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ORIGINAL ARTICLE

Association between left ventricular hypertrophy severity and hypertensive retinopathy grade in hypertensive heart disease: a cross-sectional study

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ABSTRACT

Aim: To assess the association between left ventricular hypertrophy (LVH) severity and hypertensive retinopathy grade in patients with hypertensive heart disease.

Methods: This cross-sectional study included 60 consecutive adults with hypertensive heart disease at Dr Mohammad Hoesin General Hospital, Palembang, Indonesia, from January 2024 to December 2025. Left ventricular mass index was measured by transthoracic echocardiography, and LVH severity was categorised using sex-specific guideline thresholds. Retinopathy was graded using the Keith-Wagener-Barker classification during bilateral dilated fundoscopy by a single examiner blinded to the echocardiographic findings. Associations were assessed using Spearman's rank correlation coefficient (ρ) with 95% confidence intervals (CIs).

Results: Mean age was 56.3 ± 8.9 years, and 28 (46.7%) participants were male. LVH was present in 52 (86.7%) participants. LVH severity correlated strongly with retinopathy grade (ρ = 0.71; 95% CI 0.56–0.82; $p < 0.001$). The sensitivity analysis restricted to participants with LVH yielded ρ = 0.60 (95% CI 0.39–0.75; $p < 0.001$). Exploratory subgroup correlations were directionally consistent but imprecise.

Conclusion: LVH severity was strongly associated with hypertensive retinopathy grade in this cross-sectional cohort. Fundoscopy may complement comprehensive assessment of hypertension-mediated organ damage but cannot replace echocardiography. Larger studies are needed.

Keywords: echocardiography; hypertensive retinopathy; hypertrophy, left ventricular

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INTRODUCTION

Hypertension is a leading modifiable cause of cardiovascular disease and premature mortality and affects more than one billion adults worldwide (1). International guidelines emphasise early detection and treatment to reduce cardiovascular risk (2). Sustained elevation of arterial pressure causes hypertension-mediated organ damage involving the heart, brain, kidneys, large arteries, and retinal microvasculature (3).

Left ventricular hypertrophy (LVH) is a major cardiac manifestation of chronic pressure overload. Increased afterload promotes cardiomyocyte hypertrophy, extracellular matrix expansion, and fibrosis, resulting in increased left ventricular mass (4). Echocardiographic left ventricular mass index (LVMI) is used to define and grade LVH (5). Cardiac remodelling may regress after effective blood-pressure treatment (6), but established LVH is associated with heart failure, atrial fibrillation, sudden cardiac death, and other adverse cardiovascular outcomes (7).

Hypertensive retinopathy provides a visible marker of retinal microvascular injury. Because retinal, coronary, and cerebral arterioles share structural and regulatory characteristics, retinal findings may reflect systemic vascular damage (8). The Keith-Wagener-Barker (KWB) classification grades retinopathy from I to IV (9). Retinal vascular abnormalities have been associated with cardiovascular events, stroke, and mortality (10).

Although LVH and hypertensive retinopathy are established manifestations of hypertension-mediated organ damage, evidence regarding the association between their severity is limited and inconsistent. A study of Congolese patients found no significant association between hypertensive retinopathy and LVH (11), whereas retinal vascular changes are common even in untreated essential hypertension (12). Retinal arteriolar narrowing has also been linked with LVH (13), and comparisons of retinopathy classifications indicate imperfect

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reproducibility (14). A graded association may reflect shared vascular remodelling mechanisms (15), although fundoscopy cannot replace echocardiography.

The aim of this study was to assess the association between LVH severity and hypertensive retinopathy grade in patients with hypertensive heart disease at a tertiary centre in Indonesia.

MATERIAL AND METHODS

Patients and study design

This cross-sectional study included 60 consecutive adults with hypertensive heart disease evaluated at the Department of Ophthalmology, Dr Mohammad Hoesin General Hospital, Faculty of Medicine, Universitas Sriwijaya, Palembang, Indonesia, between January 2024 and December 2025. All eligible patients during the study period were included. Hypertensive heart disease was defined as essential hypertension of at least five years' duration with echocardiographic evidence of LVH, left ventricular diastolic dysfunction, or left atrial enlargement (16). Eligible participants were aged at least 18 years, had confirmed essential hypertension, had undergone transthoracic echocardiography within the preceding six months, and were able to complete bilateral dilated fundoscopy. Exclusion criteria were secondary hypertension; retinal disease unrelated to hypertension, including diabetic retinopathy, retinal vein occlusion, inherited retinal dystrophy, or glaucomatous optic neuropathy; media opacity preventing adequate fundus examination; haemodynamically significant valvular disease; congenital cardiac anomalies; and inability to provide informed consent.

The sample-size calculation indicated that 29 participants were required to detect a correlation coefficient of 0.50 with 80% power at a two-sided $\alpha = 0.05$ (17). Sixty eligible participants were included, exceeding this minimum and improving the precision of the primary estimate.

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Methods

Age, sex, duration of hypertension, body mass index, diabetes mellitus, fasting lipid profile, smoking status, and antihypertensive treatment were obtained from medical records and participant interviews. After at least five minutes of seated rest, office blood pressure was measured with an appropriately sized cuff and a calibrated mercury sphygmomanometer (Diplomat Presameter, Rudolf Riester GmbH, Jungingen, Germany). The mean of three measurements obtained at two-minute intervals was recorded. Hypertensive heart disease was determined from medical records and echocardiographic findings; ambulatory blood pressure monitoring was not performed.

Transthoracic two-dimensional and M-mode echocardiography was performed by certified cardiologists blinded to the ophthalmic findings using an iE33 system (Philips Healthcare, Amsterdam, the Netherlands) with a 2.5–3.5 MHz phased-array transducer. Interventricular septal thickness (IVSd), left ventricular internal diameter (LVIDd), and posterior wall thickness (PWd) at end-diastole were measured in parasternal long-axis views (5). Left ventricular mass (LVM) was calculated as $0.8 \times \{1.04 \times [(IVSd + LVIDd + PWd)^3 - LVIDd^3]\} + 0.6$ g and indexed to body surface area calculated with the Mosteller formula. LVH and its severity were classified according to the American Society of Echocardiography/European Association of Cardiovascular Imaging reference limits for two-dimensionally derived LVMI (5). In men, LVMI 49–115 g/m² was considered normal, 116–131 g/m² mildly abnormal, 132–148 g/m² moderately abnormal, and ≥ 149 g/m² severely abnormal. In women, LVMI 43–95 g/m² was considered normal, 96–108 g/m² mildly abnormal, 109–121 g/m² moderately abnormal, and ≥ 122 g/m² severely abnormal. Relative wall thickness (RWT) was calculated as 2 × PWd/LVIDd. Left ventricular geometry was classified as normal geometry, concentric remodelling, eccentric hypertrophy, or concentric hypertrophy according to RWT and LVMI (5).

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A single experienced ophthalmologist, blinded to the echocardiographic findings, performed the ophthalmic examinations. Best-corrected visual acuity was assessed with a Snellen chart and intraocular pressure with Goldmann applanation tonometry. After bilateral dilation with tropicamide 1% and phenylephrine 2.5%, the fundus was examined by slit-lamp biomicroscopy using a 90-dioptre non-contact lens (Volk Optical Inc., Mentor, OH, USA) under standardised conditions. Hypertensive retinopathy was graded according to the KWB classification (9): grade I, generalised arteriolar narrowing; grade II, definite focal arteriolar narrowing with arteriovenous nicking; grade III, grade II changes plus retinal haemorrhages, hard exudates, and/or cotton-wool spots; and grade IV, grade III changes plus optic disc swelling (papilloedema). The higher grade from either eye was used in the patient-level analysis.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows (version 27.0, IBM Corp., Armonk, NY, USA). Continuous variables were reported as mean ± standard deviation (SD), and categorical variables as n (%). Normality was assessed using the Shapiro-Wilk test. The association between LVH severity, categorised as absent, mild, moderate, or severe, and hypertensive retinopathy grade, categorised as absent or grades I–IV, was assessed using Spearman's rank correlation coefficient (ρ). Ninety-five per cent confidence intervals (CIs) were calculated using Fisher's z transformation, with a standard error of $1/\sqrt{(n-3)}$ on the transformed scale, and back-transformed to the correlation scale. Prespecified exploratory analyses were stratified by sex and age, dichotomised at the median (≤ 55 years, n = 31; > 55 years, n = 29), and a sensitivity analysis was restricted to participants with echocardiographic LVH. No adjustment for multiple testing was applied to exploratory analyses. A two-sided $p < 0.05$ was considered statistically significant.

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RESULTS

The study included 60 patients with hypertensive heart disease (Table 1). Mean age was 56.3 ± 8.9 years, 28 (46.7%) participants were male, and mean duration of hypertension was 12.4 ± 6.8 years. Mean systolic and diastolic office blood pressures were 158 ± 16 and 96 ± 11 mmHg, respectively (Table 1).

Echocardiography identified LVH in 52 (86.7%) participants. Eight (13.3%) participants had normal left ventricular mass but met the study definition of hypertensive heart disease through other structural changes. Mean LVMI was 132 ± 28 g/m², mean left ventricular ejection fraction was 63 ± 8%, and mean RWT was 0.48 ± 0.08. Concentric hypertrophy was present in 38 (63.3%) participants, eccentric hypertrophy in 14 (23.3%), concentric remodelling in 4 (6.7%), and normal geometry in 4 (6.7%).

All eight participants without LVH met the definition of hypertensive heart disease through grade I left ventricular diastolic dysfunction. Of these, four also had concentric remodelling (elevated RWT with normal LVMI), and the remaining four had normal geometry accompanied by left atrial enlargement.

The distribution of hypertensive retinopathy and its cross-tabulation with LVH severity are presented in Table 2. Hypertensive retinopathy was present in 50 (83.3%) participants: grade I in 15 (25.0%), grade II in 17 (28.3%), grade III in 12 (20.0%), and grade IV in 6 (10.0%); 10 (16.7%) had no retinopathy.

LVH severity was strongly correlated with hypertensive retinopathy grade ($\rho = 0.71$, 95% CI 0.56–0.82; $p < 0.001$). Exploratory correlations were similar in direction and magnitude across sex and age subgroups, although their wide CIs indicated limited precision (Table 3). In the sensitivity analysis restricted to participants with LVH, the correlation was $\rho = 0.60$ (95% CI 0.39–0.75; $p < 0.001$).

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DISCUSSION

The concordance between cardiac structural and retinal microvascular damage is biologically plausible. Current hypertension guidelines recognise cardiac and retinal abnormalities as manifestations of hypertension-mediated organ damage (18). Pressure overload promotes cardiac hypertrophy (19), whereas vascular remodelling may contribute to retinal microvascular injury (20).

Previous evidence linking retinal microvascular abnormalities with cardiac remodelling has been inconsistent across populations, disease stages, and retinal measurement methods. Retinal arteriolar narrowing has been associated with LVH (13), whereas no significant association between hypertensive retinopathy and LVH was found in a cohort of Congolese patients with hypertension (11). Differences in referral patterns, duration of hypertension, organ involvement, and assessment methods may partly explain this heterogeneity. Retinal and cardiac damage should therefore not be assumed to progress in parallel.

Interpretation of blood pressure control is limited by reliance on a single office assessment and the absence of ambulatory monitoring. The study therefore characterises patients with established hypertensive heart disease but cannot determine long-term control, treatment adequacy, or whether fundoscopy predicts the development of LVH.

Fundoscopy is widely available and may help identify patients who require broader evaluation for hypertension-mediated organ damage, especially where echocardiography is not readily accessible (21). Nevertheless, this study did not assess the diagnostic accuracy of retinopathy grade for detecting LVH; fundoscopy should therefore be considered complementary rather than a substitute for echocardiography.

This study has several limitations. The cross-sectional design precludes causal and temporal inference, while the modest, single-centre sample limits precision, particularly in subgroup analyses, and prevents adequate adjustment for diabetes, dyslipidaemia, smoking, treatment,

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and other potential confounders. Echocardiography could precede fundoscopy by up to six months, during which blood pressure or treatment may have changed. Retinopathy was graded by one examiner using a subjective ordinal classification, preventing assessment of interobserver reliability (14). Qualitative fundoscopy provides less precise microvascular assessment than quantitative retinal imaging (22), and the absence of ambulatory blood pressure monitoring may have introduced measurement error. Larger multicentre studies using contemporaneous cardiac and quantitative retinal assessments are required.

CONCLUSION

In this cross-sectional cohort, LVH severity was strongly associated with hypertensive retinopathy grade. The findings support co-occurrence of cardiac and retinal hypertension-mediated organ damage but do not establish parallel progression or causality. Fundoscopy may complement comprehensive target-organ assessment, although it cannot replace echocardiography. Larger multicentre studies with quantitative retinal imaging and adjustment for confounders are needed.

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CONFLICTS OF INTEREST

None to declare.

AUTHOR CONTRIBUTIONS (CRediT)

Conceptualisation: RA, DD; methodology: RA, AKA; validation: AKA; formal analysis: DD; investigation: DD, NA, MAOPC; data curation: DD, NA; resources: MAOPC; writing—original draft: DD; writing—review and editing: RA, NA, AKA; supervision: RA. All authors approved the final version of the manuscript.

ETHICS STATEMENT

The Research Ethics Committee of RSUP Dr Mohammad Hoesin/Faculty of Medicine, Universitas Sriwijaya approved the study (No. 02/PL.KEPK.FK.UNSRI/2024). All participants provided written informed consent. The study was conducted in accordance with the Declaration of Helsinki.

DATA AVAILABILITY STATEMENT

The de-identified dataset analysed during this study is available from the corresponding author upon reasonable request, subject to institutional and ethics approval.

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TABLES AND FIGURES

Table 1. Demographic, clinical, treatment, and echocardiographic characteristics (n = 60)

Characteristic	Value
Age, years	56.3 ± 8.9
Sex	
Male	28 (46.7)
Female	32 (53.3)
Body mass index, kg/m²	27.4 ± 3.8
Duration of hypertension, years	12.4 ± 6.8
Systolic blood pressure, mmHg	158 ± 16
Diastolic blood pressure, mmHg	96 ± 11
Diabetes mellitus	12 (20.0)
Dyslipidaemia	26 (43.3)
Current smoking	10 (16.7)
Total cholesterol, mg/dL	208 ± 38
LDL cholesterol, mg/dL	132 ± 34
HDL cholesterol, mg/dL	46 ± 12
Triglycerides, mg/dL	164 ± 72
Antihypertensive regimen	
Monotherapy	28 (46.7)
Two agents	18 (30.0)
≥ 3 agents	14 (23.3)
Drug class	
ACE inhibitors	10 (16.7)
Angiotensin receptor blockers	36 (60.0)
Calcium channel blockers	44 (73.3)
Beta-blockers	8 (13.3)
Diuretics	22 (36.7)
Echocardiographic measures	
LVMI, g/m²	132 ± 28
LVEF, %	63 ± 8
RWT	0.48 ± 0.08
LVH severity	
Absent	8 (13.3)
Mild	16 (26.7)
Moderate	22 (36.7)
Severe	14 (23.3)
Left ventricular geometry	
Concentric hypertrophy	38 (63.3)
Eccentric hypertrophy	14 (23.3)
Concentric remodelling	4 (6.7)
Normal geometry	4 (6.7)

Values are mean±SD or n (%).

ACE, angiotensin-converting enzyme; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVEF, left ventricular ejection fraction; LVH, left ventricular hypertrophy; LVMI, left ventricular mass index; RWT, relative wall thickness; SD, standard deviation

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Table 2. Cross-tabulation of left ventricular hypertrophy severity and hypertensive retinopathy grade

LVH severity	No retinopathy	Grade I	Grade II	Grade III	Grade IV	Total
Absent	6 (10.0)	2 (3.3)	0 (0.0)	0 (0.0)	0 (0.0)	8 (13.3)
Mild	3 (5.0)	8 (13.3)	4 (6.7)	1 (1.7)	0 (0.0)	16 (26.7)
Moderate	1 (1.7)	4 (6.7)	10 (16.7)	5 (8.3)	2 (3.3)	22 (36.7)
Severe	0 (0.0)	1 (1.7)	3 (5.0)	6 (10.0)	4 (6.7)	14 (23.3)
Total	10 (16.7)	15 (25.0)	17 (28.3)	12 (20.0)	6 (10.0)	60 (100.0)

Values are n (% of the total cohort).

Spearman's $\rho = 0.71$; $p < 0.001$

LVH, left ventricular hypertrophy

Table 3. Spearman correlations between left ventricular hypertrophy severity and hypertensive retinopathy grade

Variable (subgroup)	n	ρ (95% CI)	p
Overall cohort	60	0.71 (0.56–0.82)	< 0.001
Male	28	0.68 (0.41–0.84)	< 0.001
Female	32	0.74 (0.53–0.87)	< 0.001
Age ≤ 55 years	31	0.74 (0.53–0.87)	< 0.001
Age > 55 years	29	0.62 (0.33–0.81)	< 0.001
LVH-positive participants	52	0.60 (0.39–0.75)	< 0.001

CI, confidence interval (calculated using Fisher's z-transformation); LVH, left ventricular hypertrophy; ρ, Spearman's rank correlation coefficient

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