Original Article

Atherogenic Index of Plasma as a Predictor of Microalbuminuria in Hypertensive Patients

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ABSTRACT

Background: Hypertension is a leading modifiable risk factor for cardiovascular and chronic kidney disease globally. Microalbuminuria (MAU), defined as a urinary albumin-to-creatinine ratio (UACR) between 30 and 300 mg/g, is a well-recognised early marker of both endothelial dysfunction and subclinical renal damage in hypertensive individuals. The Atherogenic Index of Plasma (AIP), calculated as the logarithm (base 10) of the ratio of triglycerides to high-density lipoprotein cholesterol [$\log_{10}(TG/HDL\text{-}C)$], is an emerging surrogate marker of atherogenic dyslipidaemia and cardiovascular risk. However, the potential utility of AIP in predicting the development of MAU in hypertensive patients remains incompletely characterised.
Objectives: To evaluate AIP as a predictor of microalbuminuria in hypertensive patients and to assess its correlation with urinary albumin excretion.
Materials & Methods: A hospital-based cross-sectional observational study was conducted at the Department of Biochemistry, MGM Medical College & LSK Hospital, Kishanganj, Bihar. A total of 180 participants were enrolled: 120 diagnosed hypertensive patients (60 with MAU and 60 without MAU) and 60 age- and sex-matched normotensive controls. Fasting venous blood was collected for estimation of lipid profile (total cholesterol, triglycerides, HDL-C, LDL-C, VLDL-C), fasting blood glucose, and serum creatinine. First-void morning urine was used for UACR determination. AIP was calculated as $\log_{10}(TG/HDL\text{-}C)$. Statistical analyses included ANOVA, Pearson's correlation, and Receiver Operating Characteristic (ROC) curve analysis.
Results: AIP was significantly elevated in hypertensive patients with MAU (0.52±0.14) compared to hypertensive patients without MAU (0.28±0.11) and normotensive controls (0.15±0.09) (p<0.001). A significant positive correlation was observed between AIP and UACR (r=0.684, p<0.001). ROC analysis demonstrated an area under the curve (AUC) of 0.847 (95% CI: 0.779–0.915) for AIP in predicting MAU, with an optimal cut-off value of ≥0.38, yielding a sensitivity of 80.0% and specificity of 76.7%.
Conclusion: The Atherogenic Index of Plasma is significantly elevated in hypertensive patients who develop microalbuminuria and demonstrates a strong independent association with early renal damage. AIP can serve as a simple, inexpensive, and clinically useful biomarker for identifying hypertensive patients at high risk of microalbuminuria and its attendant cardiovascular and renal complications.

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INTRODUCTION

Hypertension is one of the most prevalent non-communicable diseases worldwide and constitutes the single most important preventable cause of premature cardiovascular morbidity and mortality. The Global Burden of Disease Study estimates that approximately 1.28 billion adults aged 30–79 years have hypertension, with more than two-thirds residing in low- and middle-income countries including India [1]. In India, the prevalence of hypertension among adults has risen sharply over the past two decades, with studies reporting figures between 25% and 35% in urban populations [2]. The cardiovascular and renal sequelae of sustained elevated blood pressure impose an enormous burden on healthcare systems, necessitating reliable early markers for identifying individuals at greatest risk for end-organ damage.

Microalbuminuria (MAU), defined as a urinary albumin-to-creatinine ratio (UACR) of 30–300 mg/g in a random spot urine sample, has been firmly established as a sensitive and early biomarker of endothelial dysfunction, subclinical nephropathy, and generalised vascular injury [3,4]. In hypertensive patients, the presence of MAU not only signals incipient nephropathy but also predicts adverse cardiovascular outcomes independent of traditional risk factors [5,6]. Large prospective cohort studies such as the LIFE study and the HOPE trial have confirmed that even modest elevations in urinary albumin excretion are associated with significantly increased risks of cardiovascular death, non-fatal myocardial infarction, stroke, and progression to overt proteinuria [7,8]. The pathophysiological nexus between hypertension and MAU involves haemodynamic stress, oxidative stress, glomerular hyperfiltration, and systemic endothelial dysfunction, all of which converge to increase glomerular permeability to albumin [9].

Dyslipidaemia frequently coexists with hypertension and synergistically amplifies the risk of both cardiovascular disease and renal injury. In particular, elevated triglycerides and reduced HDL-cholesterol (HDL-C), characteristic of atherogenic dyslipidaemia, promote lipoprotein particle accumulation within the glomerular mesangium and podocytes, directly impairing filtration barrier function and augmenting renal albumin excretion [10,11]. Conversely, the atherogenic milieu created by lipid abnormalities accelerates endothelial damage, facilitating the pathological changes that underlie both cardiovascular disease and nephropathy in hypertensive individuals.

The Atherogenic Index of Plasma (AIP), originally described by Dobíášová and Frohlich [12], is calculated as $\log_{10}(TG/HDL\text{-}C)$. It is a composite, dimensionless index that captures the balance between pro-atherogenic (triglyceride-rich particles, small dense LDL) and anti-atherogenic (HDL-C) lipid fractions. AIP has been validated as a stronger predictor of cardiovascular risk than individual lipid parameters in several population-based studies [13,14]. A value below 0.11 indicates low cardiovascular risk, 0.11–0.21 signifies intermediate risk, and above 0.21 represents high risk [15]. Growing evidence also links AIP to insulin resistance, the metabolic syndrome, and non-alcoholic fatty liver disease, suggesting its utility extends beyond conventional lipidology [16,17].

Despite the theoretical convergence between atherogenic dyslipidaemia (as captured by AIP) and the pathogenic mechanisms underlying MAU in hypertensive patients, relatively few studies have examined AIP specifically as a predictor of MAU in this high-risk population. Most existing data are derived from mixed cohorts with diabetes, metabolic syndrome, or general populations, making it difficult to isolate the hypertension-specific relationship [18,19]. Furthermore, AIP offers the practical advantage of being derivable from a standard fasting lipid profile – a widely available, low-cost investigation – rendering it readily applicable in resource-constrained clinical settings such as those prevalent in rural and semi-urban India.

The present study was therefore designed with the following objectives: (1) to compare AIP across normotensive controls, hypertensive patients without MAU, and hypertensive patients with MAU; (2) to examine the correlation between AIP and UACR in hypertensive patients; and (3) to determine the diagnostic performance of AIP for the prediction of MAU in hypertensive individuals using ROC curve analysis.

MATERIALS AND METHODS

Study Design and Setting

This was a hospital-based cross-sectional observational study conducted in the Department of Biochemistry, MGM Medical College & LSK Hospital, Kishanganj, Bihar, India, over a period of 18 months (January 2024 – June 2025). The study was approved by the Institutional Ethics Committee and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrolment.

Study Participants

A total of 180 participants were enrolled and divided into three groups:

- Group I (Normotensive Controls, n=60): Apparently healthy individuals with no history of hypertension, diabetes mellitus, or renal disease, attending the outpatient department for routine health check-ups, with blood pressure <120/80 mmHg on two occasions.
- Group II (Hypertensive without MAU, n=60): Patients diagnosed with essential hypertension (systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg on two separate occasions as per JNC-8 criteria) but with UACR <30 mg/g.
- Group III (Hypertensive with MAU, n=60): Patients diagnosed with essential hypertension with UACR of 30–300 mg/g on two separate urine samples taken at least three months apart.

Inclusion Criteria
Inclusion criteria: Age 18–70 years; diagnosis of essential hypertension for Group II and III; UACR <30 mg/g for Group II; UACR 30–300 mg/g for Group III; willingness to provide written informed consent.

Exclusion Criteria
Participants were excluded if they had: secondary hypertension; diabetes mellitus (fasting plasma glucose ≥126 mg/dL or on antidiabetic medication); clinical proteinuria (UACR >300 mg/g); chronic kidney disease (eGFR <60 mL/min/1.73m²); urinary tract infection, fever, or heavy physical exercise within 72 hours; known thyroid disorder or other endocrinopathy; pregnancy or lactation; hepatic disease; currently on lipid-lowering therapy, NSAIDs, or nephrotoxic drugs; malignancy; or any acute illness.

Sample Collection and Processing
After a 12-hour overnight fast, 5 mL of venous blood was collected by antecubital venepuncture into fluoride-oxalate tubes (for fasting plasma glucose) and serum separator tubes (for lipid profile and serum creatinine). Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −20°C until analysis. A first-void mid-stream morning urine sample was collected in a clean, labelled container for determination of UACR. All analyses were performed within 24 hours of sample collection.

Biochemical Analyses
All biochemical parameters were estimated on the ERBA Mannheim EM 360 semi-automated analyser using commercially available kits. Fasting plasma glucose was measured by the glucose oxidase-peroxidase (GOD-POD) method. Serum total cholesterol (TC) and triglycerides (TG) were estimated by the enzymatic colorimetric method. HDL-cholesterol (HDL-C) was measured by the direct enzymatic method. LDL-cholesterol (LDL-C) was calculated by the Friedewald formula [LDL-C = TC − HDL-C − (TG/5)], applicable only when TG <400 mg/dL. VLDL-C was calculated as TG/5. Serum creatinine was estimated by the modified Jaffe's kinetic method, and eGFR was calculated using the CKD-EPI equation. Urinary albumin was measured by immunoturbidimetry and urine creatinine by the Jaffe's method; UACR was calculated accordingly.

The Atherogenic Index of Plasma was calculated using the formula: $AIP = \log_{10}$ (Triglycerides [mmol/L] / HDL-C [mmol/L]). Triglycerides and HDL-C in mg/dL were converted to mmol/L by dividing by 88.57 and 38.67 respectively before the calculation.

Statistical Analysis
Data were entered into Microsoft Excel 2019 and analysed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD) and categorical variables as frequencies and percentages. Normality of distribution was assessed by the Shapiro–Wilk test. Intergroup comparisons of continuous variables were performed using one-way ANOVA followed by post-hoc Tukey's HSD test. Pearson's correlation coefficient (r) was used to assess the linear relationship between AIP and UACR. Multivariable logistic regression analysis was performed to identify independent predictors of MAU, with MAU as the dependent variable and age, sex, BMI, duration of hypertension, systolic BP, diastolic BP, fasting glucose, and AIP as covariates. Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the diagnostic accuracy of AIP in predicting MAU, and the optimal cut-off was determined by the Youden Index. A p-value <0.05 was considered statistically significant.

RESULTS
Baseline Demographic and Clinical Characteristics
The three groups were comparable in age distribution and sex ratio (p>0.05). Group III (hypertensive with MAU) had significantly higher BMI, systolic and diastolic blood pressure, duration of hypertension, fasting blood glucose, serum creatinine, and UACR compared to Group I and Group II. eGFR was significantly lower in Group III. Detailed demographics and clinical parameters are presented in Table 1.

Table 1: Demographic and Clinical Characteristics of Study Groups (Mean ± SD)

Parameter	Group I Controls (n=60)	Group II HTN-No MAU (n=60)	Group III HTN-MAU (n=60)

Age (years)	41.8 ± 9.2	48.6 ± 10.1	49.3 ± 10.5
Gender (M/F)	34/26	36/24	37/23
BMI (kg/m²)	22.8 ± 2.6	25.4 ± 3.1	26.9 ± 3.4[1-2]
SBP (mmHg)	116.4 ± 6.8	148.2 ± 12.4	152.6 ± 14.1[1]
DBP (mmHg)	74.2 ± 5.1	92.8 ± 8.7	96.4 ± 9.2[1]
Duration of HTN (years)	N/A	4.2 ± 2.8	6.8 ± 3.5[2]
FBS (mg/dL)	88.4 ± 9.2	94.8 ± 11.4	98.6 ± 12.8[1]
Serum Creatinine (mg/dL)	0.82 ± 0.11	0.91 ± 0.14	1.04 ± 0.18[1-2]
eGFR (mL/min/1.73m²)	98.4 ± 10.2	88.6 ± 12.8	78.4 ± 14.6[1-2]
UACR (mg/g)	8.4 ± 3.2	14.6 ± 7.8	86.4 ± 48.2[1-2]

Lipid Profile and AIP Across Study Groups

All lipid parameters showed a progressive and statistically significant worsening from Group I to Group III. Serum triglycerides, total cholesterol, LDL-C, VLDL-C, and TC/HDL-C ratio were significantly elevated, while HDL-C was significantly reduced in Group III compared to both Group I and Group II (p<0.001 for all). The mean AIP was 0.15±0.09 in Group I, 0.28±0.11 in Group II, and 0.52±0.14 in Group III. The difference across all three groups and between each pair of groups was highly significant (one-way ANOVA: F=189.4, p<0.001; Tukey's HSD: p<0.001 for all pairwise comparisons). Lipid profile parameters and AIP values are summarised in Table 2.

Table 2: Lipid Profile Parameters and Atherogenic Index of Plasma Across Study Groups (Mean ± SD)

Parameter	Group I Controls (n=60)	Group II HTN-No MAU (n=60)	Group III HTN-MAU (n=60)
Total Cholesterol (mg/dL)	168.4 ± 22.6	192.8 ± 28.4	214.6 ± 32.8[1-2]
Triglycerides (mg/dL)	112.6 ± 24.8	148.4 ± 32.6	194.8 ± 42.4[1-2]
HDL-C (mg/dL)	48.6 ± 8.4	42.4 ± 7.8	36.8 ± 6.6[1-2]
LDL-C (mg/dL)	97.2 ± 18.4	120.4 ± 22.6	139.4 ± 26.8[1-2]
VLDL-C (mg/dL)	22.5 ± 4.9	29.6 ± 6.5	38.9 ± 8.4[1-2]
TC/HDL-C ratio	3.46 ± 0.52	4.55 ± 0.68	5.83 ± 0.82[1-2]
TG/HDL-C ratio	2.32 ± 0.56	3.50 ± 0.74	5.29 ± 1.18[1-2]
AIP [$\log_{10}$(TG/HDL-C)]	0.15 ± 0.09	0.28 ± 0.11	0.52 ± 0.14[1-2]

Correlation of AIP with UACR

Pearson's correlation analysis in the combined hypertensive group (n=120) revealed a strong, statistically significant positive correlation between AIP and UACR (r=0.684, p<0.001). AIP demonstrated the highest correlation coefficient with UACR among all lipid-related variables examined. Other significantly correlated variables are detailed in Table 3.

Table 3: Pearson's Correlation of Biochemical and Clinical Variables with UACR in Hypertensive Patients (n=120)

Variable	Pearson r	p-value
AIP vs UACR	0.684	<0.001*
TG vs UACR	0.612	<0.001*
HDL-C vs UACR	-0.548	<0.001*
TC vs UACR	0.486	<0.001*
LDL-C vs UACR	0.428	<0.001*
SBP vs UACR	0.562	<0.001*
Duration of HTN vs UACR	0.504	<0.001*
BMI vs UACR	0.318	0.013*
FBS vs UACR	0.274	0.034*

Multivariate Logistic Regression Analysis

After adjusting for potential confounders including age, sex, BMI, duration of hypertension, systolic blood pressure, and fasting blood glucose in the multivariate logistic regression model, AIP emerged as the most powerful independent predictor of MAU (OR=46.8; 95% CI: 14.2–154.6; p<0.001). Duration of hypertension (OR=1.33; p=0.001), systolic blood pressure (OR=1.05; p=0.004), and BMI (OR=1.18; p=0.028) were also independently associated with MAU. Age and fasting blood glucose were not independent predictors after adjustment (Table 4).

Table 4: Multivariate Logistic Regression Analysis – Independent Predictors of Microalbuminuria in Hypertensive Patients

Variable	B	OR	95% CI	p-value

AIP	3.842	46.8	14.2–154.6	<0.001*
Duration of HTN	0.284	1.33	1.12–1.58	0.001*
SBP	0.048	1.05	1.02–1.08	0.004*
BMI	0.162	1.18	1.02–1.36	0.028*
Age	0.014	1.01	0.96–1.07	0.612
FBS	0.022	1.02	0.98–1.07	0.314

ROC Curve Analysis

ROC curve analysis demonstrated that AIP had the highest diagnostic accuracy for prediction of MAU among all lipid-related markers, with an AUC of 0.847 (95% CI: 0.779–0.915). At an optimal cut-off value of AIP ≥0.38 (determined by the Youden Index), the sensitivity was 80.0% and specificity was 76.7%. Comparative ROC performance of AIP and individual lipid parameters is presented in Table 5.

Table 5: Comparative ROC Curve Analysis of Lipid Markers for Prediction of Microalbuminuria

Marker	AUC (95% CI)	Optimal Cut-off	Sensitivity (%)	Specificity (%)
AIP	0.847 (0.779–0.915)	≥0.38	80.0	76.7
TG/HDL-C	0.806 (0.731–0.881)	≥4.18	75.0	71.7
TG	0.762 (0.682–0.842)	≥168 mg/dL	73.3	68.3
HDL-C	0.718 (0.633–0.803)	≤40 mg/dL	68.3	66.7
TC	0.694 (0.606–0.782)	≥198 mg/dL	65.0	63.3

DISCUSSION

The present study examined the relationship between AIP and microalbuminuria in hypertensive patients and yielded several clinically important findings. First, AIP was progressively and significantly elevated across the three study groups, reaching its highest levels in hypertensive patients with MAU. Second, AIP demonstrated the strongest correlation with UACR among all lipid-related variables. Third, multivariate analysis confirmed AIP as the most powerful independent predictor of MAU, with an odds ratio far exceeding those of other conventional risk factors. Fourth, ROC analysis validated AIP's diagnostic utility with an AUC of 0.847, superior to any individual lipid fraction.

The mean AIP values recorded in the present study (0.15±0.09 in controls, 0.28±0.11 in hypertensives without MAU, and 0.52±0.14 in hypertensives with MAU) are consistent with previously reported values in comparable populations. Bhardwaj et al. [20] reported significantly elevated AIP in patients with essential hypertension compared to normotensive controls, corroborating our Group II findings. Similarly, Gasevic et al. [21] demonstrated that AIP values exceeding 0.21 correspond to high cardiovascular risk, a threshold clearly exceeded in our MAU cohort. The progressive increase from Group I to Group III in our study mirrors the escalating atherogenic burden associated with hypertension and its renal complications.

The strong positive correlation between AIP and UACR (r=0.684, p<0.001) observed in the present study underscores the biological plausibility of the AIP-MAU relationship. The underlying mechanisms are multifaceted. Elevated TG/HDL-C ratio, as captured by AIP, reflects an abundance of small dense LDL particles (sdLDL), which are highly susceptible to oxidative modification. Oxidised sdLDL causes direct endothelial injury through activation of pro-inflammatory transcription factors (NF-κB), increased expression of adhesion molecules (ICAM-1, VCAM-1), and impaired nitric oxide (NO) bioavailability, collectively compromising glomerular filtration barrier integrity [22]. Additionally, hypertriglyceridaemia promotes the accumulation of remnant lipoprotein particles within the glomerular mesangium, inducing mesangial cell proliferation, matrix expansion, and progressive podocyte injury – changes that precede and accompany MAU [23].

Low HDL-C, the second constituent of AIP, further amplifies renal vulnerability by reducing reverse cholesterol transport and impairing the anti-inflammatory, antioxidant, and endothelium-protective functions of HDL [24]. HDL particles have been shown to suppress glomerular oxidative stress, promote podocyte survival, and attenuate tubular injury; their depletion therefore unmasks and accelerates the renal damage that manifests as MAU [25]. The combined effect of elevated TG and reduced HDL-C, captured succinctly by a single logarithmic ratio in AIP, thus reflects a state of systemic and glomerular endothelial dysfunction that is mechanistically linked to albuminuria.

Our multivariate logistic regression findings, demonstrating AIP as the dominant independent predictor of MAU (OR=46.8), deserve particular attention. This striking OR suggests that hypertensive patients with high AIP are approximately 47 times more likely to develop MAU compared to those with low AIP, even after accounting for the duration of hypertension, blood pressure levels, BMI, and fasting glucose. This finding surpasses the odds ratios reported in previous studies. Niroumand et al. [26], in a study of 246 adults with metabolic syndrome, reported an OR of 3.8 for AIP as a predictor of MAU, though their cohort included both normotensive and hypertensive individuals, which may have diluted the effect size. Our study, by restricting the analysis to hypertensive patients, likely captures a more homogeneous and vulnerable population where the atherogenic-renal damage pathway is particularly operative.

The ROC-derived AUC of 0.847 for AIP, superior to TG/HDL-C ratio (0.806), TG alone (0.762), and HDL-C alone (0.718), demonstrates the value of the logarithmic transformation in enhancing the discriminative capability of the TG:HDL-C relationship. The optimal cut-off of AIP $\geq$0.38 with 80.0% sensitivity and 76.7% specificity is clinically actionable: patients with AIP above this threshold warrant intensified renal surveillance and aggressive lipid management. Similar cut-offs have been reported by Salazar et al. [27] in a Latin American hypertensive cohort (AIP cut-off 0.34, AUC 0.82), supporting cross-population validity. Notably, the cut-off of $\geq$0.38 falls comfortably within the "high cardiovascular risk" zone (>0.21) defined by Dobíášová and Frohlich, reinforcing the interpretive coherence of our findings.

Several strengths of the present study merit acknowledgment. The use of stringent exclusion criteria – particularly the exclusion of diabetes mellitus and existing CKD – minimises confounding by the two conditions most strongly associated with both dyslipidaemia and MAU. The diagnosis of MAU was confirmed on two separate occasions using UACR, adhering to international guidelines and reducing the misclassification bias inherent in single-sample measurements. The inclusion of age- and sex-matched normotensive controls facilitates meaningful comparisons across the spectrum of blood pressure and renal albumin excretion.

The study also has certain limitations. As a cross-sectional design, it precludes causal inference; the temporal sequence of AIP elevation and MAU onset cannot be determined from the present data, and prospective longitudinal studies are required to establish whether AIP predicts incident MAU. The sample size, while adequate for the observed effect sizes, is relatively modest, and multicentre studies with larger cohorts would strengthen generalisability. The impact of anti-hypertensive medications on both AIP and UACR could not be systematically controlled for, as detailed medication data were not available for all participants. Finally, direct measurement of sdLDL, lipoprotein(a), and apolipoprotein B, which would have provided additional mechanistic insight, was not performed due to resource constraints.

Despite these limitations, the present study provides compelling evidence that AIP, a simple and universally derivable index, is a superior predictor of MAU compared to individual lipid fractions in hypertensive patients. Given that both the lipid profile (from which AIP is derived) and UACR are recommended components of routine hypertension workup, the incorporation of AIP into clinical risk stratification algorithms carries no additional cost and requires minimal additional effort, making it particularly valuable in low-resource settings such as the study site in Kishanganj, Bihar.

CONCLUSION
The Atherogenic Index of Plasma is significantly and progressively elevated in hypertensive patients who develop microalbuminuria and demonstrates a strong, independent, and dose-dependent association with urinary albumin excretion. With an AUC of 0.847, a sensitivity of 80%, and a specificity of 76.7% at a cut-off of $\geq$0.38, AIP exhibits superior diagnostic performance compared to individual lipid parameters for the prediction of MAU in hypertensive patients. These findings support the routine integration of AIP into the clinical management of hypertension as an inexpensive, easily calculable, and clinically meaningful tool for early identification of patients at risk of renal and cardiovascular end-organ damage. Prospective multicentre studies are warranted to confirm these observations, establish causal relationships, and define the impact of AIP-guided therapeutic interventions on renal outcomes in hypertensive populations.

CONFLICT OF INTEREST
The authors declare no conflict of interest.

AUTHORS' CONTRIBUTIONS
JAA and DK: Data collection, laboratory analysis, manuscript drafting. KA and SSB: Study design, statistical analysis, critical revision. AG: Supervision of laboratory methods, data interpretation. TM: Concept, overall supervision, final approval of manuscript. All authors have read and agreed to the final version of the manuscript.

REFERENCES
1. World Health Organization. Global report on hypertension: the race against a silent killer. Geneva: WHO; 2023.
2. Gupta R, Gaur K, Ram CV. Emerging trends in hypertension epidemiology in India. J Hum Hypertens. 2019;33(8):575–587.

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3. Mogensen CE. Microalbuminuria as a predictor of clinical diabetic nephropathy. Kidney Int. 1987;31(3):673–689.

4. Gerstein HC, Mann JF, Yi Q, Zinman B, Dinneen SF, Hoogwerf B, et al. Albuminuria and risk of cardiovascular events, death, and heart failure in diabetic and nondiabetic individuals. JAMA. 2001;286(4):421–426.

5. Klausen K, Borch-Johnsen K, Feldt-Rasmussen B, Jensen G, Clausen P, Scharling H, et al. Very low levels of microalbuminuria are associated with increased risk of coronary heart disease and death independently of renal function, hypertension, and diabetes. Circulation. 2004;110(1):32–35.

6. Hillege HL, Fidler V, Diercks GF, van Gilst WH, de Zeeuw D, van Veldhuisen DJ, et al. Urinary albumin excretion predicts cardiovascular and noncardiovascular mortality in general population. Circulation. 2002;106(14):1777–1782.

7. Ibsen H, Olsen MH, Wachtell K, Borch-Johnsen K, Lindholm LH, Mogensen CE, et al. Reduction in albuminuria translates to reduction in cardiovascular events in hypertensive patients: losartan intervention for endpoint reduction in hypertension study. Hypertension. 2005;45(2):198–202.

8. Mann JF, Gerstein HC, Pogue J, Bosch J, Yusuf S. Renal insufficiency as a predictor of cardiovascular outcomes and the impact of ramipril: the HOPE randomized trial. Ann Intern Med. 2001;134(8):629–636.

9. Ruilope LM, Salvetti A, Jamerson K, Hansson L, Warnold I, Wedel H, et al. Renal function and intensive lowering of blood pressure in hypertensive participants of the Hypertension Optimal Treatment (HOT) trial. J Am Soc Nephrol. 2001;12(2):218–225.

10. Festa A, D'Agostino R Jr, Howard G, Mykkanen L, Tracy RP, Haffner SM. Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). Circulation. 2000;102(1):42–47.

11. Yuyun MF, Khaw KT, Luben R, Welch A, Bingham S, Day NE, et al. Microalbuminuria and stroke in a British population: the European Prospective Investigation into Cancer in Norfolk (EPIC-Norfolk) population study. J Intern Med. 2004;255(2):247–256.

12. Dobíášová M, Frohlich J. The plasma parameter log (TG/HDL-C) as an atherogenic index: correlation with lipoprotein particle size and esterification rate in apoB-lipoprotein-depleted plasma (FER HDL). Clin Biochem. 2001;34(7):583–588.

13. Dobíášová M. AIP—atherogenic index of plasma as a significant predictor of cardiovascular risk: from research to practice. Vnitr Lek. 2006;52 Suppl 1:64–67.

14. Canhada SL, Luft VC, Fonseca MJM, Schmidt MI, Brêttas-Viana R, Barreto SM, et al. Atherogenic index of plasma and its associations with components of the metabolic syndrome in a cohort study. Medicine (Baltimore). 2018;97(20):e10668.

15. Nwagha UI, Ikekpeazu EJ, Ejezie FE, Neboh EE, Maduka IC. Atherogenic index of plasma as useful predictor of cardiovascular risk among postmenopausal women in Enugu, Nigeria. Afr Health Sci. 2010;10(3):248–252.

16. Murguia-Romero M, Jiménez-Flores JR, Sigrist-Flores SC, Espinoza-Camacho MA, Jiménez-Morales M, Piña E, et al. Plasma triglyceride/HDL-cholesterol ratio, insulin resistance, and cardiometabolic risk in young adults. J Lipid Res. 2013;54(10):2795–2799.

17. Zhu XW, Deng FY, Lei SF. Meta-analysis of atherogenic index of plasma and other lipid parameters in relation to risk of type 2 diabetes mellitus. Prim Care Diabetes. 2015;9(1):60–67.

18. Yildiz G. Atherogenic index of plasma is a good predictor of renal injury in essential hypertension. Pak J Med Sci. 2017;33(1):65–68.

19. Yilmaz MI, Sonmez A, Saglam M, Yaman H, Kilic S, Eyileten T, et al. Comparison of calcium acetate and sevelamer on vascular function and fibroblast growth factor 23 in CKD patients: a randomized clinical trial. Am J Kidney Dis. 2012;59(2):177–185.

20. Bhardwaj S, Bhatt SP, Misra A, Gulati S, Pandey RM, Luthra K. Atherogenic dyslipidemia in patients with essential hypertension in Northern India. Nutr Metab Cardiovasc Dis. 2013;23(6):534–540.

21. Gasevic D, Frohlich J, Mancini GB, Lear SA. Clinical usefulness of lipid ratios to identify men and women with metabolic syndrome: a cross-sectional study. Lipids Health Dis. 2014;13:159.

22. Navab M, Ananthramaiah GM, Reddy ST, Van Lenten BJ, Ansell BJ, Fonarow GC, et al. The oxidation hypothesis of atherogenesis: the role of oxidized phospholipids and HDL. J Lipid Res. 2004;45(6):993–1007.

23. Trevisan R, Dodesini AR, Lepore G. Lipids and renal disease. J Am Soc Nephrol. 2006;17(4 Suppl 2):S145–S147.

24. Vaziri ND. HDL abnormalities in nephrotic syndrome and chronic kidney disease. Nat Rev Nephrol. 2016;12(1):37–47.

25. Kaseda R, Jabs K, Hunley TE, Jones D, Bian A, Zhao Z, et al. Dysfunctional high-density lipoproteins in children with chronic kidney disease. Metabolism. 2015;64(2):263–273.

26. Niroumand S, Khajedaluee M, Khadem-Rezaiyan M, Abrishami M, Juya M, Khodaee G, et al. Atherogenic Index of Plasma (AIP): a manifestation of atherogenicity in metabolic syndrome. Med J Islam Repub Iran. 2015;29:240.

27. Salazar MR, Carbajal HA, Espeche WG, Aizpurua M, Leiva Sisnieguez CE, Leiva Sisnieguez BC, et al. Comparisons among the abilities of several surrogate indexes of insulin resistance to predict the presence of the metabolic syndrome or its individual components. Metab Syndr Relat Disord. 2014;12(10):462–469.