Original Article

Evaluation of choroidal thickness via enhanced depth-imaging optical coherence tomography in patients with systemic hypertension

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Purpose: The purpose was to evaluate choroidal thickness via spectral domain optical coherence tomography (SD-OCT) and to compare the data with those of 24-h blood pressure monitoring, elastic features of the aorta, and left ventricle systolic functions, in patients with systemic hypertension. **Materials and Methods:** This was a case–control, cross-sectional prospective study. A total of 116 patients with systemic hypertension, and 116 healthy controls over 45 years of age, were included. Subfoveal choroidal thickness (SFCT) was measured using a Heidelberg SD-OCT platform operating in the enhanced depth imaging mode. Patients were also subjected to 24-h ambulatory blood pressure monitoring (ABPM) and standard transthoracic echocardiography (STTE). Patients were divided into dippers and nondippers using ABPM data and those with or without left ventricular hypertrophy (LVH+ and LVH–) based on STTE data. The elastic parameters of the aorta, thus aortic strain (AoS), the beta index (BI), aortic distensibility (AoD), and the left ventricular mass index (LVMI), were calculated from STTE data. **Results:** No significant difference in SFCT was evident between patients and controls ($P \leq 0.611$). However, a significant negative correlation was evident between age and SFCT in both groups ($r = -0.66/-0.56$, $P \leq 0.00$). No significant SFCT difference was evident between the dipper and nondipper groups ($P \leq 0.67$), or the LVH (+) and LVH (–) groups ($P \leq 0.84$). No significant correlation was evident between SFCT and any of AoS, BI, AoD, or LVMI. **Discussion**: The choroid is affected by atrophic changes associated with aging. Even in the presence of comorbid risk factors including LVH and arterial stiffness, systemic hypertension did not affect SFCT.

Key words: Choroid, hypertension, optic coherence tomography

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The primary function of the choroidal vascular network is to supply nutrients and oxygen to the outer retina. The choroidal layer of the eye has a dense and complex vasculature. Enhanced depth imaging optical coherence tomography ([EDI-OCT]; as defined by Spaide *et al*.[1]) enables *in vivo* cross-sectional imaging of the choroid. Choroidal vessels are (principally) under neurogenic control and are thus not autonomously regulated (unlike the retinal vascular network). Sympathetic innervation is afforded by noradrenergic neuropeptide mediators,[2] and parasympathetic innervation by cholinergic mediators.[3] Furthermore, the choroid is innervated by trigeminal sensorial fibers; the calcitonin-gene related peptide mediates messaging at junctions.[4]

In the past, many lesions attributable to hypertensive choroidopathy have been erroneously classified as being caused by hypertensive retinopathy. Hypertensive choroidopathy has been associated with toxemia of pregnancy, pheochromocytoma, renal disorders, and malignant hypertension.[5] Hayreh defined lesions attributable to hypertensive choroidopathy as initial, acute, and chronic, based on studies in rhesus monkeys.[6,7] The circulatory levels of various endogenous vasoactive substances increase in patients with malignant hypertension. These substances include potent vasoconstrictors such as angiotensin II, adrenaline, vasopressin, and endothelin-1 (ET-1). Such substances can easily pass from the fenestrated choriocapillaris to the interstitial area, triggering severe vasoconstriction and tissue ischemia in the choriocapillaris. Thus, the choroidal lesions (Elschings spots and Siegrist's streaks) of malignant hypertension develop. Similar changes occur in the blood and sympathetic system in patients with chronic essential hypertension.[8]

We thus evaluated the effects of primary essential hypertension on the choroidal vascular network in patients aged over 45 years. Left ventricular hypertrophy (LVH) and arterial stiffness are predictors of microvascular damage and disease severity in patients with chronic hypertension. Furthermore, we evaluated subfoveal choroidal thickness (SFCT) in association with changes in LVH and arterial stiffness.

Materials and Methods

This study was conducted with the approval of our Local Ethics Committee and in accordance with the tenets of the Declaration of Helsinki. Illustrated consent forms were given to all participants and were explained and signed. Comprehensive ophthalmic examinations were performed on all groups. All study participants had best corrected visual acuities of 20/25 or more, a refractive error in the range +3.0 to –3.0 diopters and intraocular pressure (IOP) lower than 21 mmHg. Those with systemic or ocular disease (glaucoma, uveitis, high myopia, age-related macular degeneration, diabetes mellitus, etc.) and/

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or a history of ophthalmic surgery that may have affected the choroidal vascular network were excluded. All participants in the patient group included in the study were chronic hypertensive patients receiving medical treatment and with cardiology follow-up. All these patients were diagnosed according to the criterias of the 2013 European Society of Hypertension (ESH)/ European Society of Cardiology (ESC) report.[9]

Subfoveal choroidal thickness measurements were performed using a Heidelberg Spectralis HRA + OCT platform (GmBH, Heidelberg, Germany) operating in the EDI mode, as suggested by Spaide *et al*.[1] All measurements were performed during the same daily interval (10-12 am). Prior to blood pressure measurement, each patient rested for 5 min. Patients with excessive high-blood pressures above systolic 180 mmHg and/or diastolic 110 mmHg were excluded (2013 ESH/ESC report).[9] All measurements were performed by the same technician. All choroidal thickness data were assessed by the same ophthalmologist. Measurements were manually performed in an area bounded by the outer limit of the retinal pigment epithelium and the inner scleral border [Fig. 1a and b].

A total of 82 of the 116 hypertensive patients agreed to 24-h ambulatory blood pressure monitoring (ABPM), achieved using a portable device (Spacelabs Medical ABPM Model no: 90207 Snoqualmie, WA, USA) which recorded data every 30 and 15 min from 24:00 to 08:00 and 08:00 to 24:00, respectively. All measurements were taken on normal working days, and patients were told to continue their daily routines. Patients were divided into two groups: Those in whom the mean systolic and diastolic blood pressures fell by at least 10% at night (compared to day) were "dippers," and the others "nondippers."

A total of 62 of the 116 hypertensive patients agreed to undergo standard transthoracic echocardiography (STTE), which was performed in the left lateral decubitus position, by the same cardiologist, using a GE-Vingmed Vivid 7 device (GE-Vingmed Ultrasound AS, Horten, Norway) fitted with a 2.5-MHz probe. Electrocardiographic data and arterial blood pressure measurements were simultaneously recorded. Echocardiographic procedures followed the criteria of the American Society of Echocardiography.[10]

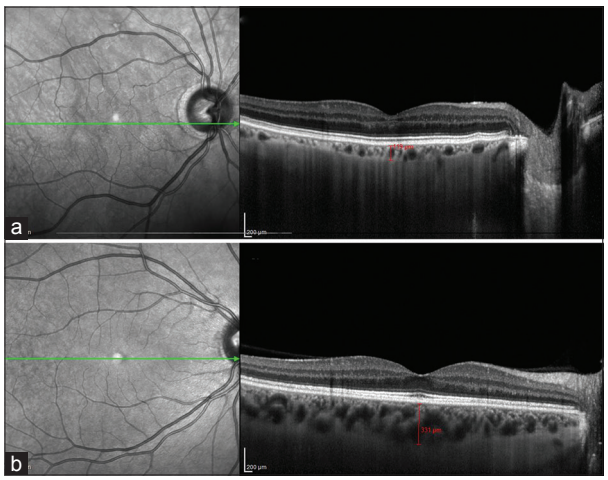


Figure 1: Right eye optical coherence tomography (OCT) image of a 79-year-old patient (a). Diffuse atrophy and thinning of choroidal plexus are remarkable. Right eye OCT image of a 52-year-old patient (b)

Left ventricular mass (LVM) and mass index were calculated using the Devereux formula suggested by the American Society of Echocardiography, employing STTE data.[11]

Thus: LVM = 1.04 ([IVSTD + LVIDD + PWTD]^3– [LVIDD]^3) – 13. 6 (g);

LVMI = LVM/body surface area (g/m^2).

Where, LVM: Left ventricular mass; IVSTD: Interventricular septum thickness in diastole; LVIDD: Left ventricle internal diameter in diastole; PWTD: Posterior wall thickness in diastole; and LVMI: Left ventricular mass index.

The upper limits of the LVMI were set at 115 g/m^2 for males and 95 g/m^2 for females.[12] Measurements higher than these values were considered to reflect LVH.

Elastic parameters of the aorta were calculated using the following formulae, employing STTE data:[13]

Aortic strain (AoS) = (SD − DD)/DD × 100

Beta index (BI) = ln (SBP/DBP)/([SD − DD]/DD)

Aortic distensibility (AoD) 10–^{6} cm^2/dyn = 2 × (SD − DD)/([SBP − DBP] × DD)

Where SD: Systolic diameter, DD: Diastolic diameter, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, and ln: Natural logarithm.

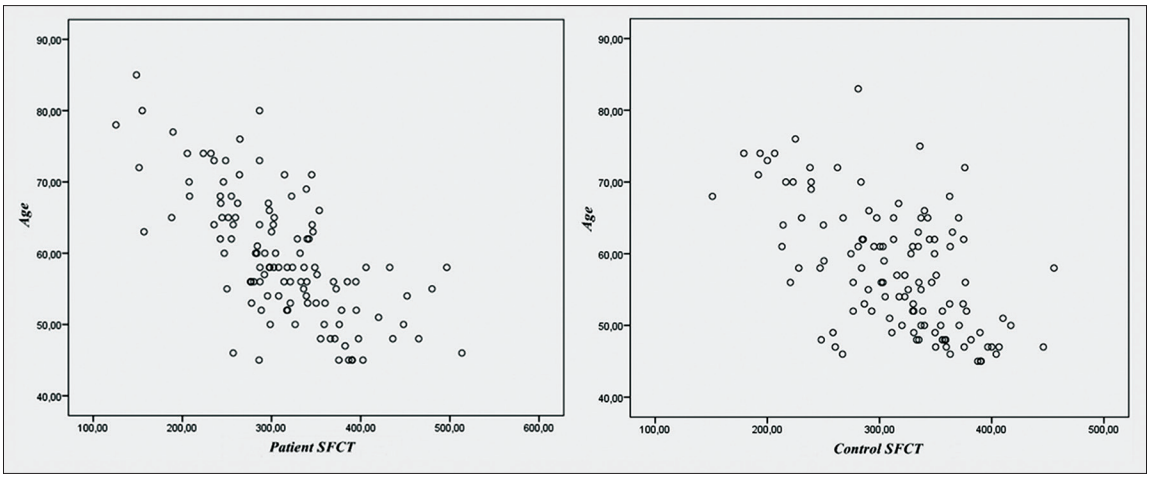
Statistical analysis was conducted using the IBM SPSS for Windows, (v 19.0, IBM-SPSS, Chicago, Illinois, USA). The Kolmogorov–Smirnow test was used to determine whether continuous variables were distributed normally. The Chi-squared test was used to compare between-gender data and the independent samples *t*-test to compare age and SFCT data between the groups. Correlations between age and SFCT data were sought using Pearson correlation analysis. All values are given as means ± standard deviations, and significance was considered attained at $P \leq 0.05$.

Results

A total of 228 eyes of 116 patients (41 males/75 females) were included in the test group (mean age: 59.7 ± 8.8 years, age range: 45–82 years). Four patients had previous histories of branch retinal vein occlusion, and only single eyes of these patients were included. In the control group, a total of 232 eyes of 116 healthy subjects (55 males/61 females) were included (mean age: 57.7 ± 8.7 years, age range: 45–79 years). No statistically significant difference in SFCT data yielded by left and right eyes was evident. The mean SFCT value for each patient was obtained by averaging the results from both eyes. No significant between-group difference was evident in terms of gender or age. No significant difference in any SFCT parameter was evident between the patient and control groups [$P \leq 0.61$; Tabl**e** 1].

A significant negative correlation was evident between SFCT and age in both groups [Tabl**e** 1]. Regression analysis showed that SFCT decreased by 3.91 µm and 5.55 µm for each year of age in the control and patient groups, respectively [Graph 1].

Patients were divided into dippers (19 patients, 38 eyes, mean age: 59.26 ± 7.98 years) and nondippers (63 patients, 124 eyes, mean age: 58.6 ± 8.25 years) in terms of mean blood



Graph 1: Age and subfoveal choroidal thickness correlations between patient and control groups

Table 1: Demographic characteristics of the patient and the control groups and comparison of SFCT between the groups

	Systemic hypertension group (*n*=116)	Control group (*n*=116)	*P*
Age	59.7±8.8	57.7±8.7	0.079[a]
Gender (male/female) (%)	41 (35.3)/75 (64.7)	55 (47.4)/61 (52.6)	0.062[b]
Mean SFCT (mμ)	310.43±72.90	314.90±59.97	0.611[a]

[a]Independent sample *t*-test, [b]Chi-square test. SFCT: Subfoveal choroidal thickness

Table 2: Demographic characteristics of dipper and the nondipper groups and comparison of SFCT between the groups

	Dipper (*n*=19)	Nondipper (*n*=63)	*P*
Age	59.26±7.98	58.6±8.25	0.759[a]
Gender (male/female) (%)	11 (57.9)/8 (42.1)	22 (34.9)/41 (65.1)	0.073[b]
Mean SFCT (mμ)	328.44±72.62	319.76±79.66	0.672[a]

[a]Independent sample *t*-test, [b]Chi-square test. SFCT: Subfoveal choroidal thickness

pressure decreases overnight. No significant between-group difference in SFCT was evident [$P \leq 0.672$; Table 2].

Left ventricular mass index values were calculated employing Devereux's formula and STTE measurements on 62 patients. LVMI values of more than 115 g/m^2 and 95 g/m^2 were considered to reflect LVH in males and females, respectively. Patients were divided into an LVH (+) group (32 patients, 64 eyes, mean age: 58.21 ± 7.94 years) and an LVH (−) group (30 patients, 60 eyes, mean age: 60.43 ± 7.95 years). No significant between-group difference in SFCT was evident [$P \leq 0.841$; Table 3].

No significant correlation was observed between SFCT and any of LVMI, AoS, BI, or AoD [Table 4].

Discussion

The pathophysiology of age-related changes in choroidal microvascular structure has been, but little investigated. Histopathological analyzes of postmortem samples revealed progressive age-related decreases in choroidal thickness. In addition, mean vascular density and choriocapillaris vessel diameter exhibited marked reductions with age.[14,15] However, these data may not be in line with *in vivo* findings. Postmortem tissue fixation and cessation of circulation cause structural changes in the choroid and render objective assessments difficult. In the present study, we found (negative) significant correlations, in both groups, between the age and choroidal thickness, as have other studies.[16-18] Upon regression analysis, SFCT decreased by 3.91 and 5.55 μm for each year of age in the control and patient groups, respectively. It is possible that the choroidal vasculature is affected by artherosclerotic and aging changes as are other microvascular tissues.[19,20]

The choroid has a dense vascular structure and the thickness thereof is affected by changes in IOP and perfusion pressure;[21] endogenous nitric oxide (NO) levels;[22] vasoactive substances produced by choroidal ganglion cells;[23] and circulating catecholamines in the blood.[24,25] Unlike retinal blood flow, which is autoregulated, choroidal blood flow is principally controlled by the autonomous nervous system and circulatory hormones.[26] A reduction in choroidal blood flow triggers sympathetic activation and noradrenaline discharge, stimulating the alpha-1 receptors, in turn triggering vasoconstriction.[27,28] The parasympathetic innervation causes blood flow to rise upon discharge of NO.[29] However, the choroid mounts a significant compensatory response to alterations in ocular perfusion pressure caused by manipulation of IOP and mean arterial pressure.[30]

Furthermore, the choroid can respond to changes in ocular perfusion pressure caused by dynamic and static exercise.[31,32] Riva *et al*.[31] showed that the choroidal blood flow increased by only 12%, despite a 60% increase in ocular perfusion pressure, upon exercise. Thus, the response of the choroidal vascular bed is confined within certain limits.

Obvious choroidal ischemia develops in patients with malignant hypertension, attributable to elevated levels of circulating vasoactive substances (angiotensin II, adrenaline, vasopressin, and ET-1) and increased sympathetic activity.[6,7] Impairments in endothelial function cause the levels of vasoactive substances (vasopressin, angiotensin II, and ET-1) to increase as chronic hypertension develops. The sympathetic

Table 3: Demographic characteristics of LVH (+) and the LVH (–) groups and comparison of SFCT between the groups

	LVH (+) (*n*=32)	LVH (–) (*n*=30)	*P*
Age	58.21±7.94	60.43±7.95	0.277[a]
Gender (male/female) (%)	15 (46.9)/17 (53.1)	13 (43.3)/17 (56.7)	0.779[b]
Mean SFCT (mμ)	319.56±80.88	315.51±76.65	0.841[a]

[a]Independent sample *t*-test, [b]Chi-square test. LVH: Left ventricular hypertrophy, SFCT: Subfoveal choroidal thickness

Table 4: Correlation analysis between SFCT and LVMI, AoS, BI and AoD

	Mean SFCT	
	r	*P*
LVMI (mean: 10,782±15,45 g/m^2)	–0.076	0.559
AoS (mean: 9.55%±0.65%)	0.083	0.521
BI (mean: 1.87±0.11)	–0.193	0.134
AoD (mean: 38.58±2.62 10^{-6} cm^2/dyn)	0.152	0.237

Pearson correlation analysis. SFCT: Subfoveal choroidal thickness, LVMI: Left ventricular mass index, AoS: Aortic strain, BI: Beta index, AoD: Aortic distensibility

nervous system is activated in such patients, and circulating epinephrine levels also increase.[33] The long-term probable complications of chronic hypertension are LVH and arterial stiffness; these are predictors of disease severity and multiple end-organ damage.

Left ventricular hypertrophy, associated with an increased incidence of cardiovascular disease, is a strong and independent risk factor for mortality, coronary artery disease, heart failure, and stroke, in both genders.[34]

Arterial stiffness is frequently used as a defining viscoelastic feature of vessel walls. AoS, BI, and AoD reflect the arterial stiffness of chronic hypertension and may be calculated using STTE data. Arterial stiffness may develop with age, perhaps associated with smoking, hypercholesterolemia, diabetes mellitus, and/or hypertension.[35,36] Arterial stiffness is an important prognostic factor for the development of vascular diseases including renal failure, stroke, dementia, heart failure, and myocardial infarction.[37]

It is important that the nocturnal blood pressure should remain in the physiological range to prevent organ damage. Verdecchia *et al*.[38] reported that nondipper hypertensive patients had higher blood pressures over 24-h than did dippers; nondippers are thus at an increased risk of cardiovascular disease.

Thus, LVH and arterial stiffness are risk factors for damage to macro-and micro-vascular vessels and end-organ damage, in patients with chronic essential hypertension. The aim of our study was to explore the effects of chronic hypertension and associated risk factors on the SFCT. All of our patients were receiving medical treatment and cardiological follow-up. Malignant hypertension was not associated with any histopathological change to the choroid in our patients. Furthermore, we found no significant difference in choroidal thickness between the patient and control groups, dippers and nondippers, or LVH (+) and LVH (–) subjects. We found no significant correlation between any arterial stiffness parameter (AoS, BI, or AoD) and SFCT.

Only one prior study evaluated choroidal thickness in hypertensive patients. Masís *et al*.[39] studied 112 patients with systemic hypertension and 15 healthy controls. The mean ages of the two groups were 67 and 51 years, respectively. The choroidal thickness was significantly thinner in hypertensive patients compared to controls. However, we suggest that this reflected only a between-group age difference.

The critical oxygen and nutrient supplies to the outer one-third of the retina are delivered by the choroid, operating under high perfusion pressure. Therefore, even a minimal change in choroidal perfusion pressure may impair retinal functioning and oxygenation.[6] The retinal and optic nerve head blood flows are held within certain limits by ocular autoregulatory mechanisms that guard against fluctuations in ocular perfusion pressure. Systemic hypertension causes ocular vascular damage by disrupting such mechanisms. When the critical compensatory limits are exceeded in those with severe uncontrolled hypertension, sclerotic changes affecting both the diameter and structure of vessel walls develop in the retinal vasculature. Accordingly negative correlations reported between blood pressure and retinal arteriolar diameter in the Beaver Dam Eye Study[40] and also an increase in retinal venular caliber was determined that was associated with hypertension in The Blue Mountains Eye Study.[41] However, we did not observe any change in the SFCT that might affect the structure of the vasculature.

Thus, the choroidal vascular network seems to be protected from the negative effects of chronic hypertension because the network plays a critical role in nourishing the retina. Neither arterial stiffness nor LVH appears to be associated with any change to the SFCT in patients with chronic essential hypertension. This may be because the choroid is controlled principally by neural and humoral factors, and responds to changes in blood pressure only within certain limits, to preserve an optimal retinal perfusion pressure. Thinning of the choroidal layer in both groups with age is an expected, secondary natural result of age-related processes that also affect other microvascular tissues.

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