BILATERAL SEROUS RETINAL DETACHMENT DUE TO HYPERTENSIVE CHORIORETINOPATHY

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ABSTRACT

Hypertension can affect the retinal vasculature and is related to a variety of retinal manifestations. We present an atypical case of bilateral serous retinal detachment due to high systemic blood pressure. A 50-year-old lady presented with acute visual loss and bilateral serous retinal detachment. Systemic blood pressure was 220/120 mmHg. Serous retinal detachment decreased after the blood pressure was stabilised. In cases of serous retinal detachment, different systemic and ocular diseases should be ruled out. Uncontrolled high blood pressure causes aggressive bilateral retinal changes. Hypertension must be under control to improve visual outcome.

KEYWORDS
Hypertension, Retinal Detachment, Fluorescein Angiography.


INTRODUCTION

One of the leading causes of death in the industrialised world today is cardiovascular disease. Hypertension is one of the important risk factors for cardiovascular disease. Hypertension has an important role to play in the pathogenesis of several ophthalmic conditions that cause visual impairment. Studies by Hayreh have shown that the effects of hypertension on retinal, optic nerve head and choroidal circulation produce distinct clinical manifestations: hypertensive retinopathy, hypertensive optic neuropathy and hypertensive choroidopathy.¹,²

CASE REPORT

A 50-year-old lady, with no previous known history of any systemic diseases, presented to our OPD with history of headache and gross diminution of vision in both eyes of week duration. On examination, the patient appeared dull and disoriented and her visual acuity was just hand movements in both eyes and the pupils were sluggish. On slit-lamp examination, anterior segment was normal and no vitreous cells. Fundus examination showed blurred disc margins, multiple dull white spots throughout the posterior pole, varying in size from pinpoint to half the disc diameter. There were scattered nerve fibre layer haemorrhages and shallow retinal detachment involving the macula. (Figure 1.)

B-scan examination showed, shallow serous retinal detachments involving the posterior poles with choroidal thickening. (Figure 2.)

Fundus fluorescein angiography showed multiple focal leaking spots, in the arteriovenous phase, corresponding to the dull white lesions seen on fundus picture. They were more concentrated around major retinal arterioles and their branches posteriorly. During the late phases, these spots increased in size and fused into bigger patches. There was pooling of dye in the serous detachments by late phases. (Figures 3 and 4.)
With the reattachment of retina, more of retinal haemorrhages are visible, linear white spots have decreased and few cotton wool spots are visible with arteriolar attenuation and venous dilatation, thus supporting the hypertensive origin of retinopathy. Temporal to fovea polymorphic RPE atrophic spots (Elschnig spots) are seen with pigmentary changes.

DISCUSSION
The potential causes of serous retinal detachment include – inflammatory, uveitic, post-surgical, neoplastic and vascular. Complete evaluation and work-up should be done to rule out different potential causes of serous retinal detachment.

An important differential diagnostic possibility in this case was Vogt-Koyanagi-Harada (VKH) disease. Fluorescein angiography in VKH shows multiple pinpoint leaks with increase in fluorescence in late phases. There may be vitreous cells and extra ocular integumentary and neurological manifestations. Absence of any of these features and presence of high blood pressure ruled out the possibility of VKH.

In the present case, the linear white lesions seen in the fundus are described by Hayreh as Focal Intraretinal Periarteriolar Transudates (FIPTs). 1, 3 These are caused by dilatation of terminal arterioles. Occlusion of terminal arterioles causes cotton wool spots which are inner retinal ischaemic spots. These terminal arteriolar changes are seen in acute phase of malignant hypertension.

In malignant hypertension, hypertensive choroidopathy occurs due to endogenous vasoconstrictor agents. 4 Choroidopathy lesions include – acute focal RPE lesions, RPE degenerative lesions and serous retinal detachment. Elschnig spots seen in the temporal fundus, after the retinal detachment has resolved, are due to chronic RPE degenerative changes due to choroidal ischemia.

Cases of serous retinal detachment due to hypertensive choriororetinopathy have been previously reported related to malignant hypertension5, pre-eclampsia6 and eclampsia.7

In summary, we report this case of bilateral serous retinal detachment due to hypertensive chorioraretinopathy which resolved after blood pressure was stabilised. Every ophthalmologist should recognise the ocular manifestations of potentially life-threatening systemic diseases.

REFERENCES