic hypertension) [1, 12, 41]** | |
| Stage 1 | Light reflex enlargement of arteriole |
| Stage 2 | Grade 1 symptoms with Arteriovenous crossing sign |
| Stage 3 | The copper-coloured light reflex of arteriole |
| Stage 4 | The silver-coloured light reflex of arteriole |
| **KEITH-WAGNER-BARKER CLASSIFICATION (1939)[11]** | |
| Grade 1 | Retinal arteriole constriction that is mild and widespread |
| Grade 2 | Definite focal narrowing of retinal arterioles with AV nicking |
| Grade 3 | Grade 2 symptoms with flame-shaped haemorrhages/cotton-wool spots/hard exudates |
| Grade 4 | Severe Grade 3 retinopathy with papilledema |
| **WONG AND MITCHELL CLASSIFICATION (2004)[42]** | |
| None | --- |
| Mild | arteriolar narrowing, arteriovenous nicking, opacity (copper wiring) of the arteriolar wall |
| Moderate | retinal haemorrhage (blot/dot/flame), microaneurysm, cotton-wool spots, hard exudate |
| Severe | moderate retinopathy with swelling of the optic disc |

A new hypertension classification (2014) based on optical coherence tomography (OCT) and the Keith-Wagner-Barker grading system incorporated with subretinal fluid (SRF) significantly correlated with the best-corrected visual acuity.[43]

- Mild-Moderate Retinopathy
- Malignant Retinopathy without SRF
- Malignant Retinopathy with SRF

The general classification of central serous chorioretinopathy typically involves condition, duration, severity, and associated features-

| No. | CONDITION | OBSERVATION | NAME | DESCRIPTION |
|---|---|---|---|---|
| 1 | Duration of symptoms | --- | Acute CSCR | Symptoms develop suddenly and resolve in a few months |
| | | | Chronic CSCR | Symptoms persist for longer durations and often management required |
| 2 | Associated features | --- | Uni/bilateral | Usually monocular but sometimes binocular involvement |
| | | | Pregnancy | Pregnancy in the postpartum period |
| | | | Corticosteroids | Prolong use of systemic or topical corticosteroid medications |
| | | indocyanine green angiography (ICGA) | Type A | Associated with choroidal vascular hyperpermeability |
| | | | Type B | Associated with choroidal vascular congestion |
| 3 | The pattern of fluid dispersal | FFA or OCT | Focal CSCR | Characterized by a single or few leakage points in the RPE |
| | | | Diffuse CSCR | Involves widespread leakage across the RPE |
| 4 | Subretinal fluid hight | --- | Shallow CSCR | subretinal fluid (SRF) is relatively superficial, closer to the retinal surface |
| | | | Deep CSCR | Thicker accumulation of SRF, extending deeper into the retinal layers |

**SIGNS**:

Hypertensive retinopathy is identified through fundus examination under the following changes[12, 44]

| | | | | |
|---|---|---|---|---|
| 1. | AV defect | Salus's sign | The retinal vein follows an "S" shape orientation passing across the arteriole. | |
| | | Gunn's sign | Tapering of the retinal vein on each side of the AV cross. | |
| | | Bonnet's sign | Retinal vein pooling away from the AV cross. | |
| 2. | Arterial Changes | reduction to 1:3 in the arterio-venous ratio (standard is 2:3). | | |
| | | Alterations to the arteriolar light reflex (named copper or silver wire) | | |
| 3. | Retinal Changes | Retinal haemorrhages | Dot and blot haemorrhages | -Deep retinal layer haemorrhage |
| | | | Flame shaped hemorrhage | -Superficial retinal layer haemorrhage |
| | | Retinal exudates | Hard exudates | -Due to lipid deposition in the retinal layer |
| | | | Soft exudates | -Ischemic property of the nerve fibres introduces soft exudates named Cotton wool spots |
| 4. | Macular Changes | Macular star formation due to accumulation of lipid around the macula. | | |
| 5. | Optic Nerve Changes | Optic disk swelling (hypertensive optic neuropathy) | | |

Malignant hypertension may produce choroidopathy,[1] and mostly concerns young hypertensive patients, initiated with localised pigment epithelium detachment and lasts to the exudative retinal detachment.[12, 45] Elschnig spots, which are hyperpigmented patches in the choroid surrounded by a ring of hypopigmentation, and Siegrist streaks, which are straight hyperpigmented lesions over choroidal arteries, are two of the key features. These are brought on by inadequate choriocapillaris perfusion.

## SYMPTOMS:[44]

**A. Acute malignant hypertension:**

- Ocular pain,
- Headaches,
- Reduced visual acuity.

**B. Chronic hypertension:**

Generally without symptoms but sometimes vision may decrease, although, the specific complaints of CSCR are central vision loss or distortion, micropsia, hyperopia, low contrast sensitivity and colour saturation.[45]

## DIAGNOSIS:

The modern and best way for a differential and proper diagnosis of hypertension and CSCR, are fundus fluorescein angiography (FFA) and optical coherence tomography (OCT) which helps to detect -

- retinal capillary nonperfusion,
- microaneurysm formation, and
- a dendritic pattern of choroidal filling/hypoperfusion (early phase) and the diffuse/sub-retinal leakage (late phase)[12]

Indocyanine green angiography expresses a moth-eaten appearance of the choriocapillaris.[12] Acute CSCR is known as a self-limited condition with the recovery of visual acuity within a couple of months.[10]

Disc leak is often absent from CSCR. Individuals concerning CSCR and other conditions, the leaking regions on FFA can correspond to the hyperreflective areas on the infrared picture in OCT associated with the ink blot pattern on fluorescein angiography, smokestack pattern and minimally enlarging spot.[46, 47]

The optimum BCVA range for CSCR patients lies between 6/6 to 6/60, and Visual loss comes through a hyperopic shift because of the anterior shift of the macular photoreceptors.[48]

Ophthalmoscopic finding advocates round or oval serous macular detachment without haemorrhage, with small, yellow sub-retinal deposits in the area of neurosensory detachment.[49] Sometimes the sub-retinal fluid of grey-white sero-fibrinous exudates may be there.[50]

## MANAGEMENT:

Lowering the B.P. is the first important treatment level for hypertension retinopathy by protecting the ischemic damage to the retina of the eye.[51]

Angiotensin-converting enzyme inhibitors, calcium channel blockers, and diuretics are the most common drugs used in hypertension. Some of the other drugs not commonly used are α-adrenergic blockers, direct vasodilators, and central α2-adrenergic agonists. The popular but less result-oriented ocular treatment is intra-vitreal antibody treatment, which acts on the vascular endothelial growth factor which lowers the macular oedema and retinal haemorrhage.[13, 52]

Fluorescein and indocyanine green (ICG) angiography determine the areas of further treatment.[53] Several wavelengths of pulse laser targets different retinal cells as green (532 nm), yellow (577 nm), and infrared (810 nm) are used to target various retinal cells.[54]

Treatment for CSCR with low-dose aspirin has been investigated.[55] The elevated levels of VEGF resulting from choroidal disease are used to estimate the role of anti-vascular endothelial growth factor (VEGF) medications.[56, 57]

Before undergoing angiography for the focused targets based on hyperfluorescence at the site of RPE detachment/disturbance, Focal argon laser and micropulse diode laser photocoagulation may be necessary as alternative therapy for individuals whose vision deficits are chronic (continue longer than a few months). Focal argon laser photocoagulation may be helpful for patients with focal RPE detachments without the involvement of the fovea; although in such cases patients may develop scotomas that match the symptomatic operation sites.[41]

The surrounding area of the laser treatment activates RPE cells by reducing inflammation and cytokine production by using sub-threshold energy. The applied property for laser energy is "high density and low-intensity" or "sub-threshold micropulse laser" (SML) for specifically target RPE cells.[58, 59]

Chronic CSCR patients are treated with photodynamic treatment (PDT), which increases the verteporfin (photosensitizer) level in the retinal blood vessels and further helps to reduce SRF and chances of improving BCVA takes place.[60]

## COMPLICATIONS:

Hypertension gives rise to the ocular disorders BRAO, BRVO, arterial macro aneurysms and Ischemia, which develop after the occlusion of vessels further formation of neovascularization, vitreous haemorrhage, epiretinal membrane, and tractional retinal detachment takes place. In addition to diabetes, hypertension retinopathy becomes more prompt, although raised blood pressure is

known as a major risk factor for the progression of diabetic retinopathy.[61] Hypertensive optic neuropathy causes chronic papilloedema, and the risk of glaucoma, Cystoid macular oedema and Age-related macular degeneration is also there. The proliferative type of hypertensive retinopathy has also been introduced recently.[12, 44, 62]. The rupture of Bruch's membrane through the laser treatment causes further vision loss and choroidal neovascularization (CNV) and additional focal argon laser photocoagulation is applied with regular follow-up.

## PROGNOSIS:

Arteriosclerotic alterations and symptoms of severe hypertensive retinopathy are reliable markers of an increased risk of peripheral vascular disease, coronary heart disease, and stroke. The arteriosclerotic changes in the retina never recover, so chances of retinal artery occlusions, retinal vein occlusions, and retinal macro-aneurysms are always there. Involvement of most of the retinal changes like AV changes, arteriolar narrowing, and damage of the optic nerve and macula could not be recovered and the visual acuity was reduced.

The increased risk of CSCR concerns the exogenous steroids too either of oral, intramuscular, intranasal, etc. so lowering of the drug potency or discontinuation is suggestive. Lifestyle modification and stress management are also important factors for improvement. H. pylori in the case of atherosclerosis has been studied for the CSCR[63] by librating cytotoxins acts over the choroidal vessels. The long-standing hypertension has concern for the CSCR so the treatment of hypertension should be taken.[64]

## CONCLUSION:

A visible window into the systemic effects of hypertension on the fragile tissues of the eye is provided by hypertensive retinopathy and choroidopathy. It underscores the critical need for effective management and control of blood pressure to prevent irreversible damage to retinal vessels and subsequent vision impairment with the spectrum of retinal change from mild arteriolar narrowing to severe haemorrhages and exudates further mirroring the progression of hypertensive vascular disease. The detection and monitoring of hypertensive retinopathy not only offers insights into the ocular health of individuals but also serves as a sentinel marker for cardiovascular risk. A comprehensive approach that includes regular ophthalmic examinations with the coordination of cardiovascular medical experts is important for preserving both vision and overall health respectively; early intervention and diligent blood pressure management are paramount in mitigating the ocular and systemic consequences of hypertensive retinopathy.

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