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IP International Journal of Ocular Oncology and Oculoplasty

Journal homepage: https://ijooo.org/

Original Research Article

Clinical study and management of various ocular manifestations in hypertensive patients at tertiary eye care centre in South India

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ARTICLE INFO

Article history:
Received 16-09-2021
Accepted 16-10-2021
Available online 07-02-2022

Keywords:
Hypertension
RVO
RAO
AION
OIS
RAM
CKD
SLE
PRE Eclampsia

ABSTRACT

Purpose: Aim of our study is to study the various ocular manifestations and management in hypertensive patients.

Materials and Methods: This was retrospective interventional study conducted over a period of 5 years from January 2016 to December 2020.

All patients of all age group having primary and secondary hypertension, referred from physicians for screening were included.

Exclusion criteria : Patients with associated diabetes mellitus were excluded.

421 patients who presented to our retina clinic underwent detail ocular and systemic examination. BCVA, slit lamp examination, fundus examination, B scan, OCT, FFA and documentation done in all cases. Patients presented with various ocular manifestations were managed based on the clinical status. Consent was taken for the management.

Results: Patients with ischemic retina showed poor outcome, patients with early presentation showed good outcome.

Conclusion: Hypertension is one comorbidity which is responsible for eye problems, need frequent screening, fundus examination is non-invasive procedure which helps in detecting the vascular changes early.

1. Background

Hypertension is one the common risk factor for ocular diseases like retinal vein occlusion, anterior ischemic optic neuropathy, retinal arterial macro aneurysm and retinal emboli. Hypertension causes vascular injury and increases the risk of cerebral and cardio vascular events. Essential hypertension was found in 62%, secondary hypertension in 29% and PET in 9% of cases.[1] Males are predominantly affected. The relationship between eye disorders and systemic hypertension has severe impact on society, management depends on the proper evaluation of the patient to prevent blindness. Frequent fundus examination is very essential part of the examination in all hypertensive patients. Hypertension can cause direct or indirect damage to the eye.

2. Introduction

High blood pressure effects many organs in the body like heart, kidneys, brain and eye. In eye retinal, choroidal and optic nerve circulation undergo pathophysiological changes and cause hypertensive retinopathy, choroidopathy and neuropathy. Hypertensive retinopathy predicts target organ damage.[2]

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https://doi.org/10.18231/j.ijooo.2021.080

2.1. Patients and methods

421 patients presented to our retina clinic were examined in detail and managed based on clinical condition. Patients who presented with history of hypertension and showed normal fundus examination were advised to control blood pressure and review after one year. Patients were examined in detail.

Table 1: Gender

Male	256 (60.8%)
Female	165(39.1%)

Table 2: Age

Male	**No of patients**	**percentage**
25-35	41	16.01%
35-45	78	30.46%
45-55	71	27.73%
Above 55	66	25.78%

Female	**No of patients**	**Percentage**
25-35	28	16.96%
35 -45	67	40.60%
45-55	41	24.84%
Above 55	29	17.57%

Various ocular symptoms noted in hypertensive patients

1. Blurring of vision
2. Sudden loss of vision
3. Amaurosis fugax
4. Redness

Various clinical manifestations in hypertensive patientsTable 3

2.2. Acute brvo

1. STBRVO-59 (59%)
2. ITBRVO- 41 (41%)

2.3. Crvo

1. Ischaemic-39 (58.20%)
2. Non ischaemic-28 (41.79%)

Management done based on the clinical presentation. Patient underwent detailed systemic examination to exclude other systemic disorders.

All acute retinal vein occlusions managed with anti vegf injections after documentation.

Patients with ischaemic component underwent PRPC, patients with vitreous haemorrhage, TRD and Combined retinal detachment underwent surgical procedures after explaining the prognosis.

Table 3:

Clinical condition	**Number of patients**	**Percentage**
Branch retinal vein occlusion (BRVO)	100	(23.75%)
CRVO	67	(15.91%)
CRAO	23	(5.46%)
BRAO	8	(1.90%)
AION	81	(19.23%)
OIS	16	(3.80%)
RAM	9	(2.13%)
IPCV	11	(2.61%)
Grade 1 hypertensive retinopathy	10	(2.37%)
Grade 2 hypertensive retinopathy	21	(4.98%)
Grade 3 hypertensive retinopathy	11	(2.61%)
Grade 4 hypertensive retinopathy	7	(1.66%)
Hypertensive choroidopathy	3	(0.71%)
Vitreous haemorrhage	19	(4.51%)
Vitreous haemorrhage plus TRD	11	(2.61%)
Combined retinal detachment	9	(2.13%)
Sub conjunctival haemorrhage	3	(0.71)
Central serous chorio retinopathy	2	(0.47%)
Normal Fundus	10	(2.37%)

Patients with other conditions were advised to control blood pressure and have low salt diet. All patients were advised to consult physician for regular check-up of blood pressure and continue anti-hypertensive medication.

3. Discussion

Systemic hypertension causes vascular changes in retina leading to hypertensive retinopathy, uncontrolled blood pressure causes various ocular manifestations like vascular occlusions, choroidopathy and anterior ischaemic optic neuropathy.

Various stages of retinopathy have been described in literature like vaso constriction of vessels, exudative stage, sclerotic stage and late sequelae. Various classifications are proposed based on the vascular changes.[3] Liebreich first described hypertensive retinopathy in 1859. Since then, many researchers have proposed numerous classification schemes, most notably, the Keith-Wagener Barker (KWB,), the Scheie, and the Modified Scheie systems.

Definition of normal blood pressure according to Joint National Committee is less than 120/80 mm of Hg. If there is elevated blood pressure on at least two chronologically separate visits it is considered as hypertension.[4]

Retinal vascular changes occur in response to both acute and chronic elevated blood pressure, and often affect the retina and choroid. Ophthalmic vascular changes may be warning signs of organ damage associated with HTN, ophthalmologist play an important role in diagnosing and managing hypertensive patients.[4]

All patients in our study were referred to cardiologist for cardiac evaluation. Hypertensive heart disease was noted in 10% of our patients, 4% of patients were presented after angioplasty with blurred vision due to acute vascular occlusion, 3% presented after CABG with ocular problem like sudden loss of vision and remaining 3% had ischaemic heart disease.

Pregnancy Induced Hypertension (PIH) effects both mother and foetus. Most common symptoms are blurring of vision, photopsias, scotomas, and diplopia. Ocular involvement includes conjunctival vascular anomalies, hypertensive retinopathy, exudative retinal detachment, vitreous and pre-retinal haemorrhages, ischemic optic neuropathy and hypertensive choroidopathy.[5] In our study number of patients with PIH were 21 young females, most of them were primi gravida with uncontrolled blood pressure, all these patients were referred from maternity hospital for screening the ocular changes in fundus.

One third of patients was normal without any ocular changes, all patients were screened in every trimester, 5 patients showed malignant hypertensive retinopathy with disc oedema, macular oedema, congested venules and retinal oedema. Three patients showed exudative retinal detachment. Two patients presented with amaurosis fugax. Advised strict control of blood pressure and to consult physician.

Permanent blindness rarely occurs in pre-eclampsia; blindness may be due to primary ocular problem like optic atrophy or permanent macular change causing visual loss or cerebrovascular lesion. Termination of pregnancy helps in rapid resolution of retinal changes and complete visual recovery. In our study patients where exudative retinal detachment noted were treated with magnesium sulphate in patients with eclamptic seizures, magnesium sulphate acts through central anti convulsant action and anti-hypertensive drugs to control blood pressure, no patient in our study were advised termination of pregnancy.

Retinal vein occlusions (RVOs) second most common cause of retinal vascular disease after diabetic retinopathy, with a prevalence between 1% -2% in patients older than 40 years of age. Atherosclerosis and hypertensive changes cause endothelial dysfunction and thrombocyte activation leading to branch retinal vein occlusion (BRVO).[6] Mean platelet volume (MPV) considered as prognostic biomarker BRVO patients with hypertension.

Number of injections required were more than three in some cases, ischemic vascular occlusions were not much benefitted with the anti vegf injections. Prognosis was poor in ischaemic vascular occlusion patients. After monthly doses for 3 months of anti vegf injections, patients were followed regularly every 3 months to check the macular status and visual acuity.

One third of the non-ischaemic patients were progressed to ischaemic vascular occlusions and presented with vision drop. BRVO near to disc showed ischaemic component more. All these patients underwent FFA and OCT to study the perfusion status of retina and macula. Patients with more than 5 DD of CNPA areas were treated with laser therapy, sectoral pan retinal photo coagulation done and anti vegf injected. Six patients showed enlarged FAZ and also abnormal foveal contour suggestive of ischaemic maculopathy. Patients were explained regarding prognosis and tried anti vegf injection. Two patients showed improvement in visual acuity of one line where as other four patients did not show any response at that stage.

Response of anti vegf in ischaemic vascular occlusions is limited, even repeated injections showed no improvement in visual acuity, probably extinguished retinal dysfunction due to ischaemia.

CRVO also managed with anti vegf injections. Ischaemic CRVO presented with multiple cotton wool spots and massive macular oedema. NVD was noted in most of the ischaemic CRVOs. NVG also developed in these patients, PRPC done in clear media cases, cases where media hazy due to raised IOP were injected anti vegf followed by PRPC. Patients with no light perception underwent cyclo destructive procedure to reduce IOP. Patients with NVG advised topical steroids, cycloplegics and anti-glaucoma medication. Long-standing hypertension can result in the development of compensatory shunt vessels and arteriovenous nicking (impeded circulation in the retina).[7]

Young adults who developed NVI and NVA were treated with intra cameral anti vegf, next day after injection if media cleared PRPC done.

Painful loss of vision indicates high IOP and the development of rubeotic complications of retinal vein occlusion (classically within 6 weeks). New blood vessels grow across iris and angle obstructs aqueous outflow.

CRAO and BRAO cases were evaluated thoroughly to exclude carotid artery stenosis, acute cases were managed by doing digital massage and tablet acetazolamide was given, patients with transient attacks recovered vision after 24 to 48 hours. Different treatment modalities were tried in our institute, including medical therapies, laser embolysis/embolectomy and invasive surgical procedure like pars plana vitrectomy with induction of PVD (posterior vitreous detachment) for reperfusion of retina but success is always limited in these cases.

Pars plana vitrectomy in these cases is to displace the emboli, PVD induction lead to the release of abnormal adhesion at the level of the CRA. Sometimes the hyaloid

will be tightly adherent to the disc, it will take several attempts to detach from disc. We tried in few cases at our centre in patients who presented with in one week, outcome was not good in all cases, induction of PVD was not easy in many cases, after PVD induction also the prognosis is poor. I think this procedure should be tried in cases where emboli are visible. Other manoeuvres described in literature are active aspiration over the disc creates a negative suction force that can open the collapsed central retinal artery lumen or dislodge a thrombus.[8] PVD induction will also dislodge a thrombus or an embolus. Prognosis in cases of CRAO depends on the time of presentation, early presentation of patients will have chance in recovery of the retina.

Patients with dense vitreous haemorrhage underwent simple pars plana vitrectomy, patients with TRD and Combined underwent vitrectomy, membrane peeling, endo laser and silicone oil tamponade. Once the retina status was stable silicone oil removal done. Three patients who developed inferior break while membrane peel showed elevated retina with macula off. Risk intra operative bleed and retinal breaks were noted in few patients.

One patient developed retinal break after injecting PFCL to stabilize retina for membrane peeling, after break there was profuse intra operative bleed which caused hazy media to further continue the procedure. Retinal break was due to jet flow injury by PFCL at the area of ischaemic retina. Endo laser done to the break, bleeding controlled and outcome was better due to superior break which was sealed and retina attached.

Various surgical procedures like radial optic neurotomy, arteriovenous sheathotomy, chorioretinal venous anastomosis and cannulations of branch retinal venules are described in literature, we did not try in our institute.[9]

Systemic diseases cause decreased perfusion to the optic nerve head secondary to microvascular compromise might increase the patient's risk of NAION. Factors like hypertension, diabetes, and hypercholesterolemia. Other risk factors noted in the literature are nocturnal hypotension, smoking, obstructive sleep apnea, anemia, hypercoagulable states, disc drusen, ocular and nonocular surgery, and migraines.[10] Crowded disc have high risk for AION due to ischaemia of disc micro vessels. Clinically we need to differentiate AION from malignant hypertensive retinopathy properly to manage. Malignant hypertensive retinopathy causing optic nerve head edema occurs usually when the median blood pressure is 190 mm of Hg. Management is to control the blood pressure to relieve ischaemia of micro vessels and vasogenic oedema.

Ocular ischaemic syndrome (OIS) is a hallmark of carotid insufficiency causing chronic hypo perfusion of the arterial supply to the eye. Detailed ophthalmic and systemic workup is required in all patients with ocular ischemia to look for systemic associations such as carotid or coronary occlusive disease, atherosclerosis, hypertension and diabetes mellitus.

OIS can present like non ischaemic CRVO and anterior segment ischaemia with delayed presentation. Anterior segment ischaemia causing pain due to congestion of ciliary body needs medical management whereas patients with macular oedema in OIS were treated with anti vegf intra vitreal injections, patients treated for macular oedema showed good prognosis in vision and also there was marked reduction of central macular thickness.

OIS can present as early manifestation of carotid artery occlusion without any systemic major vascular events. On FFA, the choroid normally fills completely within five seconds of the appearance of the dye in the choroidal arteries. The characteristic findings of delayed choroidal perfusion and non-filling of retinal vessels on FFA is considered as most specific angiographic sign of ocular ischemic syndrome.[11]

Apart from ocular investigations other systemic imaging techniques required for diagnosis are Carotid duplex ultrasonography and MRA or CTA is required. If the results of both tests are informative carotid arteriography may be omitted. If results are inconclusive conventional digital subtraction angiography should be done for confirmation of the diagnosis.[12]

Retinal arterial macro aneurysm (RAM), IPCV were other manifestations reported in hypertensive patients, all these patients were treated with anti vegf injections to reduce the macular thickness and also the angiogenesis. Polyps which were identified on FFA were treated with laser therapy to stop the leak. Anti vegf helps in reducing the size of haemorrhagic PED.

Patients with massive sub conjunctival haemorrhage were advised cold compresses and strict control of blood pressure. Patients with serous PED and Central serous chorioretinopathy (CSCR)were managed with topical Nevanac eye drops, but no response noted, anti vegf tried in serous PED, patient showed no response to the anti vegf injections.

Impaired circulation in the choroid can cause CSCR as complication of hypertensive retinopathy. Hypertensive choroid vasculature is more vulnerable for the systemic changes in CSCR than normotensive CSCR.

Retinal and choroidal changes that occur due to continuous elevated BP can be resolved within a short period of time following BP control, they can lead to visual loss caused by incomplete photoreceptor recovery or nerve fibre layer defects. Elevated BP leads to choroidal fibrinoid necrosis, choriocapillaris non perfusion, RPE ischemic necrosis, outer blood–retinal barrier disruption and SRF accumulation.[13]

Hypertensive choroidopathy is one clinical entity seen in hypertensive patients, very difficult to diagnose, clinically elschnigs spots will be seen due to retinal pigment epithelial ischaemia. One elderly female presented with macular

changes in both eyes. Her BCVA in RE CF 1mt and LE 6/12. On examination her macula in BE showed RPE changes with macular thickening surrounding the foveal area. FFA showed window defects plus hyper fluorescence in macular area in early phase with increase in size and intensity from mid to late phases of angiography, hypo fluorescent irregular spot with surrounding hypo pigmented halo seen near the temporal arcade suggestive of old elschnig spot in RE where as in LE one single hyperfluorescene granular leaking point in papillo macular area with surrounding window defects. OCT of RE showed altered foveal contour with multiple inner retinal cystic spaces with fusiform irregular thickening in the outer retinal layers suggestive of CNVM and in LE shallow neurosensory detachment in papillomacular area. B Scan of both eyes showed diffuse choroidal thickening. VKH was excluded, no signs of inflammation noted. Patient was given anti vegf in RE and LE treated with topical Nevanac eye drops. One month after injection the fusiform thickening and cystic spaces resolved with decrease in macular thickening but patient vision improved only to CF 2mts in RE. Hypertensive choroidopathy is a silent disease which causes many changes in choroid which leads to secondary changes in outer layers of retina.

Choroidal changes are seen in young patients with elevated blood pressure, young patients are more susceptible to these changes because of the flexibility of blood vessels.[14]

Chronic nephropathy patients can present with sudden loss of vision; these patients should undergo regular screening to prevent the blindness.[15] Management in these patients is very difficult, they don't even respond to the anti vegf agents when given to reduce the macular oedema, especially when serum creatinine level is high and when there is severe proteinuria. In our study most of the patients presented with sudden vision loss were both male and female patients, all patients were above 40 years, but they did not show any fundus changes as well as no anterior segment changes. All patients underwent systemic evaluation for the further management.

4. Conclusion

Hypertension is a serious systemic disorder which can damage any organ in the human body, need to evaluate and manage properly to prevent the major vascular events. Young patients who are presenting with hypertensive retinopathy and choroidopathy requires proper management to prevent the eye damage.

5. Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

6. Source of Funding

None.

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Cite this article: Sultana A. Clinical study and management of various ocular manifestations in hypertensive patients at tertiary eye care centre in South India. *IP Int J Ocul Oncol Oculoplasty* 2021;7(4):383-387.