*IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*
*e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 19, Issue 4 Ser.10 (April. 2020), PP 11-14*
*www.iosrjournals.org*

# Fundus Findings in Patients of Malignant Hypertension

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***Abstract:***
***Purpose -*** *to study the fundus findings in patients of malignant hypertension.*
***Methods-*** *This was a prospective observational study that involved 50 eyes of 25 patients with malignant hypertension complaining of diminution of vision. Complete ophthalmic examination was done in diffuse light followed by direct opthalmoscopic examination and optical coherence tomography.*
***Results-****There were 14 females and 11 males .Fundus findings in patients of malignant hypertension include arteriolar narrowing in all the patients followed by cotton wool spots in 86% patients , flame shaped haemorrhages in 62% patients, hard exudates in 58% patients, disc edema in 56%patients and disc haemorrhage in 12% patients. Optical coherence tomography findings included macular edema (central macular thickness > 300 μm), irregularly reflective regions, retinal nerve fiber layer thickening, subretinal fluid (SRF), intraretinal fluid, and intraretinal hyperreflective dots. .*
***Conclusion-****All the patients with hypertensive retinopathy had poor vision with fundus findings like arteriolar narrowing, cotton wool spots, hard exudates, flame shaped haemorrhages, disc edema and disc haemorrhage. OCT findings include macular edema , retinal nerve fiber layer thickening, subretinal fluid and intraretinal fluid.*
***Keywords:*** *hypertensive retinopathy,cotton wool spots,hard exudates,disc edema*

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Date of Submission: 13-04-2020 Date of Acceptance: 28-04-2020

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## I. Introduction

Hypertension is one of the most common adult conditions in industrialized countries. In the seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, hypertension is defined as a systolic and/or a diastolic blood pressure consistently higher than the accepted norm (systolic: ≤139 mm Hg, diastolic: ≤89 mm Hg).[4] In the 2007 European Society of Hypertension–European Society of Cardiology guideline[5] and the report of the fourth working party of the British Hypertension Society,[6] severe hypertension (stage III hypertension), also termed malignant hypertension, was defined as a systolic blood pressure (SBP) exceeding 179 mm Hg or a diastolic blood pressure (DBP) exceeding 109 mm Hg.

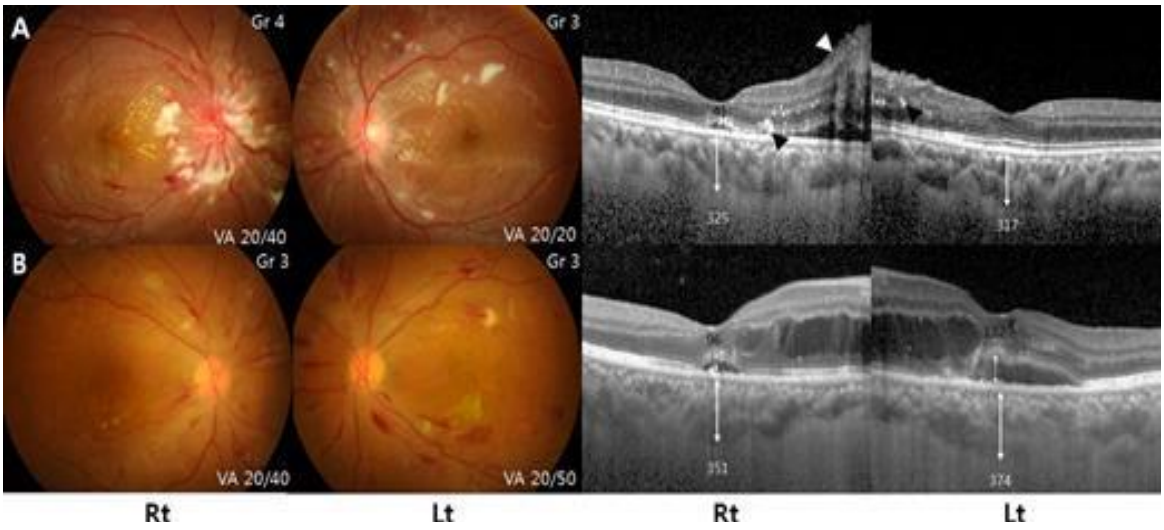

Hypertension causes microvascular damage in both the cerebral and retinal circulations (1–3). Because the retinal and cerebral vessels share embryological and anatomical characteristics, they also may show similar patterns of damage from diseases such as hypertension (3). Thus, it has long been suggested that examination of the ocular fundus could provide a noninvasive view of intracranial vascular pathology (2).

Hypertension is associated with vascular abnormalities in the brain, heart, kidneys, and eyes. Hypertension may cause retinal hemorrhages, cotton wool spots, intraretinal lipid accumulation, and vessel closure in the retinal capillaries and choriocapillaris. In patients with severe hypertension, ocular changes can be striking and include optic neuropathy, choroidopathy, and retinopathy.[4,5]

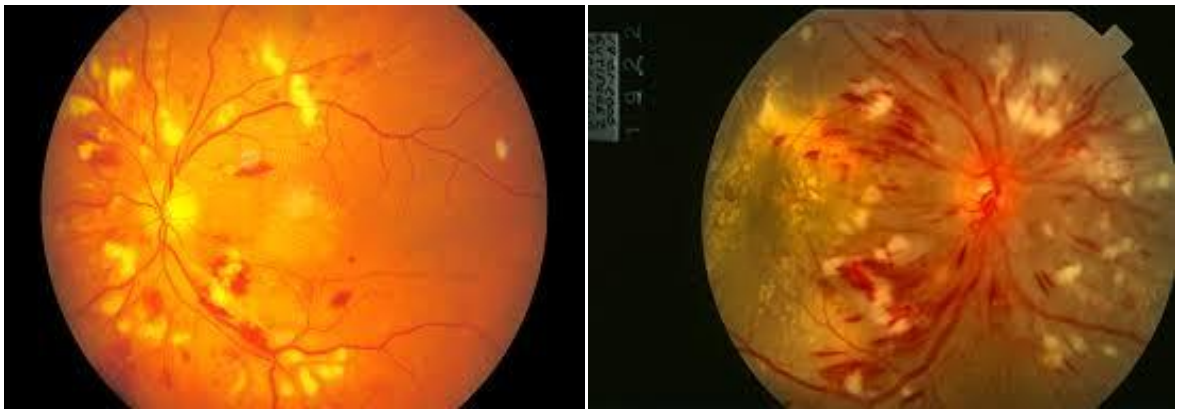
Optical coherence tomography (OCT) has revolutionized our ability to visualize structural abnormalities in the retina. In comparison with older time-domain OCT (TD-OCT) technologies, spectral-domain OCT (SD-OCT) provides significantly better image resolution, allowing detailed images of retinal morphologic features to be obtained. Morphologic abnormalities associated with severe hypertension, as identified with TD-OCT, include subretinal fluid (SRF) and macular edema.[6]

**CLINICAL FEATURES**

Retinal changes observed in patients with severe hypertension included intraretinal transudate, multiple cotton wool spots, and retinal hemorrhages. Additionally, OCT revealed intraretinal fluid and SRF in approximately one-third of patients with severe hypertension. Intraretinal transudate results from breakdown of the retinal arteriole blood–retinal barrier caused by highly elevated BP.[7] Retinal changes were generally observed in the peripapillary and periarteriolar areas, as shown in Figure 1. Additionally, patients with more severely elevated BP (i.e., >240 mm Hg) had more extensive areas of retinal exudates. This indicates that large arterioles in the peripapillary area had remarkably high intravascular pressure, high enough to break the blood–retinal barrier. However, the intravascular pressure in smaller arterioles may not have been quite as elevated because pressure is attenuated across the peripapillary arterioles. The peripapillary and periarteriolar distribution of retinal abnormalities is a unique feature of hypertensive retinopathy and may aid in distinguishing it from other conditions causing retinal hemorrhage, edema, and exudates.




Cotton wool spots were very common in our patients with severe hypertension. Malignant hypertension is characterized by arteriolar fibrinoid necrosis and retinal nerve fiber layer ischemia,[8,9] which lead to the spots. Using SD-OCT, irregular reflection and swelling of the retinal nerve fiber layer were identified and are believed to result from ischemic damage to the layer in which the spot is located. Irregular reflections in OCT images can also be caused by flame-shaped retinal hemorrhages located in the nerve fiber layer. Cotton wool spots, although generally resolved within 1 month of BP control, are clinically important because they can represent permanent nerve fiber layer defects.



Accumulation of SRF was particularly important in eyes examined here because it was associated with vision loss. It is generally believed that SRF accumulation is caused by choroidal permeability changes. These changes lead to an increase in choroidal interstitial fluid, which eventually extends into the subretinal space.

## II. Method And Material

This was a prospective observational study that involved 50 eyes of 25 patients with malignant hypertension complaining of diminution of vision. Patients were recruited from the OPD of MLB MEDICAL college, Jhansi ,Uttar Pradesh and were followed from 1st October 2019 - 1st march 2020 . It was performed under the Helsinki Declaration of 1975, as revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

**Inclusion criteria**

1. All patients who presented to the OPD of MLB medical College Jhansi with the complaint of dimunition of vision and high blood pressure who were found to have malignant hypertension (BP > 180/120 ) were included.

**Exclusion criteria**

1.Patients with ocular systemic diseases(like diabetes) that could affect the retina.
2.Patients with other retinal disorders
3.Patients with recent intraocular surgery
4.Patients with the history of trauma
5.Mentally or physically unfit patients

All patients were subjected to a detailed history taking, refraction using Topcon autorefractometer and best corrected visual acuity (VA) measurement. All patients had complete ophthalmic examination including biomicroscopic slit lamp examination , fundus examination with 90D lens and fundusphotography and optical coherence tomography.

Optical coherence tomography examination was done through dilated pupils, OCT examination was done through a dilated pupil using commercially available Cirrus HD-OCT Model 4000 - Carl Zeiss Meditec, Inc., Dublin,California, USA or Spectralis OCT Heidelberg Engineering.

## III. Results

A total of 50 eyes of 25 patients were studied. We included eyes with complaint of diminution of vision. There were 11 males and 14 females and 60% of the studied eyes were the right eyes.

All eyes had one or more features typical of hypertensive retinopathy (arteriolar narrowing, cotton wool spots, hard exudates, flame shaped haemorrhages, disc edema and disc haemorrhage)

**Table1: Fundus finding in patients of malignant hypertension**

| Features | Total % |
|---|---|
| Arteriolar narrowing | 100 |
| Cotton wool spots | 86 |
| Flame shaped haemorrhage | 62 |
| Hard exudates | 58 |
| Disc edema | 56 |
| Disc haemorrhage | 12 |

**Table2: Optical coherence tomography finding in patients of malignant hypertension**

| Features | Total % |
|---|---|
| Macular edema | 36 |
| Irregular reflection | 34 |
| Thickening of RNFL | 42 |
| Subretinal fluid | 51 |
| Inner retinal fluid | 20 |
| Hyperreflective dots within retina | 62 |

## IV. Discussion

Hypertension may cause retinal hemorrhages, cotton wool spots, intraretinal lipid accumulation, and vessel closure in the retinal capillaries and choriocapillaris. Based on these fundoscopic features, Keith et al.[10] developed a classification system for hypertensive retinopathy to categorize these signs into four groups of severity. This classification is widely used in current clinical practice, but Wong and Mitchell[11] most recently proposed a simplified hypertensive retinopathy grading system with the following classifications: none, mild, moderate, and malignant retinopathy. Ugarte M Horgan S Rassam S Leong T Kon CH concluded that highly elevated BP can lead to choroidal fibrinoid necrosis, choriocapillaris nonperfusion, RPE ischemic necrosis, outer blood–retinal barrier, and SRF accumulation.[12,] .Ong YT Wong TY Klein R have shown a significant association between some hypertensive retinopathy grades (i.e., moderate or malignant) and risk of stroke, coronary artery disease, and death.[13] Hammond S concliuded that hypertensive *choroidopathy* occurs as a result of choroidal ischemia, which develops because the choroidal vasculature does not possess the autoregulatory capacity of the retinal vasculature and *hypertensive retinopathy* describes a spectrum of microvascular abnormalities in people with elevated blood pressure and may be divided into several phases like vasoconstrictive and sclerotic phase.[14]

## V. Conclusion

Individuals with malignant hypertension have characteristic peripapillary and periarteriolar hypertensive retinopathy features, SRF accumulation, and increased choroid thickness. The presence of SRF was associated with choroidal thickening and with poor visual outcome in patients with severe hypertension. Thus, OCT may be useful to document hypertensive retinopathy and choroidopathy severity. Periodic ophthalmic examination should be done and strict blood pressure monitoring and control should be done to control the progression of hypertensive retinopathy.

## References


[1]. Wong TY, Mitchell P. The eye in hypertension. Lancet. 2007;369:425–435. [PubMed] [Google Scholar]
[2]. Wong TY, Klein R, Klein BE, et al. Retinal microvascular abnormalities and their relationship with hypertension, cardiovascular disease, and mortality. Surv Ophthalmol. 2001;46:59–80. [PubMed] [Google Scholar]
[3]. Goto I, Katsuki S, Ikui H, et al. Pathological studies on the intracerebral and retinal arteries in cerebrovascular and noncerebrovascular diseases. Stroke. 1975;6:263–269. [PubMed] [Google Scholar]
[4]. Hammond S Wells JR Marcus DM Prisant LM. Ophthalmoscopic findings in malignant hypertension. *J Clin Hypertens (Greenwich)*. 2006; 8: 221–223. [CrossRef] [PubMed]
[5]. Tso MO Jampol LM. Pathophysiology of hypertensive retinopathy. *Ophthalmology*. 1982; 89: 1132–1145. [CrossRef] [PubMed]
[6]. Suzuki M Minamoto A Yamane K Uka J Aoki S Mishima HK. Malignant hypertensive retinopathy studied with optical coherence tomography. *Retina*. 2005; 25: 383–384. [CrossRef] [PubMed]
[7]. Hayreh SS Servais GE Virdi PS. Fundus lesions in malignant hypertension. IV. Focal intraretinal periarteriolar transudates. *Ophthalmology*. 1986; 93: 60–73. [CrossRef] [PubMed]
[8]. Kerenyi T Jellinek H Huttner I Goracz G Konyar E. Fibrinoid necrosis of the vascular wall in experimental malignant hypertension. *Acta Morphol Acad Sci Hung*. 1966; 14: 175–182. [PubMed]
[9]. Wolfgarten M Magarey FR. Vascular fibrinoid necrosis in hypertension. *J Pathol Bacteriol*. 1959; 77: 597–603. [CrossRef] [PubMed]
[10]. Keith NM Wagener HP Barker NW. Some different types of essential hypertension: their course and prognosis. *Am J Med Sci*. 1939; 197: 332–343. [CrossRef]
[11]. Wong TY Mitchell P. Hypertensive retinopathy. *N Engl J Med*. 2004; 351: 2310–2317. [CrossRef] [PubMed]
[12]. Ugarte M Horgan S Rassam S Leong T Kon CH. Hypertensive choroidopathy: recognizing clinically significant end-organ damage. *Acta Ophthalmol*. 2008; 86: 227–228. [CrossRef] [PubMed]
[13]. Ong YT Wong TY Klein R Hypertensive retinopathy and risk of stroke. *Hypertension*. 2013; 62: 706–711. [CrossRef] [PubMed
[14]. Hammond S, Wells JR, Marcus DM, et al. Ophthalmoscopic findings in malignant hypertension. Journal of clinical hypertension (Greenwich, Conn) 2006;8:221–223. [PubMed] [Google Scholar]