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RESEARCH ARTICLE

OCULAR CHANGES IN HYPERTENSION

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ABSTRACT

Hypertension is a systemic medical condition that has a range of effects on the eye. Hypertension triggers a series of pathophysiological ocular modifications affecting significantly the retinal, choroidal, and optic nerve circulations that result in vaso- occlusion, arteriolar macroaneurysms, embolic events and optic neuropathy. Several grading systems have been proposed that attempt to correlate observed ocular hypertensive changes with systemic vascular disease severity, morbidity and mortality.

INTRODUCTION

Systemic hypertension affects the arteries, veins, choroid, and the optic nerve, depending on the chronicity and severity of the disease. Ocular changes in malignant hypertension can be striking, with optic neuropathy, choroidopathy and retinopathy.[1] Elevation of the systemic blood pressure causes both focal and generalized constriction of the retinal arterioles mediated by autoregulation. A prolonged duration of hypertension can lead to a breakdown of the inner blood-retinal barrier, with extravasations of plasma and RBC. Retinal hemorrhages, cotton-wool spots, intraretinal lipid, and in severe cases macular star and closure of the retinal capillaries can be seen.

When the choroidal vessels are severely affected, fibrinoid necrosis of the choroidal arterioles can cause occlusion of choriocapillaries.

The optic nerve head can also be involved.[2]

Pathophysiological Changes In Hypertensive Ocular Disease

1. Hypertensive choroidopathy
2. Hypertensive retinopathy
3. Hypertensive optic neuropathy

Hypertensive Choroidopathy

The hypertensive choroidopathy is divided into three phases, i.e. acute ischemic phase, chronic occlusive phase, and chronic reparative phase[3]. Vasoconstriction of the choroidal arterioles is the initial primary response of the choroidal vasculature to severe hypertension, influenced by sympathetic tone and angiotensin II. In the chronic occlusive phase,the choroidal arteries and arterioles show hyperplastic changes with fibrinoid necrosis including overlying RPE. In the chronic reparative phase, recanalization of the occluded choroidal arteries begins. The overlying RPE shows focal depigmentation. In the early phase of choroidopathy, the fundus exhibits pale white or reddish patches of outer retina (Elschnig's spots) corresponding to areas of hypoperfusion of the underlying choriocapillaries which leak fluorescein dye diffusely in late phases of the angiogram. Other lesions are focal posterior pole serous detachment of retina, macular star and mild arterial constriction.

Hypertensive Retinopathy

Hypertensive retinopathy represents the ophthalmic findings of end-organ damage secondary to systemic arterial hypertension. It can be divided into the following four phases:

1. Vasoconstrictive phase
2. Sclerotic phase
3. Exudative phase
4. Complications of the sclerotic phase.

In the vasoconstrictive phase, the elevated blood pressure stimulates pliable and non-sclerotic retinal blood vessels to increase their tone by autoregulation. This manifests in focal and diffuse constriction of arteries. [4, 5]

In sclerotic phase, if the blood pressure in the vasoconstrictive phase remains elevated over a period of time, sclerotic changes develop. Clinically the features are sclerosis of vessel wall,

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vascular tortuosity and an increased angle of branching of the arteries and arterioles. Generalised arterial narrowing with or without localised constriction is a good indicator of hypertension. Arteriovenous crossing changes can be evaluated when the artery passes anteriorly over the vein but not the vice versa. Mild moderate, and branch vein occlusion are the three grades of this physical sign. In mild stage of arteriovenous nicking, there is a slight deflection of the vein beneath the artery, with early concealment of the vein. In moderate stage, the vein tapers under the artery, with apparent constriction and deflection which is known as Gunn's sign. The vein may be mildly distended for some distance peripheral to the crossing. In branch vein occlusion, hemorrhages and exudates are seen distal to the arteriovenous crossing. Sclerosis of vessel wall is divided into three grades-mild increased light reflex, copper wiring and silver wiring. Arterial tortuosity occurs in prolonged hypertension. Because of the intraluminal pressure, fibrosis of muscle fibres occurs, resulting in an increase in the length of the arteries. The angle of arterial branching is related to elevated blood pressure. A mild branching angle between the branching arteries is from 45^0 to 60^0, a moderate branching angle is from 60^0 to 90^0, and a severe angle is over 90^0.[2]

Exudative phase may accompany or follow hypertensive choroidopathy or the vasoconstrictive/sclerotic phase of hypertensive retinopathy. One of the earlier signs of exudative retinopathy is the appearance of small linear or flame shaped hemorrhages, mostly in the nerve fiber layer in the peripapillary region. Hemorrhages in the nerve fiber layer of the retina extend along axons of the ganglion cells and so appear as linear. When the bleeding occurs in the deeper layers of the retina, the spread of extravasated blood is limited by the processes of Muller's cells, and so the hemorrhages assume an oval outline in the forms of blot and dots. The hemorrhage may break through the internal limiting membrane and occupy a subhyaloid location, presenting as a boat-shaped hemorrhage in the posterior pole. Hard, waxy exudates are most frequently scattered in the posterior pole. Exudates may be arranged in a star-shaped configuration radiating from the macular area along Henle's nerve fibre layer, or in a circular fashion, known as circinate retinopathy. Cotton-wool spots in the nerve fiber layer of the retina indicate retinal ischemia. Disruption of the blood-retinal barrier in the exudative phase of hypertensive retinopathy can be quantitated by vitreous fluorophotometry.[6] Complications of retinal hypertensive arteriosclerosis are central or branch retinal vein occlusion, macroaneurysm, epiretinal membrane, macular edema[7], retinal neovascularization, vitreous hemorrhage and tractional retinal detachment.

Hypertensive Optic Neuropathy

The pathophysiology lies in delay in axoplasmic transport and an accumulation of axoplasmic components in the region of the lamina retinalis and lamina choroidalis. This results in swelling of the axons at the optic nerve head leading to ischemia.[8] It includes optic disc edema, optic atrophy and ischemic optic neuropathy.

Classification of Hypertensive Retinopathy[9]

Grade 1

- Generalized arteriolar attenuation
- Broadening of arteriolar light reflex
- Concealment of vein at A-V crossings.

Grade 2

- Severe generalized and focal arteriolar constriction
- A-V crossing changes (Salus sign).

Grade 3

- Copper wiring of arterioles
- Venous banking distal to A-V crossing (Bonnet's sign)
- Venous tapering on either side of the crossing (Gunn's sign)
- Right angled deflection of veins
- Flame shaped hemorrhages, cotton-wool spots, hard exudates.

Grade 4

- All changes of grade 3, with silver wiring of arterioles
- Disc edema.

CONCLUSION

Systemic hypertension is associated with severe morbidity. The brain, heart, and the eyes are the likely organs to be affected. Therefore, control of hypertension is essential for both good visual function and long term survival. The general measures include weight control, sodium restriction, exercise, relaxation, control of lifestyles, antihypertensive drugs and proper health education.

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