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INDEPENDENT AND CUMULATIVE EFFECTS OF AGE, GENDER, HYPERTENSION DURATION, AND BODY MASS INDEX ON HYPERTENSIVE RETINOPATHY SEVERITY IN NORMOGLYCEMIC SUBJECTS: A TERTIARY CARE CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Hypertensive retinopathy represents a significant microvascular complication of essential hypertension, contributing to vision-threatening consequences and cardiovascular morbidity. While sustained blood pressure elevation is the primary pathogenic mechanism, the independent and cumulative contributions of specific demographic, anthropometric, and metabolic factors on retinopathy severity classification remain incompletely characterized in normoglycemic hypertensive populations. The objective is to examine the independent and cumulative influence of age, gender, disease duration, and body mass index on the development and severity classification of hypertensive retinopathy in normoglycemic hypertensive subjects.

Materials and Methods: A cross-sectional study of 140 normoglycemic hypertensive participants was conducted at a tertiary care institution. Participants underwent comprehensive ophthalmologic examination with fundoscopic assessment employing the modified Keith-Wagener-Barker classification system. Anthropometric measurements, disease duration documentation, and fasting serum lipid profiling were performed. Statistical analysis utilized chi-square test for categorical variables and one-way ANOVA for continuous variables, with $p<0.05$ considered statistically significant.

Results: Hypertensive retinopathy was present in 103 participants (73.6%). Duration of hypertension exceeding 10 years demonstrated the strongest association with retinopathy presence (94.2% vs. 24.3%; $p<0.000001$). Obesity (BMI ≥30 kg/m²) was observed in 77.7% of retinopathic versus 18.9% of non-retinopathic individuals ($p=0.0001$). Dyslipidemia parameters exhibited progressive escalation across retinopathy grades: total cholesterol increased from 192±28 mg/dL (no retinopathy) to 285±15 mg/dL (Grade IV), triglycerides from 138±35 to 245±12 mg/dL, and LDL cholesterol from 118±22 to 210±8 mg/dL. Age and gender showed non-significant associations ($p=0.255$ and $p=1.000$, respectively).

Conclusion: Hypertension duration and obesity represent the strongest independent determinants of hypertensive retinopathy severity. Comprehensive cardiovascular risk management incorporating blood pressure control, weight reduction, and aggressive lipid-lowering therapy is essential for preventing progressive retinal microvascular disease.

Keywords: Hypertensive retinopathy, Lipid profile, Body mass index, Microvascular complications and Tertiary care.

INTRODUCTION

Hypertension represents a leading global public health challenge, affecting approximately 1.13 billion individuals worldwide and contributing to an estimated 10.4 million deaths annually.[1,2] Hypertensive retinopathy (HR), a microvascular complication of chronic hypertension, develops through sustained elevation of intraocular perfusion pressure, resulting in endothelial dysfunction, vascular remodeling, and progressive retinal ischemic damage.[3,4] The retina serves as a unique window for observing systemic vascular alterations, and hypertensive changes in retinal vessels frequently correlate with cardiovascular and cerebrovascular complications.[5,6] While the association between blood pressure elevation and retinal vascular damage has been well-documented, the independent contributions of specific demographic and anthropometric determinants—namely age, gender, disease duration, and adiposity—on retinopathy severity classification remain incompletely characterized in normoglycemic hypertensive populations.[7,8]

Prior investigations have identified hypertension duration as a critical predictor of microvascular complications, with obesity emerging as a significant modifiable risk factor. However, limited research has systematically examined the cumulative influence of these variables on retinopathy grade stratification in glucose-tolerant individuals.[9,10] Such investigation is clinically relevant, as accurate identification of independent demographic and morphometric risk factors facilitates targeted therapeutic interventions and prognostic stratification. This study therefore aimed to examine the independent and cumulative effects of age, gender, hypertension duration, and body mass index on the development and severity classification of hypertensive retinopathy in normoglycemic hypertensive subjects attending a tertiary care institution.

MATERIALS AND METHODS

A cross-sectional observational study was conducted at the Department of Ophthalmology of a tertiary care hospital in Indore, Madhya Pradesh, over a 12-month period following ethics committee approval. The study enrolled patients presenting to the outpatient departments of Ophthalmology and Medicine, as well as those referred from other departments of the institution.

A total of 140 participants with essential hypertension were systematically recruited for this investigation. Inclusion criteria comprised: (1) documented diagnosis of essential hypertension or newly diagnosed hypertension at the time of presentation; (2) age ≥40 years; and (3) provision of written informed consent in the participant's native language. Exclusion criteria encompassed: (1) pre-existing diabetes mellitus; (2) high myopia or refractive error exceeding −8.0 dioptres; (3) media opacities precluding adequate fundus visualization; (4) pregnancy; (5) diagnosed glaucoma; (6) chronic kidney disease; (7) hemoglobin levels indicative of anemia; and (8) other primary retinal vascular pathologies unrelated to hypertension.

Following provision of informed consent, a structured proforma was completed for each participant, documenting: demographic characteristics (age, gender); temporal disease parameters (duration of hypertension in years); anthropometric measurements (weight and height for body mass index calculation); and fasting serum lipid parameters.

Operational Definitions

1. Hypertension

Hypertension was operationally defined as systolic blood pressure exceeding 140 millimetres of mercury (mmHg) and/or diastolic blood pressure surpassing 90 mmHg, assessed on two independent measurement occasions, or documented current utilization of antihypertensive pharmacologic agents.[11]

2. Duration of Hypertension

Duration of hypertension was operationally characterized as the temporal interval, measured in complete years, extending from the date of initial clinical diagnosis through the enrolment date for study participation.[11]

3. Body Mass Index and Obesity Classification

Body mass index (BMI) was calculated as the ratio of body weight in kilograms to the square of height in meters (kg/m²). Obesity classification adhered to World Health Organization (WHO) criteria, wherein BMI exceeding 30 kg/m² constituted obesity, while BMI values of 30 kg/m² or less were classified as non-obese.[12]

4. Normoglycemia

Normoglycemia was operationally confirmed through documentation of fasting plasma glucose concentration not exceeding 100 mg/dL, coupled with the absence of diagnostic criteria fulfilling diabetes mellitus classification, thereby systematically excluding glucose-intolerant individuals from investigation.[13]

5. Lipid Profile Parameters

Serum lipid parameters were assessed through fasting venous blood collection following a minimum 12-hour overnight fasting period, with measurements performed utilizing standardized laboratory methodology. Operational definitions comprised the following:[12,13]

- Total Cholesterol (TC): Cumulative serum cholesterol concentration derived from all lipoprotein fractions, classified as elevated when ≥240 mg/dL.
- Low-Density Lipoprotein Cholesterol (LDL-C): Serum LDL cholesterol concentration calculated according to the Friedewald equation, designated as elevated when ≥160 mg/dL.

- High-Density Lipoprotein Cholesterol (HDL-C): Serum HDL cholesterol concentration measured through direct quantitative methodology, classified as low when <35 mg/dL.
- Triglycerides (TG): Fasting serum triglyceride concentration determined via standardized assay procedures, designated as elevated when ≥150 mg/dL.
- HDL/LDL Ratio: Composite lipid parameter derived through division of HDL-C by LDL-C concentrations, with ratios ≤2.5 indicating atherogenic lipid profile phenotypes.

6. Hypertensive Retinopathy

Hypertensive retinopathy (HR) was operationally defined as retinal microvascular alterations directly attributable to sustained systemic hypertension, diagnosed through indirect ophthalmoscopic examination performed following pupillary dilation with 1% Tropicamide solution.[14]

7. Hypertensive Retinopathy Severity Grading

Retinopathy severity classification employed the modified Keith-Wagener-Barker classification system, stratified into the following categories:7

- Grade 0 (No Hypertensive Retinopathy): Complete absence of detectable retinal vascular abnormalities upon ophthalmoscopic examination.
- Grade I (Mild): Generalized or focal arteriolar narrowing characterized by diminished arterial calibre, manifesting an arteriole-to-venule ratio approximating 1:2 or less, without supplementary vascular alterations.
- Grade II (Moderate): Definite focal arteriolar constriction and arteriovenous nicking, representing locations where crossing arterioles mechanically compress underlying venules, potentially indicating vascular wall remodelling and increased arteriolar rigidity.
- Grade III (Severe): All Grade II characteristics combined with retinal haemorrhages, cotton-wool spots indicating nerve fibre layer ischemia, and hard exudates representing lipid deposits from vascular leakage, collectively signifying substantial microvascular compromise.
- Grade IV (Malignant/Accelerated): All Grade III characteristics plus optic disc oedema (papilledema), representing acute hypertensive vascular crisis with blood-retinal barrier breakdown and severe ischemic microvascular damage.

Ophthalmologic Examination: All participants underwent comprehensive ophthalmic evaluation including visual acuity assessment, intraocular pressure measurement, and dilated fundoscopic examination using a 20-dioptre indirect ophthalmoscope lens after pharmacologic pupillary dilation with 1% Tropicamide. Two independent ophthalmologists performed masked retinopathy grading to ensure diagnostic consistency and minimize observer bias.

Statistical Analysis: Data were entered into Microsoft Excel spreadsheets and analyzed using Open Epi statistical software (version 3.01). Continuous variables were summarized as mean ± standard deviation (SD), while categorical variables were expressed as frequencies and percentages. Categorical associations were evaluated using the chi-square (χ^2) test, with a predetermined alpha level of 0.05 defining statistical significance. For continuous variables demonstrating normal distribution, comparison across multiple HR severity groups (no retinopathy, Grade I, II, III, IV) was performed using one-way analysis of variance (ANOVA) with post-hoc testing for pairwise comparisons. Pearson correlation coefficients were calculated to assess the strength and direction of linear relationships between continuous lipid parameters and retinopathy severity grade. A two-sided p-value <0.05 was considered statistically significant for all statistical tests.

RESULTS

The cohort demonstrated a predominant age range between 50–79 years, representing 67.9% of participants, with nearly equivalent gender distribution (46.4% male; 53.6% female). Notably, 56.5% of subjects had hypertension duration exceeding 10 years, while 57.1% met obesity criteria. Overall retinopathy prevalence reached 73.6%. [Table 1]

[Table 2] illustrates a statistically highly significant dose-response relationship was established between prolonged hypertension duration and retinopathy occurrence (p<0.000001). Retinopathy prevalence escalated progressively: 40.0% in individuals with ≤5 years of disease, 67.7% in the 5–10-year interval, 76.9% in the 10–15-year interval, and universally 100.0% among patients exceeding 15 years disease duration. This demonstrates cumulative microvascular alteration with complete retinopathy penetrance in chronic disease states.

Table 1: Baseline Socio-Demographic and Clinical Characteristics of Study Participants (n = 140)

Characteristics	No HR n (%)	With HR n (%)	Total n (%)
Age Group (years)			
40–49	7 (5.0)	11 (7.9)	18 (12.9)
50–59	9 (6.4)	25 (17.9)	34 (24.3)
60–69	4 (2.9)	31 (22.1)	35 (25.0)
70–79	6 (4.3)	20 (14.3)	26 (18.6)
80–89	6 (4.3)	11 (7.9)	17 (12.1)
≥89	5 (3.6)	5 (3.6)	10 (7.1)
Gender			

Male	17 (24.3)	48 (68.6)	65 (46.4)
Female	20 (28.6)	55 (71.4)	75 (53.6)
Hypertension Duration (years)			
≤5	18 (12.9)	12 (8.6)	30 (21.4)
5–10	10 (7.1)	21 (15)	31 (22.1)
10–15	9 (6.4)	30 (21.4)	39 (27.9)
>15	0 (0.0)	40 (28.6)	40 (28.6)
Body Mass Index Classification			
Non-obese (≤30 kg/m²)	30 (21.4)	30 (21.4)	60 (42.9)
Obese (>30 kg/m²)	7 (5.0)	73 (52.1)	80 (57.1)
Total	37 (26.4)	103 (73.6)	140 (100.0)

Table 2: Association of Hypertension Duration with Presence and Severity of Hypertensive Retinopathy (n = 140)

Duration of Hypertension (years)	No HR n (%)	With HR n (%)	Total n (%)	p-value
≤5	18 (12.9)	12 (8.6)	30 (21.4)	<0.000001
5–10	10 (7.1)	21 (15.0)	31 (22.1)	
10–15	9 (6.4)	30 (21.4)	39 (27.9)	
>15	0 (0.0)	40 (28.6)	40 (28.6)	
Total	37 (26.4)	103 (73.6)	140 (100)	

Table 3: Escalation of Total Cholesterol Across Hypertensive Retinopathy Severity Grades (n = 140)

Retinopathy Grade	TC <200 mg/dL n (%)	TC 200–239 mg/dL n (%)	TC ≥240 mg/dL n (%)	Total n (%)	p-value
No retinopathy	29 (20.7)	7 (5.0)	1 (0.7)	37 (26.4)	<0.0001
Grade I	18 (10.7)	7 (5.0)	3 (2.1)	28 (20.0)	
Grade II	17 (12.1)	8 (5.7)	21 (15.0)	46 (32.9)	
Grade III	6 (4.3)	4 (2.9)	17 (12.1)	27 (19.3)	
Grade IV	0 (0.0)	0 (0.0)	2 (1.4)	2 (1.4)	
Total	70 (50.0)	26 (18.6)	44 (31.4)	140 (100)	

[Table 3] depicts a highly significant association emerged between elevated total cholesterol and retinopathy severity (p<0.0001). Among patients without retinopathy, 78.6% maintained total cholesterol <200 mg/dL, whereas elevated cholesterol (≥240 mg/dL) affected only 2.7%. Total cholesterol ≥240 mg/dL was present in 45.7% of Grade II, 63.0% of Grade III, and 100% of Grade IV cases. This progressive escalation underscores hypercholesterolemia as critical determinant of retinopathy severity.

Hypertriglyceridemia (TG ≥150 mg/dL) demonstrated the strongest association with retinopathy occurrence and severity (p=0.0001). Among participants without retinopathy, 83.8% had triglycerides <150 mg/dL. Elevated triglycerides were markedly prevalent in retinopathy cases: 53.6% in Grade I, 78.3% in Grade II, 85.2% in Grade III, and 100% in Grade IV. This escalation establishes hypertriglyceridemia as particularly potent lipid determinant of retinal microvascular damage.
[Table 4]

Table 4: Escalation of Triglycerides Across Hypertensive Retinopathy Severity Grades (n = 140)

Retinopathy Grade	TG <150 mg/dL n (%)	TG ≥150 mg/dL n (%)	Total n (%)	p-value
No retinopathy	31 (22.1)	6 (4.3)	37 (26.4)	<0.0001
Grade I	13 (9.3)	15 (10.7)	28 (20.0)	
Grade II	10 (7.1)	36 (25.7)	46 (32.9)	
Grade III	4 (2.9)	23 (16.4)	27 (19.3)	
Grade IV	0 (0.0)	2 (1.4)	2 (1.4)	
Total	58 (41.4)	82 (58.6)	140 (100)	

A multi-line graph [Figure 1] illustrates the striking dose-response escalation of atherogenic lipid parameters—total cholesterol ≥240 mg/dL, triglycerides ≥150 mg/dL, and LDL cholesterol ≥160 mg/dL—alongside progressive deterioration of the protective HDL/LDL ratio across advancing retinopathy severity. The visualization demonstrates progressively steeper gradients in atherogenic parameters from no retinopathy through Grade IV, establishing clear and powerful association between lipid profile intensity and retinal microvascular deterioration.

Obesity demonstrated compelling progressive association with increasing retinopathy severity (p<0.0001). Among patients without retinopathy, 81.1% maintained non-obese status, whereas obesity prevalence escalated: 53.6% in Grade I, 69.6% in Grade II, 88.9% in Grade III, and 100% in Grade IV. This progressive concentration in advanced stages establishes excessive adiposity as critical modifiable determinant of hypertensive retinal microvascular damage. [Table 5]

Table 5: Distribution of Body Mass Index Classification Across Hypertensive Retinopathy Severity Grades (n = 140)

Retinopathy Grade	Non-obese (BMI ≤30) n (%)	Obese (BMI >30) n (%)	Total n (%)	p-value
No retinopathy	30 (21.4)	7 (5.0)	37 (26.4)	<0.0001
Grade I	13 (9.3)	15 (10.7)	28 (20.0)	
Grade II	14 (10.0)	32 (22.9)	46 (32.9)	
Grade III	3 (2.1)	24 (17.1)	27 (19.3)	
Grade IV	0 (0.0)	2 (1.4)	2 (1.4)	
Total	60 (42.9)	80 (57.1)	140 (100)	

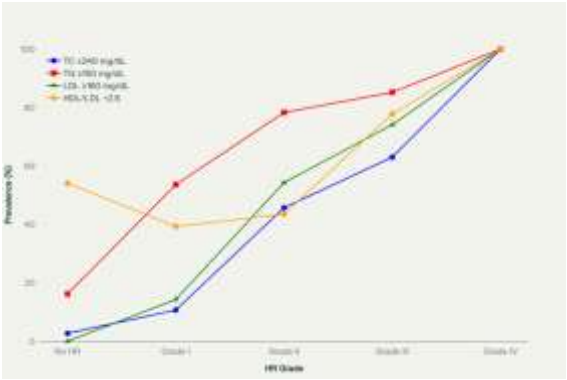

Figure 1: Progressive Alterations in Lipid Profile Parameters Across Hypertensive Retinopathy Severity Grades

A dual-axis combination chart [Figure 2] demonstrates the progressive dominance of obesity (blue columns) achieving near-universal prevalence in advanced stages, alongside the exclusive occurrence of prolonged hypertension duration ≥15 years (red line) in Grade IV category. The visualization emphasizes the synergistic interaction between sustained blood pressure elevation and adiposity-related vascular dysfunction in determining the severity trajectory of hypertensive retinal microvascular disease.

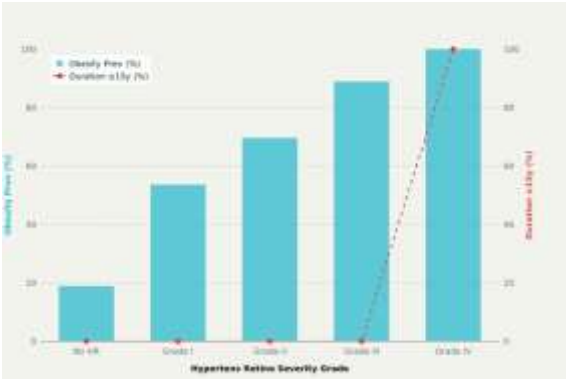

Figure 2: Cumulative Impact of Obesity Prevalence and Prolonged Hypertension Duration (≥15 Years) on Retinopathy Severity

DISCUSSION

The present cross-sectional investigation systematically evaluated the independent and cumulative determinants of hypertensive retinopathy severity in 140 normoglycemic hypertensive individuals. The findings revealed hypertension duration and obesity as the most potent predictors of retinopathy manifestation and progression, with dyslipidaemia parameters exhibiting pronounced dose-dependent associations.

Hypertension duration emerged as the single most statistically robust determinant ($p<0.000001$), demonstrating a compelling dose-response relationship with retinopathy prevalence escalating from 40% in those with ≤5 years disease to 100% among patients exceeding 15 years of sustained pressure elevation. Van Leiden et al. (2002) investigated 2,484 participants in the Hoorn Study and documented that hypertension, dyslipidaemia, and obesity independently predicted elevated retinopathy prevalence, a finding corroborated by the present institutional cohort.[15] The temporal accumulation of microvascular injury from chronic pressure elevation represents the mechanistic basis for this powerful association. Hegde et al. (2013) similarly reported proportional relationships between disease duration and retinopathy manifestation, with 84% prevalence in individuals exceeding 10 years disease duration versus 48% in those below 5 years, supporting the cumulative vascular injury hypothesis evident in our results.[16]

Obesity (BMI >30 kg/m²) demonstrated striking prevalence across ascending retinopathy severity grades, affecting 18.9% of non-retinopathic versus 53.6%, 69.6%, 88.9%, and 100% of Grade I through Grade IV cases respectively ($p<0.0001$). The near-universal adiposity concentration in advanced retinopathy stages identifies excessive adiposity as a critical modifiable determinant warranting aggressive therapeutic intervention. This progressive relationship extends observations from prior literature (Gupta et al. and Xiao et al.) examining metabolic contributors to microvascular complications.[17,18]

Dyslipidaemia parameters exhibited striking dose-dependent escalation across retinopathy severity progression. Singh et al. (2013) documented positive correlations between elevated total cholesterol and LDL cholesterol with hypertensive retinopathy severity, findings substantially reinforced by the present investigation.[19] Total cholesterol ≥240 mg/dL increased from 2.7% in non-retinopathic subjects to 63.0% in Grade III and 100% in Grade IV cases ($p<0.0001$), representing a 48.4% absolute elevation across disease progression. Alattas et al. (2022) identified elevated triglycerides as significantly associated with retinopathy progression in their retrospective institutional review,[20] and our cohort similarly demonstrated triglycerides ≥150 mg/dL in 85.2% of Grade III and 100% of Grade IV cases compared to only 16.2% of

non-retinopathic individuals ($p<0.0001$). LDL cholesterol ≥160 mg/dL exhibited significant discriminatory capacity, progressing from 0% in the no-retinopathy group to 100% in Grade IV cases ($p<0.0001$). Badhu et al. (2003) previously demonstrated that elevated serum LDL independently predicted hypertensive retinopathy presence with 67% prevalence in retinopathic versus 23% in non-retinopathic patients, substantially lower than our findings, possibly reflecting differences in case severity distribution.[21]

The composite HDL/LDL ratio emerged as a superior lipid risk marker ($p<0.001$) compared to isolated HDL levels ($p=0.898$), with ratios ≤2.5 present in 92.2% of retinopathic versus 54.1% of non-retinopathic participants. Cheung et al. (2017) identified elevated serum lipid parameters as correlates of narrower retinal arteriolar calibres, suggesting dyslipidaemia-induced vascular remodelling represents a generalized phenomenon independent of age strata.[22] The mechanistic intersection between lipid-mediated endothelial dysfunction and hypertensive microvascular injury establishes dyslipidaemia as an integral pathogenic contributor to progressive retinopathy. The cumulative evidence establishes hypertensive retinopathy as a multifactorial complication wherein temporal disease accumulation, adiposity burden, and atherogenic lipid phenotypes conjointly drive progressive retinal microvascular deterioration. Comprehensive cardiovascular risk management incorporating blood pressure control, aggressive weight reduction strategies, and intensified lipid-lowering pharmacotherapy represents an essential preventive approach for hypertensive retinopathy in clinical practice.

CONCLUSION

This institutional study establishes hypertension duration and obesity as the strongest determinants of hypertensive retinopathy severity in normoglycemic subjects, while dyslipidaemia exhibits striking dose-dependent associations with advancing disease progression. Integrated cardiovascular risk management incorporating blood pressure control, weight reduction, and aggressive lipid-lowering therapy represents an essential preventive strategy for mitigating progressive retinal microvascular complications.

Strengths and Limitations

Strengths: Masked retinopathy grading by two independent ophthalmologists minimized observer bias; systematic lipid profiling utilized standardized laboratory methodology; comprehensive demographic documentation. Limitations: Cross-sectional design precludes temporal causality; single-centre recruitment limits generalizability; absence of advanced retinal imaging modalities restricts mechanistic insight.

Recommendations: Routine lipid screening should be systematically integrated into hypertensive patient management protocols; high-risk dyslipidemic hypertensive subjects require intensified lipid-lowering therapy and regular ophthalmologic surveillance.

Relevance of the Study: This investigation addresses a clinically significant evidence gap by systematically examining cumulative demographic-anthropometric influences on hypertensive retinopathy in normoglycemic populations, thereby informing evidence-based risk stratification algorithms in tertiary care settings.

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