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A Short Note on Hypertensive Retinopathy

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Editorial

The cardiovascular, renal, cerebrovascular, and retina systems are all affected by poorly managed hypertension (HTN). Target-organ damage is the term for the harm to these systems (TOD). Choroidopathy, retinopathy, and optic neuropathy are three types of ocular injury caused by HTN. High blood pressure damages the retinal vessels, resulting in hypertensive retinopathy (HR). There is substantial evidence that hypertensive retinopathy is a predictor of TOD-related systemic morbidity and mortality. According to Erden the severity and duration of HTN are associated to the rise in the incidence of retinopathy [1,2].

Other factors, in addition to essential and secondary hypertension, have a role in the development of hypertensive retinopathy. Hypertensive retinopathy is more common among Afro-Caribbeans than in Europeans, and it is more common in women than in men. Certain genotypes linked to an elevated risk of hypertensive retinopathy can also be attributed to genetic factors. Pontremoli investigated the genetic factors linked to hypertensive retinopathy and discovered that deletion of the angiotensin-converting enzyme allele is associated with a higher risk of hypertensive retinopathy development. According to Poulter research's smoking has a substantial link to severe or malignant hypertensive retinopathy.

According to Erden the severity and duration of hypertension are directly linked to the occurrence of hypertensive retinopathy. In their study, 66.3 percent of the participants had hypertensive retinopathy. According to Kabedi hypertensive retinopathy affects 83.6 percent of all hypertension patients, with chronic renal disease being the most important predictor of severe hypertensive retinopathy. According to Del Brutto study's hypertension retinopathy grade 1 was found in 37% of hypertensive patients, and grade 2 hypertensive retinopathy was found in 17%.

The following characteristics distinguish retinal blood vessels from other blood vessels: Insufficient sympathetic nerve supply Blood flow autoregulation Blood-retinal barrier is present. As a result, an increase in blood pressure (BP) is directly conveyed to the arteries, which constrict at first. However, an increase in blood pressure overrides this compensatory tone, resulting in injury to the muscle layer and endothelium.

Stages of hypertensive

The stages of hypertensive retinopathy are as follows:

Phase of vasoconstriction: The local autoregulatory systems kick in at this phase. This results in vasospasm and narrowing of the retinal arterioles, as seen by a decrease in the arteriole to venule ratio (normal = 2:3). As damaged arterial segments cannot narrow, localised arteriolar constriction develops in older patients with arteriosclerosis.

Phase of scleroderma: A persistent rise in blood pressure induces the following changes in the vessel wall:Thickening the intima layer, Hyperplasia in the media, Hyaline degeneration of the arteriolar wall

This causes severe arteriolar constriction, changes in arteriovenous (AV) crossing, and a widening and accentuation of the light reflex (silver and copper wiring). Because the vessels share a shared adventitious sheath, AV crossing alterations occur when a thicker arteriole crosses over a venule and compresses it. Distal to the AV crossing, the vein seems dilated and tortuous.

Phase of exudation: The rupture of the blood-brain barrier and leaking of blood and plasma into the vessel wall, affecting the autoregulatory systems, is seen in patients with extremely elevated blood pressure. Retinal symptoms such as retinal haemorrhage (flame-shaped and dot blot), hard exudate development, smooth muscle cell death, and retinal ischemia occur at this stage (cotton-wool spots).

Hypertension with malignancy: Ischemia and edoema of the optic nerve are caused by severe intracranial hypertension (papilledema). Fibrinoid necrosis of choroidal arterioles also occurs, resulting in choriocapillary segmental infarction. Elschnig's spots are the result of this. Siegrist's streak occurs when the underlying retinal pigment epithelium (RPE) appears yellow. Hyperplasia of the RPE over choroidal infarct RPE detachments in the neurosensory system. Choroidopathy is the medical term for these symptoms [3-5].

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Conflict of Interest

None

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