

Research Article

Association of Atherogenic Lipid Profile with Blood Pressure Control in Adults with Essential Hypertension: a Cross-Sectional Study

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ABSTRACT

Background: Routine lipid panels contain information that can be integrated into atherogenic indices, but the relation of these composite measures to contemporaneous blood pressure control in established essential hypertension has not been fully resolved.

Objective: To determine whether conventional lipid fractions and calculated atherogenic indices differ according to blood pressure control and to quantify their independent associations with uncontrolled hypertension.

Methods: We performed an analytical cross-sectional investigation of 100 adults with essential hypertension recruited consecutively at Rashid Latif Khan University Medical College, Lahore, Pakistan, from January 2025 through February 2026. Office blood pressure below both 140 mmHg systolic and 90 mmHg diastolic defined the controlled group. Fasting total cholesterol, LDL-C, HDL-C, and triglycerides were measured, after which non-HDL-C, TG/HDL-C, Castelli indices I and II, the atherogenic coefficient, and the atherogenic index of plasma (AIP) were derived. Between-group testing, correlation analysis, multivariable logistic regression, and receiver operating characteristic analysis were used.

Results: Fifty-four participants were men, and 46 were women; 55 participants (55.0%) had uncontrolled blood pressure. Compared with the controlled group, the uncontrolled group had higher triglycerides (187.6 vs 143.2 mg/dL), lower HDL-C (39.9 vs 46.8 mg/dL), greater TG/HDL-C (4.95 vs 3.16), and greater AIP (0.33 vs 0.13; all $p < 0.001$). AIP showed the largest correlation with systolic pressure ($r = 0.51$, $p < 0.001$). In adjusted analysis, a 0.1-unit rise in AIP corresponded to an adjusted odds ratio of 1.53 (95% CI 1.20-1.94) for uncontrolled hypertension. Its ROC AUC was 0.79.

Conclusion: Uncontrolled hypertension in this cohort clustered with a more atherogenic lipid phenotype. Indices incorporating triglycerides and HDL-C, especially AIP and TG/HDL-C, conveyed stronger cross-sectional discrimination than LDL-C alone and may complement routine cardiometabolic assessment.

Keywords: Atherogenic Dyslipidemia, Atherogenic Index of Plasma, Essential Hypertension, High-Density Lipoprotein Cholesterol, Triglycerides, Blood Pressure Control.

INTRODUCTION

Hypertension and dyslipidemia often coexist within the same cardiometabolic phenotype, yet clinical assessment commonly treats them as parallel rather than interacting risk domains¹. This distinction is important because the vascular environment in which blood pressure is regulated is not only influenced by blood pressure, but also by lipid transport, endothelial function, insulin sensitivity, and vascular remodeling. Therefore, persistent increases in pressure in a metabolically inhospitable environment may be a more comprehensive vascular phenotype than that captured by office blood pressure².

While low-density lipoprotein cholesterol (LDL-C) remains a central part of the assessment of atherosclerotic risk, one level of LDL-C does not suffice to characterize the overall distribution of the abnormalities in triglyceride-rich lipoproteins and HDL³. This is especially important when hypertriglyceridemia and/or low HDL-C, obesity, and/or insulin-resistant physiology are involved. Several of these features can be summarized by calculated lipid metrics without the need for additional testing. Non-HDL-C captures cholesterol carried by atherogenic particles; TG/HDL-C expresses the balance between triglyceride burden and HDL; and the Castelli indices and atherogenic

coefficient place proatherogenic fractions in relation to HDL⁴.

AIP uses a different mathematical equation to represent the same metabolic axis since it is the log₁₀ of the molar ratio of TG: HDL-C. Higher values thus detect a pattern of more triglyceride exposure in relation to HDL⁵. This pattern may be associated with sub-optimal insulin function, modified lipoprotein remodeling, and sub-optimal LDL particle distribution. These metabolic characteristics are also shared by mechanisms of pressure regulation, rendering AIP a feasible way to reflect the cardiometabolic state in which blood pressure is hard to control⁶.

The link between a negative lipid phenotype and high arterial pressure may be established via several biological pathways⁸. Triglyceride-rich lipoproteins, diminished HDL-related activity, visceral adiposity, and insulin resistance may coexist with reduced nitric-oxide bioavailability, enhanced sympathetic activity, renal sodium retention, oxidative stress, and higher peripheral resistance. There are progressive endothelial dysfunction and arterial stiffening that can further contribute to systolic load. Conversely, long-standing pressure elevation may accelerate vascular injury, so a cross-sectional association may reflect reciprocal vascular and metabolic processes rather than a one-way causal pathway⁹.

Because every index evaluated here can be obtained from an ordinary fasting lipid panel, their potential clinical value lies in risk characterization rather than added laboratory burden. Evidence is stronger for the relation of dyslipidemia to the development of hypertension than for head-to-head comparison of several derived lipid indices among patients who already have established hypertension¹⁰. We therefore examined whether conventional lipid fractions and composite atherogenic measures differed between adults with controlled and uncontrolled office blood pressure, how these measures related to systolic and diastolic pressure, and whether their associations with uncontrolled hypertension persisted after adjustment for relevant clinical covariates.

MATERIALS AND METHODS

Study Design and Setting:

A single-center analytical cross-sectional design was conducted at Rashid Latif Khan University Medical College, Lahore, Pakistan, between January 2025 and February 2026. Consecutive eligible adults with an established diagnosis of

essential hypertension were approached until the prespecified sample of 100 participants was reached.

Study Population:

Adults aged 18 years or older were eligible when both office blood pressure measurements and a fasting lipid profile were available. Men and women were included. We excluded secondary hypertension, pregnancy, acute coronary syndrome, recent acute stroke, decompensated heart failure, severe hepatic or renal disease, active systemic infection, familial dyslipidemia, and records lacking the clinical or laboratory information required for the planned analyses.

Clinical and Blood Pressure Assessment:

Age, sex, body mass index (BMI), smoking, diabetes mellitus, duration of hypertension, antihypertensive treatment, and lipid-lowering therapy usage were documented. BP was measured using a validated automated device after 5 minutes of sitting. Two measurements separated by 1–2 minutes were averaged. Participants were assigned to the controlled category only when both systolic pressure was <140 mmHg and diastolic pressure was <90 mmHg; all others were classified as uncontrolled.

Biochemical Assessment and Atherogenic Indices:

Venous samples were collected after an overnight fast of 8–12 hours. Total cholesterol, triglycerides, HDL-C, and LDL-C were quantified using standard enzymatic laboratory methods. Non-HDL-C was obtained by subtracting HDL-C from total cholesterol. Additional measures were calculated as follows: TG/HDL-C; Castelli risk index I, total cholesterol/HDL-C; Castelli risk index II, LDL-C/HDL-C; and atherogenic coefficient, (total cholesterol – HDL-C)/HDL-C. AIP was defined as log₁₀(TG/HDL-C) after conversion of both triglycerides and HDL-C to mmol/L.

Study Outcomes:

The primary analysis contrasted the lipid phenotype of participants with controlled versus uncontrolled office blood pressure. Secondary analyses quantified correlations of lipid variables with systolic and diastolic pressure and estimated adjusted associations between selected lipid measures and uncontrolled hypertension.

Statistical Analysis:

Analyses were conducted in IBM SPSS Statistics 26.0. Continuous data were summarized by mean $\pm$ standard deviation or median with interquartile range according to distribution; categorical data were summarized by count and percentage. Between-group comparisons used independent-samples t tests, Mann–Whitney U tests, chi-square tests, or Fisher exact tests as appropriate. Pearson or Spearman coefficients were selected for correlation analysis according to variable distribution. Binary logistic regression provided adjusted odds ratios for uncontrolled hypertension. Models incorporated age, sex, BMI, diabetes mellitus, smoking, hypertension duration, antihypertensive treatment burden, and lipid-lowering therapy. Because the derived lipid indices share constituent variables and are highly correlated, the principal indices were entered in separate adjusted models. Discrimination was summarized by ROC area under the curve (AUC). All tests were two-sided, with $p < 0.05$ defining statistical significance.

Ethical Considerations:

Institutional ethical clearance was obtained from Rashid Latif Khan University Medical College, Lahore, Pakistan. All participants

provided written informed consent and study information was processed under procedures designed to maintain participant confidentiality.

RESULTS

Participant Characteristics and Blood Pressure Control:

The analytic cohort comprised 100 adults: 54 men (54.0%) and 46 women (46.0%). There were 45 participants (45.0%) with office blood pressure that met the study criteria for control, while 55 (55.0%) had uncontrolled blood pressure.

The blood pressure categories were accompanied by clinical differences. Mean age was 56.2 ± 10.0 years in the uncontrolled group and 50.8 ± 8.3 years in the controlled group ($p = 0.003$). Mean BMI was 29.4 ± 4.0 versus 26.9 ± 3.5 kg/m² ($p = 0.001$), and mean hypertension duration was 7.6 ± 3.2 versus 6.1 ± 3.2 years ($p = 0.025$). Frequencies of diabetes were 38.2% in uncontrolled pressure, and 20.0% in controlled pressure group ($p = 0.048$). No significant difference was found between the sex distribution of the groups ($p = 0.778$). The entire clinical comparison is made in Table 1.

Table 1. Clinical profile according to office blood pressure control

Variable	Controlled BP (n=45)	Uncontrolled BP (n=55)	p value
Age, years	50.8 $\pm$ 8.3	56.2 $\pm$ 10.0	0.003
Male, n (%)	25 (55.6)	29 (52.7)	0.778
Female, n (%)	20 (44.4)	26 (47.3)	
BMI, kg/m ²	26.9 $\pm$ 3.5	29.4 $\pm$ 4.0	0.001
Duration of hypertension, years	6.1 $\pm$ 3.2	7.6 $\pm$ 3.2	0.025
Diabetes mellitus, n (%)	9 (20.0)	21 (38.2)	0.048
Current smoking, n (%)	9 (20.0)	15 (27.3)	0.397
≥ 2 antihypertensive agents, n (%)	15 (33.3)	28 (50.9)	0.077
Lipid-lowering therapy, n (%)	18 (40.0)	24 (43.6)	0.714
Systolic BP, mmHg	128.9 $\pm$ 6.8	149.6 $\pm$ 10.4	<0.001
Diastolic BP, mmHg	78.6 $\pm$ 5.9	90.2 $\pm$ 7.1	<0.001

Lipid Profile and Atherogenic Indices:

The lipid distribution was consistently less favorable in participants with uncontrolled blood pressure. Total cholesterol, LDL-C, triglycerides, and non-HDL-C were higher, whereas HDL-C was lower. The largest separation was seen in metrics that integrate

triglycerides with HDL-C: TG/HDL-C averaged 4.95 ± 1.62 in the uncontrolled group versus 3.16 ± 1.10 in the controlled group, and AIP averaged 0.33 ± 0.13 versus 0.13 ± 0.14 , respectively (both $p < 0.001$). Castelli indices and the atherogenic coefficient followed the same direction (Table 2).

Table 2. Fasting lipid measures by blood pressure-control category

Parameter	Controlled BP (n=45)	Uncontrolled BP (n=55)	p value
Total cholesterol, mg/dL	183.6 $\pm$ 29.4	205.8 $\pm$ 31.8	<0.001
LDL-C, mg/dL	110.7 $\pm$ 24.2	128.9 $\pm$ 27.6	0.001
HDL-C, mg/dL	46.8 $\pm$ 7.5	39.9 $\pm$ 6.8	<0.001

Triglycerides, mg/dL	143.2 ± 41.5	187.6 ± 52.8	<0.001
Non-HDL-C, mg/dL	136.8 ± 27.8	165.9 ± 31.9	<0.001
TG/HDL-C ratio	3.16 ± 1.10	4.95 ± 1.62	<0.001
Castelli risk index I	4.00 ± 0.82	5.25 ± 1.05	<0.001
Castelli risk index II	2.43 ± 0.67	3.32 ± 0.84	<0.001
Atherogenic coefficient	3.00 ± 0.82	4.25 ± 1.05	<0.001
Atherogenic index of plasma	0.13 ± 0.14	0.33 ± 0.13	<0.001

Values are mean ± SD. HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TG, triglycerides.

Correlation of Lipid Variables with Blood Pressure:

The lipid measures were found to have a coherent gradient by correlation analysis. The

largest coefficient for systolic pressure was observed with AIP ($r=0.51$, $p<0.001$), followed by TG/HDL-C ($r=0.49$, $p<0.001$) and Castelli index I ($r=0.46$, $p<0.001$). HDL-C exhibited an inverse relationship with both systolic ($r=-0.38$, $p<0.001$) and diastolic pressure ($r=-0.29$, $p=0.003$). All correlation estimates are given in Table 3.

Table 3. Correlation coefficients for lipid measures and office blood pressure

Lipid parameter	SBP, r	p value	DBP, r	p value
Total cholesterol	0.31	0.002	0.23	0.021
LDL-C	0.29	0.003	0.20	0.045
HDL-C	-0.38	<0.001	-0.29	0.003
Triglycerides	0.41	<0.001	0.33	0.001
Non-HDL-C	0.39	<0.001	0.29	0.003
TG/HDL-C ratio	0.49	<0.001	0.37	<0.001
Castelli risk index I	0.46	<0.001	0.34	0.001
Castelli risk index II	0.40	<0.001	0.29	0.003
Atherogenic index of plasma	0.51	<0.001	0.39	<0.001

DBP, diastolic blood pressure; SBP, systolic blood pressure.

Multivariable Association with Uncontrolled Hypertension:

The primary atherogenic measures remained associated with uncontrolled hypertension after covariate adjustment. As the indices have lipid components in common, they were assessed in separate multivariable models. For AIP, each 0.1-unit increment was associated with 1.53-fold higher adjusted odds of uncontrolled

hypertension (95% CI 1.20–1.94; $p<0.001$). For TG/HDL-C, the adjusted odds ratio was 1.48 per one-unit increase (95% CI 1.20–1.83; $p<0.001$). Non-HDL-C, both Castelli indices, and LDL-C also remained statistically significant (Table 4).

Discrimination was moderate rather than diagnostic. AIP produced the largest AUC (0.79), followed by TG/HDL-C (0.78) and Castelli index I (0.77); LDL-C yielded an AUC of 0.69. Figure 1 displays the adjusted effect estimates and their 95% confidence intervals.

Table 4. Adjusted relation of selected lipid measures with uncontrolled hypertension

Lipid measure	Adjusted OR	95% CI	p value	AUC
AIP, per 0.1 increase	1.53	1.20–1.94	<0.001	0.79
TG/HDL-C ratio, per 1.0	1.48	1.20–1.83	<0.001	0.78
Non-HDL-C, per 10 mg/dL	1.29	1.11–1.50	<0.001	0.74
Castelli risk index I, per 1.0	1.81	1.30–2.51	<0.001	0.77
Castelli risk index II, per 1.0	1.67	1.18–2.37	0.004	0.74
LDL-C, per 10 mg/dL	1.18	1.02–1.36	0.024	0.69

Each lipid exposure was entered in a separate logistic model adjusted for age, sex, BMI, diabetes mellitus, hypertension duration, antihypertensive medication burden, and lipid-

lowering therapy. AIP, atherogenic index of plasma; AUC, area under the curve; CI, confidence interval; OR, odds ratio.

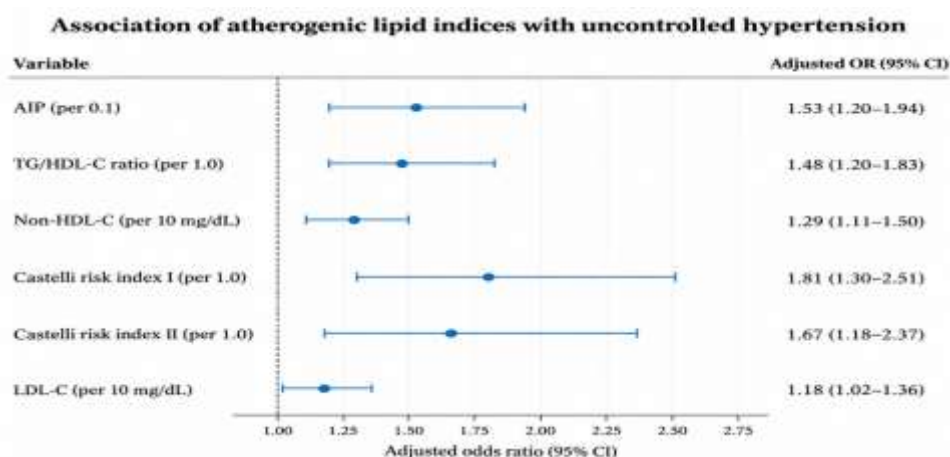


Figure 1. Multivariable effect estimates for uncontrolled hypertension. Points denote adjusted odds ratios and horizontal bars denote 95% confidence intervals. Each lipid measure was modeled separately with adjustment for age, sex, BMI, diabetes mellitus, hypertension duration, antihypertensive treatment burden, and lipid-lowering therapy.

DISCUSSION

In this cohort of adults with established essential hypertension, failure to meet the study threshold for office blood pressure control was accompanied by a broad shift toward atherogenic dyslipidemia¹. Total cholesterol, LDL-C, triglycerides and non-HDL-C were higher, and HDL-C lower, in the uncontrolled group. The between group difference was even more apparent when the lipid components were presented as composite indices. AIP, TG/HDL-C, and Castelli index I showed the clearest separation, and the same pattern was reproduced across correlation, adjusted regression, and ROC analyses^{2,3}.

AIP was the lipid variable most strongly related to systolic pressure and the one lipid variable that remained independently associated with uncontrolled hypertension after accounting for major clinical covariates⁴. This is explainable since AIP is an indicator of combined action of triglycerides and HDL-C and not of each of these fractions separately. More elevated levels may thus represent a metabolic setting of triglyceride enrichment, less HDL-related protection, insulin resistant physiology and less favorable lipoprotein distribution. Data do not support a causal link between AIP and poor pressure control but do suggest that AIP is a cardiometabolic phenotype in which uncontrolled blood pressure is more prevalent^{5,6}.

The signal for the TG/HDL-C ratio was closely parallel. It was still associated after adjustment, and its AUC was almost the same as AIP⁷. Although mathematically related, the clinical meanings of both are simple – both reflect the relative increase of triglycerides in the context

of lower HDL-C. It can coexist with endothelial dysfunction and impaired nitric-oxide signaling, increased sympathetic drive, and increased vascular resistance. This co-occurrence of adverse lipid traits and elevated office blood pressure is possible to explain with regard to common physiology⁸.

Logically, non-HDL-C and the Castelli indices were also able to distinguish between the two blood pressure-control groups, and the indices were also independently related to uncontrolled hypertension in separate adjusted models⁹. These are conceptually different from an LDL-C only approach because they represent either the cholesterol in a larger group of atherogenic particles or the net effect of atherogenic cholesterol versus HDL-C. This finding in the context of LDL-C does not affect the well-recognized importance of this lipid for lipid management, but the comparison may be pertinent to the metabolic context of hypertension in regard to the role of triglyceride-rich particles and HDL^{10,11}.

Sex was evenly distributed between the two blood pressure groups, and so this was an unlikely explanation for the lipid results. Compared to the uncontrolled group, the controlled group had fewer years of hypertension, a lower BMI, less diabetes, and was younger. These accompanying traits are important because the observed lipid associations are not just lab phenomena, but should be regarded as part of a complex metabolic and vascular profile^{12,13}.

A cross-sectional design also allows interpretation in both directions. Exposure to atherogenic lipoproteins can also lead to endothelial injury and stiffening of the arteries

and chronic increases in blood pressure can cause vascular remodeling and atherosclerotic change. These processes may be repeated over time, which may make pressure control more challenging over time, but cannot be presumed in a single assessment on the basis of temporal sequence. The present analysis thus supports phenotypic association, and not causation^{14,15}. The convenience of the assessed indices is that they do not need any other specimen or special assay. A routine lipid panel is enough to compute AIP, TG/HDL-C, non-HDL-C, and Castelli indices. They can be considered adjunctive; they may aid in identification of a more harmful cardiometabolic pattern in patients with hypertension but should not be a substitute for guideline-based determination of blood pressure or standard lipid targets^{16,17}. Several limitations define the scope of inference. The study was cross-sectional, and this does not allow the directionality or prognostic significance to be determined. The sample was small and limited to one institutional context, limiting generalizability¹⁸. The blood pressure status was defined from standardized office blood pressure measurements, and masked and white coat patterns were not addressed. There is also the possibility of residual confounding due to incomplete assessments of diet and physical activity, and to the use of medications and detailed assessment of antihypertensive drug exposure. Lastly, there was significant mathematical overlap between the derived lipid indices; the derived principal indices were not mathematically independent of each other. Multi-center longitudinal studies with serial lipid level assessments, ambulatory blood pressure, and cardiovascular outcomes are required to assess whether these indices would provide additional information over current risk factors^{19,20}.

CONCLUSION

Adults with uncontrolled essential hypertension in this study exhibited a more atherogenic lipid pattern than those whose office blood pressure was controlled. AIP and TG/HDL-C showed the most consistent relationships across group comparison, correlation, adjusted association, and ROC discrimination, whereas LDL-C alone was less discriminative in this dataset. As these indices can be derived from standard lipid measurements, they may offer a low-cost way of supplementing information for cardiometabolic phenotyping. They must be

validated longitudinally before use for prognosis or to inform treatment decisions.

Authors' Contributions:

SF contributed to study conceptualization and manuscript drafting; FF to methodology and data collection; MI to data analysis and interpretation; AN to literature review and data organization; AN to manuscript editing and critical revision; and ZL to research supervision and final manuscript review. All authors reviewed and approved the final manuscript.

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Conflict of Interest:

The authors declare no competing interests.

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