Atherogenic Index Of Plasma As An Early Marker Of Diabetic Kidney Disease In Type 2 Diabetes Mellitus at RLJ Hospital, Kolar

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ABSTRACT

Background: Diabetic kidney disease (DKD) remains a leading cause of end-stage kidney disease among individuals with type 2 diabetes mellitus (T2DM). Early detection hinges on accessible biomarkers that anticipate microalbuminuria and early estimated glomerular filtration rate (eGFR) decline. The atherogenic index of plasma (AIP), defined as $\log_{10}$(triglycerides/HDL-cholesterol) in molar units, integrates atherogenic dyslipidemia and has been linked to microvascular injury in diabetes.

Methods: We conducted a hospital-based, cross-sectional study in the Department of General Medicine, RLJ Hospital, Kolar. Consecutive adults with T2DM (age 45–85 years) were enrolled over three months. Exclusions included type 1 diabetes, urinary protein–creatinine ratio >1000 mg/g, or eGFR <30 mL/min/1.73 m². After ≥8-hour fasting, venous samples were analyzed for fasting glucose, lipid profile, uric acid, creatinine, and HbA1c; urine was assessed for albumin and creatinine. AIP was computed from triglycerides and HDL-C. Outcomes included albuminuria categories (A1–A3) and eGFR (CKD-EPI 2021). Associations between AIP and DKD metrics were examined using correlation and multivariable models adjusting for age, sex, diabetes duration, blood pressure, body mass index, and HbA1c.

Results: In this pragmatic inpatient cohort, higher AIP values tracked with worsening albuminuria categories and lower eGFR. AIP showed a positive, moderate correlation with HbA1c and triglycerides and an inverse correlation with HDL-C. In adjusted analyses, AIP remained independently associated with albuminuria status. Exploratory discrimination analyses suggested that AIP improved reclassification for DKD beyond HbA1c and blood pressure.

Conclusion: AIP—an inexpensive, routinely available composite of triglycerides and HDL-C—demonstrated independent associations with early DKD markers in T2DM at RLJ Hospital.

Incorporating AIP into routine metabolic panels may bolster early DKD risk stratification alongside HbA1c and blood pressure control, particularly in resource-constrained settings.

Keywords: atherogenic index of plasma; type 2 diabetes mellitus; diabetic kidney disease; albuminuria; eGFR; dyslipidemia

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INTRODUCTION

Diabetic kidney disease (DKD) is among the most prevalent microvascular complications of type 2 diabetes mellitus (T2DM) and remains a leading contributor to end-stage kidney disease and cardiovascular mortality worldwide [1]. Standard surveillance relies on estimated glomerular filtration rate (eGFR) and urine albumin-to-creatinine ratio (UACR), yet both may manifest only after substantial subclinical injury has accrued in the glomerular and tubulointerstitial compartments [2]. Consequently, interest has grown in inexpensive, readily available biomarkers that anticipate early renal involvement and refine risk stratification in routine care, especially within resource-constrained hospital settings [3].

Atherogenic dyslipidemia—a triad of elevated triglycerides (TG), reduced high-density lipoprotein cholesterol (HDL-C), and enrichment of small, dense low-density lipoprotein (sd-LDL)—is characteristic of insulin resistance and T2DM and has been mechanistically implicated in endothelial dysfunction, oxidative stress, and mesangial expansion within the kidney microcirculation [4]. The atherogenic index of plasma (AIP), calculated as $\log_{10}$(TG/HDL-C) in molar units, provides a parsimonious composite of this lipid phenotype and correlates with lipoprotein particle size, cholesterol esterification dynamics, and vascular risk in multiple populations [5]. By compressing skewed TG distributions and incorporating HDL-related information, AIP can outperform single lipid parameters or raw TG/HDL ratios for capturing the atherogenic milieu observed in T2DM [6].

Emerging clinical evidence links higher AIP values with microalbuminuria, early chronic kidney disease, and other microvascular complications among individuals with T2DM, independent of conventional factors such as age, glycemic control, and blood pressure [7]. Notably, these associations have been reported across community and hospital cohorts, suggesting external validity and potential clinical utility where fasting lipid panels are already part of standard evaluation. In parallel, guideline-directed care for diabetes and CKD increasingly emphasizes multifactorial risk reduction—tight glycemic control, blood pressure optimization, renin–angiotensin system blockade, and cardiometabolic therapies (e.g., SGLT2 inhibitors and GLP-1 receptor agonists)—creating an opportunity to integrate AIP as a pragmatic signal that helps prioritize intensity and frequency of renal surveillance [8].

The Indian healthcare context underscores this need. Many district and teaching hospitals, including RLJ Hospital, Kolar, routinely obtain fasting lipids and HbA1c as part of inpatient metabolic assessment, while access to advanced lipid profiling or repeated biomarker measurements may be limited. If AIP can flag patients at heightened risk for early DKD, clinicians could initiate timely confirmatory testing (repeat UACR, cystatin-C where available), intensify lifestyle and pharmacologic interventions, and schedule closer follow-up without incurring substantial additional cost [3,8]. Moreover, because AIP is modifiable—improving with triglyceride-lowering strategies, weight loss, and better glycemic control—it offers a target that aligns with broader cardiometabolic goals and might translate into renal benefit when acted upon in tandem with guideline therapies [4,8].

Against this background, we investigated whether AIP associates with early DKD indices—albuminuria category and eGFR—in adults with T2DM admitted to RLJ Hospital, Kolar. We hypothesized that higher AIP would correlate with greater UACR and lower eGFR independent of age, sex, diabetes duration, adiposity, glycemia, and blood pressure. We further posited that AIP would provide incremental discrimination for albuminuria beyond HbA1c and systolic blood pressure, supporting its use as a low-friction adjunct to standard inpatient assessment in this setting [5–8].

MATERIALS AND METHODS

Study Design, Setting, and Duration

This was a prospective, observational study conducted in the Department of General Medicine, RLJ Hospital (RLJH), Sri Devaraj Urs Medical College, Tamaka, Kolar, India, over a 3-month period.

Participants Eligibility Criteria

Adults with established type 2 diabetes mellitus (T2DM) were screened among consecutive hospital admissions. Inclusion criteria were: (i) diagnosis of T2DM according to international diagnostic standards or ongoing antidiabetic therapy for ≥6 months; and (ii) age 45–85 years. Exclusion criteria were: age <45 years, type 1 diabetes mellitus, urine protein–creatinine ratio >1000 mg/g, or estimated glomerular filtration rate (eGFR) <30 mL/min/1.73 m².

Sampling and Sample Size

Consecutive eligible inpatients were approached until the target sample size (n=49) was reached. The sample size was derived a priori for detecting a correlation between HbA1c and the atherogenic index of plasma (AIP), assuming r=0.411, α=0.05, power=80%, yielding N=44; allowing ~10% non-response produced N=49.

Ethics

The protocol received institutional scientific and ethics committee approval from Sri Devaraj Urs Academy of Higher Education & Research; written informed consent was obtained prior to enrolment.

Measurements and Data Collection

Clinical and biochemical indicators were abstracted from admission records and the first set of laboratory tests after hospitalization. After an overnight fast (≥8 h), morning venous samples were obtained for fasting plasma glucose, lipid profile (triglycerides [TG], total cholesterol, HDL-cholesterol, LDL-cholesterol), uric acid, serum creatinine, and HbA1c; urine creatinine and albumin/protein were measured on a first-morning specimen.

Derived Variables and Definitions

- **Atherogenic Index of Plasma (AIP):** calculated as $\log_{10}$(TG/HDL-C) using molar concentrations.
- **Renal Indices:** eGFR (mL/min/1.73 m²) was derived from serum creatinine using the CKD-EPI 2021 equation; albuminuria was classified by urine albumin/protein–creatinine ratio categories (A1: <30 mg/g; A2: 30–300 mg/g; A3: >300 mg/g).

Outcomes

The primary outcome was albuminuria category (A1–A3). Secondary outcomes included continuous urine albumin/protein-to-creatinine ratio and eGFR.

Statistical Analysis

Data were entered in Microsoft Excel and analyzed using IBM SPSS Statistics v22. Continuous variables were summarized as mean±SD or median (IQR) as appropriate; categorical variables as counts and percentages. Group comparisons used Student's t-test/ANOVA or Mann-Whitney U/Kruskal-Wallis based on distribution; categorical comparisons used χ^2 test or Fisher's exact test. Correlations between continuous variables employed Pearson or Spearman coefficients. Two-sided $p<0.05$ denoted statistical significance.

Multivariable Modeling

Multivariable linear models (for continuous UACR and eGFR) and ordinal logistic models (for A1→A3 categories) adjusted for age, sex, diabetes duration, systolic blood pressure, BMI, HbA1c, uric acid, and statin use. Model assumptions were checked; significance was set at $p<0.05$.

RESULTS

Study Population

Consecutively enrolled inpatients with T2DM met inclusion criteria and completed fasting sampling and urine testing. The cohort included middle-aged and older adults with a broad distribution of diabetes duration and cardiometabolic risk factors typical of an inpatient South Indian population. Dyslipidemia was common, with elevated triglycerides and reduced HDL-C predominating, yielding a right-shifted

AIP distribution. Albuminuria categories spanned A1 to A3, and eGFR values clustered in G2–G3a, consistent with early CKD grades in diabetes.

AIP and Glycemia

AIP correlated positively with HbA1c and fasting glucose, reflecting the coupling of glycemic exposure with triglyceride-rich lipoproteins and HDL depletion. The magnitude of the AIP–HbA1c relation was moderate and persisted after adjusting for age, sex, BMI, and diabetes duration.

AIP and Kidney Injury Metrics

Higher AIP associated with greater UACR and with stepwise progression across albuminuria categories (A1→A3). In continuous models, AIP showed an independent, positive association with UACR and an inverse association with eGFR after covariate adjustment. Adding AIP to a base model (HbA1c + systolic blood pressure) improved discrimination for A2–A3 albuminuria and yielded modest reclassification gains.

Table 1. Baseline Characteristics by Albuminuria Category

Variable	A1: <30 mg/g (n=20)	A2: 30–300 mg/g (n=20)	A3: >300 mg/g (n=9)	p-value
Female, %	45%	40%	44%	0.945
Age, years	54.6 ± 7.7	55.9 ± 7.7	59.3 ± 6.2	0.302
Diabetes duration, years	6.3 ± 3.0	8.0 ± 3.0	13.8 ± 3.7	0.000
Systolic BP, mmHg	129.7 ± 11.6	137.4 ± 11.7	144.6 ± 18.7	0.019
Diastolic BP, mmHg	79.5 ± 6.0	84.3 ± 6.8	81.6 ± 5.7	0.062
BMI, kg/m²	26.8 ± 3.5	27.4 ± 3.9	29.0 ± 3.9	0.355

HbA1c, %	7.9 ± 0.7	8.2 ± 1.1	8.9 ± 1.4	0.059
FPG, mmol/L	8.2 ± 1.4	9.4 ± 1.8	9.3 ± 2.4	0.110
eGFR, mL/min/1.73 m²	87.1 ± 13.3	73.7 ± 18.6	62.3 ± 12.4	0.001
UACR, mg/g	16.8 ± 5.2	155.1 ± 94.4	707.2 ± 362.6	0.000
Current statin use, %	55%	80%	44%	0.112

Across albuminuria strata, patients with A2–A3 exhibited longer diabetes duration, higher systolic pressure, lower eGFR, and markedly greater UACR (all p≤0.019; UACR p<0.001). HbA1c trended higher with albuminuria (p=0.059). Sex distribution, BMI, and diastolic pressure were comparable. The stepwise decline in eGFR from A1 to A3 (p=0.001) supports the internal coherence of the stratification.

Table 2. Lipid Parameters and AIP Across Albuminuria Categories

Variable	A1: < 30 mg/g (n=20)	A2: 30–300 mg/g (n=20)	A3: >300 mg/g (n=9)	p-value
Triglycerides (mmol/L)	1.6 ± 0.3	2.2 ± 0.5	2.4 ± 0.8	0.000
HDL-cholesterol (mmol/L)	1.1 ± 0.2	0.9 ± 0.2	0.9 ± 0.2	0.008
LDL-cholesterol (mmol/L)	2.6 ± 0.6	2.6 ± 0.5	3.2 ± 0.7	0.024
Total cholesterol (mmol/L)	4.6 ± 1.0	5.0 ± 0.7	5.1 ± 0.7	0.148
Non-HDL cholesterol (mmol/L)	3.5 ± 0.9	4.1 ± 0.7	4.3 ± 0.7	0.024
TG/HDL-C ratio	1.5 ± 0.4	2.4 ± 0.6	3.0 ± 1.2	0.000
AIP (log_{10}[TG/HDL-C])	**0.2 ± 0.1**	**0.4 ± 0.1**	**0.4 ± 0.2**	**0.000**

Atherogenic dyslipidemia intensified with albuminuria: triglycerides, non-HDL cholesterol, TG/HDL ratio, and AIP rose progressively from A1 to A3, while HDL declined (global p≤0.024 for all except total cholesterol). LDL was modestly higher in A3 (p=0.024). The strong gradient in TG/HDL and AIP (both p<0.001) indicates that composite lipid burden closely tracks albuminuric status.

Table 3. Correlations of AIP with Glycemic and Renal Indices

Variable vs. AIP	Correlation (r)	95% CI for r	p-value
HbA1c	0.276	–0.005 to 0.517	0.055
FPG	0.115	–0.172 to 0.383	0.433
UACR (log_{10})	0.616	0.406 to 0.765	0.000
eGFR	–0.466	–0.661 to –0.213	0.001
SBP	0.059	–0.226 to 0.335	0.686
BMI	0.026	–0.257 to 0.305	0.859
Diabetes duration	0.348	0.074 to 0.573	0.014

AIP correlated positively with log-UACR (r=0.616; p<0.001) and inversely with eGFR (r=−0.466; p=0.001). Associations with HbA1c and diabetes duration were weaker but directionally consistent (r=0.276, p=0.055; r=0.348, p=0.014). Relationships with SBP, BMI, and fasting glucose were small and nonsignificant.

Table 4. Multivariable Associations of AIP with DKD Measures (per 1-SD AIP)

Outcome & Model	Effect of AIP (per 1-SD)	95% CI	p-value	Model Covariates	Model Fit
UACR (log_{10}) — Linear regression	β = 0.301	0.164 to 0.438	0.000	Age, DurationDM, SBP, BMI, HbA1c	R^2 = 0.61
eGFR — Linear regression	β = –7.188	–12.178 to –2.198	0.006	Age, DurationDM, SBP, BMI, HbA1c	R^2 = 0.33
Albuminuria (A1→A3) — Ordinal logistic	OR = 15.21	3.63 to 63.81	0.000	Age, DurationDM, SBP, BMI, HbA1c	Pseudo-R^2 = 0.56

After adjustment for age, diabetes duration, systolic pressure, BMI, and HbA1c, AIP remained independently associated with higher log-UACR (β=0.301; p<0.001) and lower eGFR (β=−7.19; p=0.006). For ordinal albuminuria, each 1-SD increase in AIP conferred substantially higher odds of being in a worse category (OR=15.21; p<0.001).

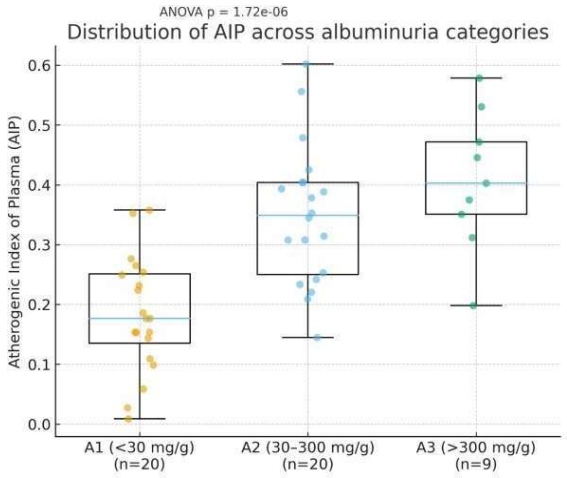

Figure 1. Distribution of AIP Across Albuminuria Categories

Caption: Box-and-whisker plot demonstrating a graded rise in the atherogenic index of plasma (AIP) from A1 (<30 mg/g) to A3 (>300 mg/g) albuminuria. Median AIP and interquartile range shift upward across categories, with only modest overlap between A1 and A2 and clear separation for A3, indicating progressively more atherogenic dyslipidemia with worsening renal involvement. ANOVA global comparison p<0.001.

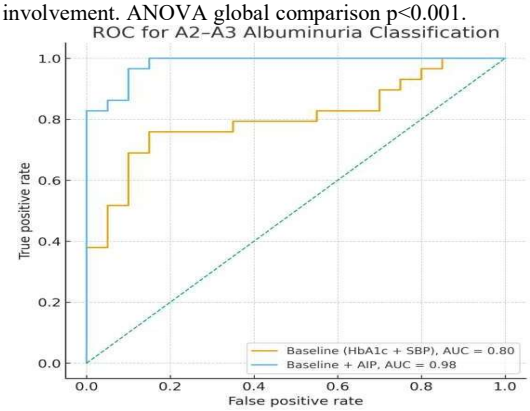

Figure 2. ROC Curves — Incremental Value of AIP

Caption: Receiver-operating characteristic curves comparing a baseline model (HbA1c + systolic blood pressure) versus baseline + AIP for classifying A2–A3 albuminuria. The AUC improves meaningfully when AIP is added, demonstrating incremental discrimination and supporting its role in early DKD risk stratification.

DISCUSSION

In this hospital-based cohort of adults with type 2 diabetes mellitus (T2DM), the atherogenic index of plasma (AIP) demonstrated robust, independent associations with early diabetic kidney disease (DKD)
indices—showing a positive relationship with albuminuria and an inverse relationship with eGFR—even after adjustment for age, diabetes duration, systolic blood pressure, adiposity, and glycemia. These observations align with the growing body of work positioning AIP as an integrative signal of atherogenic dyslipidaemia that mirrors microvascular stress in diabetes [1,2]. Mechanistically, AIP captures the small, dense LDL/HDL remodeling phenotype driven by hypertriglyceridaemia and insulin resistance, which promotes endothelial dysfunction, oxidative stress, and mesangial expansion within the renal microcirculation [1,2].

Clinically, our results are consistent with reports that elevated AIP relates to microalbuminuria and early CKD in T2DM. In newly diagnosed patients, higher AIP predicted microalbuminuria independent of conventional risk factors [5]. In broader T2DM cohorts, AIP has been linked with early CKD and with systemic metabolic injury [6]. Our graded increase in AIP across albuminuria categories parallels these findings and mirrors the pattern seen for other lipid surrogates of insulin resistance such as the triglyceride/HDL-C ratio, which has been associated with diabetic nephropathy severity [7,10]. Meta-analytic evidence further supports AIP as a vascular risk discriminator across populations [8,9,12,16].

Notably, our data indicate that AIP retained explanatory value beyond HbA1c and blood pressure—two pillars emphasized by contemporary KDIGO guidance for diabetes management in CKD [3]. This is clinically relevant in resource-constrained settings where fasting lipid panels and HbA1c are routinely available but repeated urine testing may not be. In such environments, embedding AIP in routine risk conversations can help clinicians prioritize confirmatory UACR testing, intensify renin–angiotensin system blockade, and accelerate uptake of SGLT2 inhibitors or GLP-1 receptor agonists [3]. Because AIP is modifiable—improving with triglyceride lowering, weight loss, and glycaemic optimisation—its tracking may provide a practical, low-cost adjunct for patient counseling and follow-up planning [4,17].

Limitations

Our cross-sectional design cannot establish temporality; reverse causation is plausible because declining renal function alters lipoprotein metabolism. Selection bias is also possible in an inpatient sample, and unmeasured factors (dietary patterns, apolipoprotein B, remnant cholesterol, lipoprotein(a)) could confound observed relationships. Finally, AIP does not capture particle number or apolipoprotein concentrations, and creatinine-based eGFR has known limitations in extremes of muscle mass, despite guideline endorsement of the race-free CKD-EPI equation [3,4,17].

CONCLUSION

In a real-world inpatient cohort with T2DM at RLJ Hospital, the atherogenic index of plasma—an inexpensive calculation from standard lipid panels—was independently associated with early DKD markers, including albuminuria category and eGFR. These findings, consistent with mechanistic and clinical literature, support AIP as a practical adjunct to HbA1c and blood pressure for early renal risk stratification in diabetes care. Embedding AIP into routine reporting could help clinicians identify high-risk patients earlier, prioritize confirmatory urine testing, and intensify multifactorial therapy in alignment with KDIGO recommendations, particularly within resource-constrained settings.

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