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MICROVASCULAR CHANGES IN HYPERTENSIVE RETINOPATHY

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Abstract: Hypertensive retinopathy refers to the spectrum of retinal vascular changes resulting from sustained elevation of systemic arterial blood pressure. Chronic hypertension affects the ocular circulation, leading to retinopathy, choroidopathy, and optic neuropathy, and also predisposes individuals to other vision-threatening conditions such as retinal artery and vein occlusions, retinal artery macroaneurysms, and non-arteritic anterior ischemic optic neuropathy. In addition, hypertension increases the risk of diabetic retinopathy, glaucoma, and age-related macular degeneration and may complicate ophthalmic surgical procedures.

The pathophysiology of hypertensive retinopathy involves autoregulatory vasoconstriction in response to acute blood pressure elevation, followed by progressive structural vascular changes due to chronic hypertension. These changes occur in three stages: the vasoconstrictive phase, characterised by generalised and focal arteriolar narrowing; the sclerotic phase, marked by vascular wall thickening, arteriovenous crossing changes, and characteristic copper and silver wiring; and the exudative phase, resulting from breakdown of the blood-retinal barrier with haemorrhages, cotton wool spots, and hard exudates.

Early identification of hypertensive retinopathy is clinically important, as retinal findings reflect the severity and duration of systemic hypertension and are associated with increased cardiovascular morbidity and mortality. Effective management primarily involves prompt and sustained blood pressure control, along with multidisciplinary evaluation to prevent ocular complications.

Keywords: Hypertensive retinopathy; Systemic hypertension; Retinal vascular changes; Fundus examination; Ocular complications.

Introduction:
The term hypertensive retinopathy encompasses all funduschanges resulting from elevated systemic arterial blood pressure. High blood pressure (HTN) can lead to three main types of ocular damage: choroidopathy, retinopathy, and optic neuropathy. Hypertension can manifest in various ocular complications, including retinopathy, choroidopathy, and optic neuropathy. Furthermore, it serves as a predisposing factor for other sight-threatening conditions such as branch retinal artery occlusion (BRAO), central retinal artery occlusion (CRAO), branch retinal vein occlusion (BRVO), central retinal vein occlusion (CRVO), retinal artery macroaneurysms, and non-arteritic anterior ischemic optic neuropathy (NAION)[1]. Additionally, hypertension elevates the risk of diabetic retinopathy, glaucoma, and age-related macular degeneration. Moreover, it increases the likelihood of experiencing suprachoroidal hemorrhage during ophthalmic surgery.

Pathophysiology and Clinical features:
When blood pressure remains elevated for an extended period, it triggers vasospasm through autoregulation. This results in increased blood flow to specific organs due to the heightened pressure. Consequently, blood vessels attempt to regulate this excessive blood flow by undergoing vasoconstriction, thereby moderating the supply to these organs. This mechanism, known as autoregulation, involves the vessels constricting in response to acute spikes in blood pressure. Chronic elevation of blood pressure leads to atherosclerosis, characterized by the thickening of blood vessels.
The phases of hypertensive retinopathy can be divided into-

1.The vasoconstrictive phase:
During the vasoconstrictive phase in response to acute conditions, autoregulation occurs within the blood vessels, leading to vasospasm or constriction. Elevated luminal pressure or blood pressure triggers local autoregulatory mechanisms, resulting in narrowing of retinal arteries and vasospasm, thereby reducing blood flow. This often manifests as generalized vasospasm. Typically, the normal artery-to-vein ratio (AV ratio) in the fundus is two to three. However, in cases of hypertensive retinopathy, this ratio deviates significantly from the norm, with veins appearing thicker and arteries narrowed due to vasospasm, indicating the initial phase of the condition. Following generalized constriction, further thinning may occur, particularly in specific vessels, termed as focal vasospasm or vasoconstriction. In severe cases, sections of the artery may even become nearly occluded due to vasospasm.

2. Sclerotic phase:
During this phase, attention is directed towards the layers of blood vessels, including the tunica intima, tunica media, and tunica adventitia. Chronic elevation of blood pressure leads to thickening of the tunica intima, while the smooth muscle cells in the tunica media undergo proliferation, causing vasoconstriction and medial hyperplasia. Additionally, the adventitia and middle portion are replaced by hyaline tissue. Consequently, vessel narrowing reduces blood flow to the retina and choroid. As vessels thicken, they begin to reflect light directed onto the retina. This reflection obscures the inner blood flow column, making only the vessel walls visible. With light reflection, these walls exhibit characteristics resembling copper wiring and silver wiring. During the initial thickening phase, vessels exhibit yellowish reflections, known as copper wiring. As the Sclerotic phase advances, extensive thickening causes widespread light reflection, resulting in a whitish appearance termed silver wiring. (Fig 1) Hypertensive retinopathy leads to arteriovenous (AV) crossings, causing morphological changes in veins, known as AV signs. These include the gun signs, bonnet signs, and Salu'ssigns (fig 2).

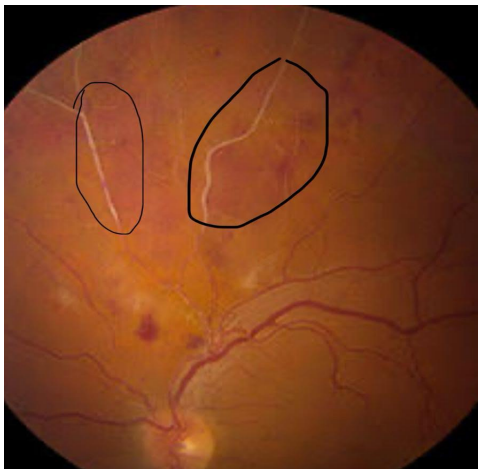

Figure 1: Advanced thickening of blood vessels leads to widespread light reflection, giving rise to a whitish appearance(source: https://quizlet.com/575747422/hypertensive-retinopathy-flash-cards/)

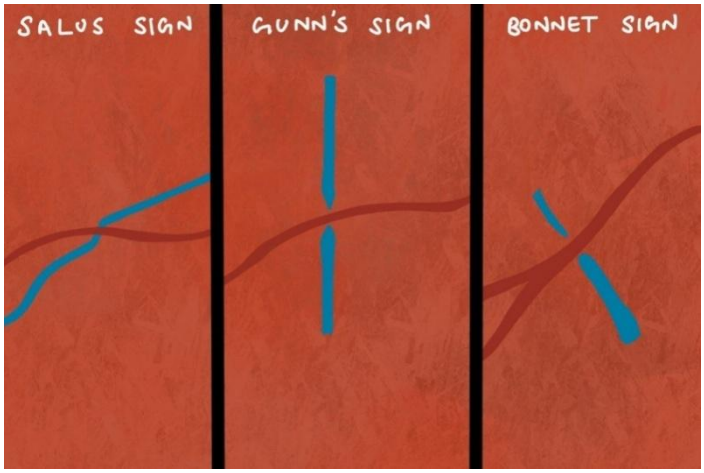

Figure 2: a) Salu's signs(the artery thickens, the vein deflects sideways instead of following a straight path), b) gun signs (When thickened arteries cross over veins, the veins narrow at the points of compression by the arteries), c) bonnet signs (when the artery crosses the vein. In this scenario, the distal portion of the vein dilates).
(source: https://www.firstclassmed.com/articles/2021-hypertensive-retinopathy)

3.The exudative phase:

The third stage of hypertensive retinopathy is the Exudative phase. Here, chronic elevation of blood pressure and endothelial changes disrupt the blood-retinal barrier. This disruption leads to plasma and blood leakage, termed exudation. This phase may feature dot blot or flame-shaped hemorrhages and cotton wool spots, resulting from fibrinous necrosis and narrowing. In advanced stages, organized hard exudates resembling a star may also be observed.

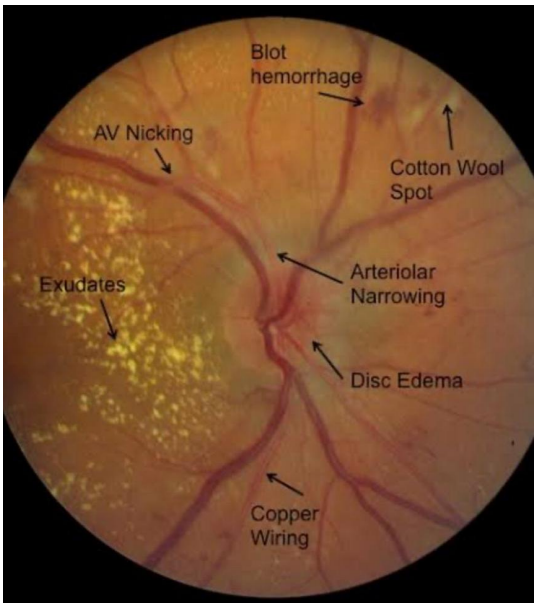

Figure 3: Clinical findings in Hypertensive Retinopathy
(source: https://www.firstclassmed.com/articles/2021-hypertensive-retinopathy)

Management:

Underlying cause by normalizing blood pressure. Early detection of hypertensive retinopathy should prompt physicians to initiate blood pressure control. Fundusfindings often improve with systemic condition treatment. For mild cases, blood pressure control through regular monitoring is recommended. In moderate cases, referral to a physician is necessary to rule out other associated factors like diabetes mellitus and cardiovascular abnormalities, with routine BP control and monitoring. Severe hypertensive retinopathy requires urgent treatment and referral due to its strong association with mortality. Monitoring of other systems such as renal, cardiovascular, and neurological for signs of target organ damage is crucial. Eye care professionals should be knowledgeable about the signs and symptoms of

hypertensive retinopathy and chorioretinopathy as they are linked to the patient's overall health and mortality in both short and long term.

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