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Atherogenic Dyslipidemia and Atherogenic Index of Plasma in Sudanese Adults with Type 2 Diabetes Mellitus: A Case Control Study

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Abstract

Background: The atherogenic index of plasma (AIP) is an inexpensive composite marker of atherogenic lipid risk that has emerged as a valuable tool in patients with type 2 diabetes mellitus (T2DM), a population which cardiovascular risk is driven substantially by atherogenic dyslipidemia. However case–control data quantifying AIP performance in Sudanese adults with T2DM are lacking.

Objective: To quantify the burden of atherogenic dyslipidemia and evaluate the diagnostic and independent predictive performance of AIP in Sudanese adults with T2DM relative to healthy controls.

Methods: In this case control study, 245 adults with T2DM and 245 age- and sex-matched healthy controls were recruited from three teaching hospitals in Al Jazirah State, Sudan. Anthropometric indices and a fasting lipid and glycemic profile were measured, and AIP was calculated as $\log_{10}$(triglycerides/HDL-cholesterol). Group differences were assessed with the Mann-Whitney U and Chi-square tests, associations with Spearman correlation, diagnostic performance with ROC curve analysis, and independent predictors with binary logistic regression adjusting for BMI, waist–hip ratio, and age.

Results: Cases showed a fully atherogenic lipid profile relative to controls, including significantly higher AIP (median 0.778 vs. 0.497, $p < 0.001$). Among cases, AIP correlated strongly with VLDL-C ($r = 0.638$, $p < 0.001$) but not with FBG, HbA1c, insulin, or HOMA-IR. AIP discriminated cases from controls with good accuracy (AUC = 0.821, 95% CI: 0.784–0.857; optimal cutoff 0.654, sensitivity 75.1%, specificity 73.5%) and remained an independent predictor of T2DM status after adjustment for BMI, waist–hip ratio, and age (Wald = 64.53, $p < 0.001$; overall model accuracy 87.6%).

Conclusion: Sudanese adults with T2DM carry a substantially greater atherogenic lipid burden than healthy controls, and AIP is an independent, low-cost marker of this risk that is distinct from glycemic status. AIP may serve as a practical adjunct for cardiovascular risk stratification in resource-limited settings.
Keywords: Type 2 diabetes mellitus; atherogenic dyslipidemia; atherogenic index of plasma; cardiovascular risk; case control study; Sudan.

Introduction

Type 2 diabetes mellitus (T2DM) affects approximately one in ten adults worldwide, and cardiovascular disease (CVD) is the leading cause of morbidity and mortality among this population. Adults with T2DM carry a two to four fold higher cardiovascular risk than their non-diabetic counterparts, and diabetes is associated with a 75% increase in all-cause mortality, with cardiovascular causes accounting for the majority of this excess (1,2). Monitoring this elevated risk in routine clinical practice, however, requires a marker that is both accessible and easy to apply. A need the atherogenic index of plasma (AIP) is well positioned to meet, as it can be calculated directly from a standard lipid panel without additional testing.

This elevated risk is driven substantially by a characteristic pattern of lipid disturbance known as atherogenic, or diabetic, dyslipidemia: elevated triglycerides, reduced HDL-cholesterol, and a preponderance of small, dense LDL particles (3). This triad arises primarily from insulin resistance, which increases hepatic secretion of large, triglyceride-rich VLDL1 particles that are subsequently remodeled into small, dense, highly atherogenic LDL and HDL particles (3,4).

Standard LDL-cholesterol measurement reflects total cholesterol mass rather than particle number or density, and can therefore appear misleadingly normal even when the underlying particle profile is highly atherogenic (4). AIP, calculated as the base-10 logarithm of the ratio of triglycerides to HDL-cholesterol, is obtained directly from a routine lipid panel and correlates strongly with directly measured LDL particle size, cholesterol esterification rate, and the concentration of atherogenic remnant lipoproteins making it a more accurate reflection of true atherogenic risk than LDL-C alone, at no additional cost (5).

Elevated AIP has been independently associated with major adverse cardiovascular events and all-cause mortality in a large cohort of patients with T2DM (6), and correlates significantly with established cardiovascular risk indices among African patients with T2DM in Uganda (7).

Despite this evidence, comparable case control data quantifying the prevalence and magnitude of atherogenic dyslipidemia and the discriminative performance of AIP against healthy controls remain scarce for Sudanese patients, where the prevalence of T2DM is substantial and largely characterized by poor glycemic control (8,9).

Most previous studies of AIP in T2DM have examined patients with diabetes alone, without a healthy reference group. Comparing T2DM patients directly against age and sex-matched healthy controls provides a stronger basis for judging how large the atherogenic burden of T2DM actually is, and allows AIP's ability to distinguish diabetic from non-diabetic status to be tested directly rather than simply describing associations within a single diabetic population. To our knowledge, no prior case control study has compared the full atherogenic lipid profile and AIP between Sudanese adults with T2DM and healthy, age- and sex-matched controls. We therefore conducted this

study to quantify the burden of atherogenic dyslipidemia in Sudanese adults with T2DM relative to non-diabetic controls, to characterize the relationship between AIP and glycemic and anthropometric parameters, to evaluate the diagnostic performance of AIP in distinguishing patients with T2DM from healthy controls, and to determine whether AIP remains independently associated with T2DM after adjustment for adiposity. Based on prior evidence linking AIP to insulin resistance and cardiovascular risk in T2DM populations, we hypothesized that AIP would be significantly elevated in Sudanese adults with T2DM compared with healthy controls, and would perform well in distinguishing between the two groups.

Materials and Methods

Study Design, Setting, and Population

This case–control study was conducted at three teaching-hospital outpatient clinics in Al Jazirah State, Sudan: the diabetes clinics of Alhasahisa Teaching Hospital and Rufaa Teaching Hospital, and the general medicine clinic of Elmanagel Teaching Hospital.

Two hundred forty-five adults with a prior diagnosis of T2DM made according to established clinical guidelines (cases) and 245 age- and sex-matched adults without diabetes (controls; fasting glucose <100 mg/dL, no known chronic disease, and no lipid-lowering therapy) were consecutively recruited. Individuals with type 1 diabetes, acute infectious illness, hepatic failure, renal failure, or pregnancy were excluded from both groups.

The required sample size was calculated using the standard formula for estimating a single population proportion: $n = Z^2p(1-p)/d^2$ (10), where Z is the standard normal deviate for a 95% confidence level (1.96), d is the acceptable margin of error (0.05), and p is the expected prevalence of the outcome of interest. Using the reported prevalence of intermediate-to-high-risk AIP category among African patients with T2DM (20.2%) (7), this calculation yielded a minimum required sample size of 248 participants per group; a sample of 245 age- and sex-matched participants per group was recruited for cases and controls, closely meeting this requirement.

Anthropometric and Biochemical Measurements

Anthropometric measurements were obtained with participants in light clothing and without shoes. Height and weight were recorded to the nearest 0.1 cm and 0.1 kg, respectively, and body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Waist circumference was measured at the midpoint between the lowest rib and the iliac crest, and hip circumference at the level of the greater trochanters; the waist–hip ratio (WHR) was calculated as waist/hip circumference.

Venous blood (5 mL) was collected from all participants after a 10–12-hour overnight fast. Fasting blood glucose, total cholesterol, triglycerides, and HDL-cholesterol were measured by enzymatic methods on a fully automated clinical chemistry analyzer; HbA1c and fasting insulin were measured by chemiluminescence immunoassay. LDL-cholesterol was calculated using the Friedewald equation (11), and VLDL-cholesterol as triglycerides/5, per the same formula (11). Non-HDL-cholesterol was calculated as total cholesterol minus HDL-cholesterol (12). The triglyceride/HDL-cholesterol ratio (13) and HDL/LDL-cholesterol ratio (14) were also calculated as complementary atherogenic lipid ratios. Insulin resistance was estimated using HOMA-IR [fasting insulin (μIU/mL) × fasting glucose (mg/dL)/405] (15). AIP was calculated as $\log_{10}$(triglycerides/HDL-cholesterol) (5).

Demographic and clinical data, as well as relevant medical history, were collected from all participants using a pre-tested, structured questionnaire developed specifically for this study. An English-language version of the questionnaire is available as Supplementary File (I).

Statistical Analysis

Data were analyzed using SPSS software (IBM Corp.). The Shapiro-Wilk test was used to assess normality of distribution for all continuous variables. Because study variables were non-normally distributed, continuous variables are presented as median (interquartile range) and were compared between cases and controls using the Mann-Whitney U test; categorical variables are presented as frequencies and percentages and were compared using the Chi-square test. Spearman's rho was used to assess correlations between AIP and metabolic and anthropometric parameters among cases. Receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminative performance of AIP and other study variables for case status, with the optimal cutoff identified using the Youden index (sensitivity + specificity − 1). Binary logistic regression, with AIP, BMI, WHR, and age entered as covariates, was used to identify independent predictors of T2DM case status, with results expressed as odds ratios (OR) and 95% confidence intervals (CI). A two-tailed p-value <0.05 was considered statistically significant throughout.

Ethical Consideration

This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of the Sudan University of Science and Technology Science. The Ministry of Health of Gezira State, Sudan (Date: 28th December 2025, Approval No: 21). Written informed consent was obtained from each participant. Demographic and clinical data were collected through a structured questionnaire.

Clinical trial number: Not applicable.

Results

Baseline Characteristics

A total of 245 patients with T2DM and 245 age- and sex-matched healthy controls were included. As shown in Table 1, cases and controls did not differ significantly in age (60 vs. 57 years, p = 0.078) or sex distribution (p = 0.277). However, cases had significantly higher BMI (28.0 vs. 21.1 kg/m², p < 0.001) and waist–hip ratio (1.00 vs. 0.87, p = 0.017) than controls, consistent with the known association between adiposity and T2DM.

Table 1. Demographic and Anthropometric Characteristics of T2DM Cases and Controls (n = 245 each)

Parameter	Cases (n=245) Median (IQR)	Controls (n=245) Median (IQR)	P-value
Age (years)	60 (53–66)	57 (54–61)	0.078
BMI (kg/m²)	28 (24.50–31.10)	21.10 (19.10–23.90)	<0.001
Waist–Hip Ratio	1 (0.96–1.03)	0.87 (0.83–0.93)	0.017
Male, n (%)	109 (44.5%)	121 (49.4%)	0.277

Parameter	Cases (n=245) Median (IQR)	Controls (n=245) Median (IQR)	P-value
Female, n (%)	136 (55.5%)	124 (50.6%)	0.277
Duration of Diabetes (years)	10 (7–13)	—	—

Continuous variables were non-normally distributed and are presented as median (interquartile range); compared using the Mann-Whitney U test. Sex distribution was compared using the Chi-square test.

Glycemic, Insulin Resistance, and Lipid Profile

Cases showed markedly higher fasting glucose, HbA1c, and a fully atherogenic lipid profile compared with controls higher total cholesterol, triglycerides, LDL-C, VLDL-C, non-HDL-C, and TG/HDL ratio, alongside lower HDL-C and HDL/LDL ratio (all $p < 0.001$; Table 2). Notably, fasting insulin was significantly lower in cases despite higher glucose, while HOMA-IR did not differ significantly between groups ($p = 0.092$) a pattern consistent with progressive β-cell secretory decline blunting the discriminative value of insulin-dependent indices in established T2DM. AIP was significantly higher in cases than controls (0.778 vs. 0.497, $p < 0.001$), confirming a substantially greater atherogenic burden in patients with T2DM.

Table 2. Glycemic, Lipid, and Atherogenic Index Parameters of T2DM Cases and Controls (n = 245 each)

Parameter	Cases Median (IQR)	Controls Median (IQR)	P-value
FBG (mg/dL)	149 (117–206)	78 (72–88)	<0.001
Insulin (μIU/mL)	8.60 (6.34–12.37)	19.80 (15.34–22.48)	<0.001
HbA1c (%)	8 (7–10.55)	5 (4.56–5.50)	<0.001
HOMA-IR	3.41 (2.30–4.77)	3.67 (2.98–4.46)	0.092
Total Cholesterol (mg/dL)	120 (92–190)	75 (70–87)	<0.001
Triglycerides (mg/dL)	158 (140–210)	110 (90–130)	<0.001
HDL-C (mg/dL)	28 (20–33)	34 (25–48)	<0.001
LDL-C (mg/dL)	62 (38.10–115.10)	20.20 (14.80–26)	<0.001
VLDL-C (mg/dL)	31.60 (28–42)	22 (18–26)	<0.001
Non-HDL-C (mg/dL)	97 (68–154.6)	43 (38–49)	<0.001
HDL/LDL Ratio	0.3929 (0.24–0.66)	1.7368 (1.12–2.83)	<0.001
TG/HDL Ratio	6 (4.49–8.83)	3.14 (2.42–4.63)	<0.001
AIP	0.7780 (0.6535–0.9465)	0.4970 (0.3855–0.6660)	<0.001

All variables were non-normally distributed and are presented as median (interquartile range); compared using the Mann-Whitney U test.

Correlation of AIP with Metabolic and Anthropometric Parameters

Among T2DM cases, AIP showed a strong, significant positive correlation with VLDL-C ($r = 0.638$, $p < 0.001$), consistent with VLDL being the principal triglyceride-carrying lipoprotein that drives the AIP calculation. AIP was

not significantly correlated with age, BMI, waist–hip ratio, diabetes duration, FBG, insulin, HbA1c, total cholesterol, LDL-C, or HOMA-IR (Table 3), indicating that AIP captures an atherogenic lipid dimension largely independent of glycemic control and conventional insulin resistance indices in this cohort.

Table 3. Spearman's rho Correlation of AIP with Study Parameters among T2DM Cases (n = 245)

Parameter	r	P-value
Age (years)	0.028	0.661
BMI (kg/m²)	−0.071	0.271
Waist–Hip Ratio	−0.017	0.796
Duration of Diabetes (years)	0.076	0.234
FBG (mg/dL)	−0.066	0.302
Insulin (µIU/mL)	−0.012	0.852
HbA1c (%)	0.018	0.777
Total Cholesterol (mg/dL)	−0.065	0.314
LDL-C (mg/dL)	−0.077	0.228
VLDL-C (mg/dL)	0.638**	<0.001
HOMA-IR	−0.094	0.144

Spearman's rho correlation analysis was used to assess the relationship between AIP and study parameters among T2DM cases, consistent with the non-normal distribution of study variables. Correlation is significant at the 0.05 level (2-tailed).

Diagnostic Performance (ROC Analysis)

ROC curve analysis (Table 4, Figure 1) identified HbA1c and FBG as the strongest discriminators of case status, as expected given their role in the diagnostic definition of T2DM itself (AUC = 0.995 and 0.970, respectively). Among the atherogenic lipid indices, AIP and the mathematically equivalent TG/HDL ratio showed good discriminative performance (AUC = 0.821, 95% CI: 0.784–0.857, $p < 0.001$), with an optimal cutoff of 0.654 yielding 75.1% sensitivity and 73.5% specificity. HOMA-IR showed no significant discriminative ability (AUC = 0.456, $p = 0.092$), reinforcing that AIP captures atherogenic risk information that conventional insulin resistance indices do not.

Table 4. Diagnostic Performance (ROC Analysis) of Study Variables for Discriminating T2DM Cases from Controls

Variable	AUC	Std. Error	95% CI	P-value	Optimal Cutoff	Sensitivity (%)	Specificity (%)
HbA1c	0.995	0.002	0.990–1.000	<0.001	5.95–6.10	97.6–98.8	96.7–98.0
FBG	0.970	0.008	0.955–0.986	<0.001	98.50	89.8	97.1
Non-HDL-C	0.955	0.009	0.937–0.972	<0.001	54.50	91.8	87.3
LDL-C	0.907	0.014	0.880–0.934	<0.001	30.20–32.50	81.2–84.5	85.7–89.0
Insulin	0.898*	0.014	0.871–0.926	<0.001	13.31	82.0	85.3
HDL/LDL Ratio	0.921*	0.012	0.896–0.945	<0.001	0.83	82.4	90.2

Variable	AUC	Std. Error	95% CI	P-value	Optimal Cutoff	Sensitivity (%)	Specificity (%)
AIP	0.821	0.018	0.784–0.857	<0.001	0.654	75.1	73.5
TG/HDL Ratio	0.821	0.018	0.784–0.857	<0.001	4.51	75.1	73.5
Triglycerides	0.809	0.020	0.770–0.848	<0.001	137.0–142.5	73.5–76.3	78.8–81.6
HOMA-IR	0.456	0.027	0.404–0.508	0.092	1.94	18.8	98.0

** Complementary AUC (1 − raw AUC) reported for Insulin and HDL/LDL Ratio, as the raw test direction was inverse (lower values associated with the case group); the 95% CI was correspondingly transformed as [1 − upper bound, 1 − lower bound]. Optimal cutoffs were identified using the Youden Index (Sensitivity + Specificity − 1). Abbreviations: AUC, area under the curve; CI, confidence interval.*

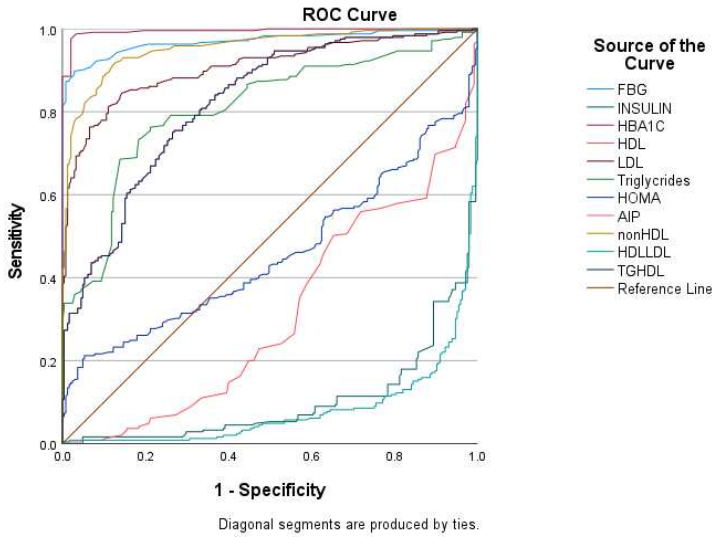


Figure 1. ROC curves of study variables in discriminating T2DM cases from controls.

Independent Predictors of T2DM Case Status

To determine whether AIP remained associated with T2DM after accounting for adiposity, a binary logistic regression model was fitted with AIP, BMI, waist–hip ratio, and age as covariates (Table 5). The model correctly classified 87.6% of participants overall (87.3% of cases, 87.8% of controls). AIP remained a highly significant independent predictor of case status after adjustment (Wald = 64.53, $p < 0.001$), alongside BMI and waist–hip ratio, while age was not significant ($p = 0.671$). This indicates that the atherogenic lipid burden captured by AIP contributes information about T2DM status beyond what is explained by adiposity alone.

Table 5. Binary Logistic Regression — Independent Predictors of T2DM Case Status (n = 490)

Variable	B	SE	Wald	P-value	OR (Exp B)	95% CI
BMI	−0.261	0.037	50.09	<0.001	0.770	0.717–0.828
Waist–Hip Ratio	−12.980	1.930	45.23	<0.001	<0.001	<0.001
Age	0.008	0.018	0.18	0.671	1.008	0.972–1.045
AIP	−6.291	0.783	64.53	<0.001	0.002	0.000–0.009

Dependent variable: participant status, coded with Control as the modeled outcome (1) per SPSS default alphabetical encoding. Consequently, negative B / Exp(B) < 1 indicates that higher values of the predictor are associated with increased odds of Case (T2DM) status — consistent in direction with Tables 1 and 2, where cases had higher AIP, BMI, and waist–hip ratio than controls. Overall classification accuracy: 87.6% (sensitivity for cases: 87.3%; specificity for controls: 87.8%).

Discussion

In this case control study of 245 Sudanese adults with T2DM and 245 healthy controls, patients with T2DM exhibited a substantially greater atherogenic lipid burden, with AIP more than 50% higher than in controls (median 0.778 vs. 0.497, $p < 0.001$). AIP discriminated cases from controls with good accuracy (AUC = 0.821) and remained a highly significant independent predictor of T2DM status after adjustment for BMI, waist–hip ratio, and age (Wald = 64.53, $p < 0.001$), indicating that the atherogenic burden it captures is not merely a proxy for adiposity.

These findings are consistent with the established pathophysiology of atherogenic dyslipidemia, in which insulin resistance drives hepatic overproduction of triglyceride-rich VLDL1 particles that are subsequently remodeled into small, dense LDL and HDL particles (3,4). This mechanism explains our correlation findings well: AIP correlated strongly with VLDL-C ($r = 0.638$, $p < 0.001$), the principal triglyceride-carrying lipoprotein, but showed no significant correlation with FBG, HbA1c, insulin, or HOMA-IR. Together, these results indicate that the atherogenic dyslipidemia captured by AIP arises in parallel with insulin resistance as a feature of the T2DM state itself, rather than as a direct downstream consequence of glycemic status consistent with evidence that lipid abnormalities often precede overt hyperglycemia in the natural history of T2DM (3,4).

Our diagnostic performance findings align with, and extend, prior evidence linking AIP to adverse outcomes in T2DM. Elevated AIP has previously been associated with major adverse cardiovascular events and mortality in a large cohort of patients with T2DM (6), and has correlated with established cardiovascular risk indices among African patients with T2DM in Uganda (7). Our AUC of 0.821 for distinguishing T2DM cases from healthy controls suggests that AIP not only tracks cardiovascular risk within T2DM populations, as shown previously, but also robustly distinguishes the diabetic atherogenic phenotype from a healthy metabolic state supporting its candidacy as a screening adjunct in populations, such as this one, where routine assessment of LDL particle size or advanced lipid subfractionation is not feasible. Non-HDL-cholesterol, which reflects the cholesterol content of all atherogenic apolipoprotein B-containing particles, also showed strong discriminative performance in our cohort (AUC = 0.955) and is well established as a superior predictor of cardiovascular risk compared with LDL-C alone (16), including specifically among patients with T2DM (17); together with AIP, it reinforces the broader picture of a comprehensively atherogenic lipid profile in this population.

The independent association between AIP and case status after adjustment for BMI and waist–hip ratio is a key strength of these findings. Given that CVD accounts for the majority of the two- to four-fold excess mortality risk observed in T2DM (1,2), and that this risk is driven substantially by atherogenic dyslipidemia rather than hyperglycemia alone (3,4), a marker that flags this risk independently of adiposity and glycemic control offers incremental clinical value. The strength of waist–hip ratio as an independent predictor in our model is consistent with the landmark INTERHEART case–control study, which found waist–hip ratio to be a substantially stronger predictor of myocardial infarction than BMI across 52 countries (18), reinforcing that central adiposity, alongside atherogenic dyslipidemia, is a key contributor to cardiovascular risk in this population. Because AIP requires only triglycerides and HDL-cholesterol both already part of routine lipid panels it can be calculated at no additional cost, making it a practical tool for cardiovascular risk stratification in resource-limited settings such as Sudan (7).

Strengths and Limitations

This study is, to our knowledge, the first case–control comparison of the full atherogenic lipid profile and AIP between Sudanese adults with T2DM and healthy, age- and sex-matched controls, with the strength of a multivariable model confirming independence from adiposity. Several limitations should be considered. The case control design precludes causal inference and does not permit estimation of true population incidence or relative risk. Recruitment from three centers within a single state limits generalizability to other regions of Sudan or other African populations with different dietary and genetic backgrounds. Data on antidiabetic and lipid-lowering medication history, which could independently influence AIP, were limited to the exclusion criteria applied at enrollment rather than adjusted for in the multivariable model. Finally, this study assessed AIP as a marker of atherogenic risk but did not include direct clinical cardiovascular endpoints; prospective studies linking AIP to incident cardiovascular events in Sudanese patients with T2DM are needed to confirm its prognostic utility in this population.

Conclusion

Sudanese adults with T2DM carry a substantially greater atherogenic lipid burden than healthy controls, reflected in significantly elevated AIP. AIP is an independent, low-cost marker of this burden, distinct from glycemic status and adiposity, and discriminates T2DM cases from healthy controls with good diagnostic accuracy. Incorporating AIP into routine lipid panel reporting could provide a practical, no-cost tool for cardiovascular risk stratification in resource-limited settings. Prospective studies linking AIP to incident cardiovascular outcomes in this population are warranted.

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Conflict of Interest

The authors declare that they have no conflicts of interest regarding the publication of this study.

Declaration of generative AI and AI-assisted technologies

During the preparation of this work, the authors used Gemini (Google) to assist with content organization, language polishing, improving readability, structural re-editing of the Discussion section, and drafting the cover letter. After using this tool, the authors reviewed, edited, and verified all content as needed, and take full responsibility and accountability for the content of the published article.

Consent for publication: Not applicable.

Abbreviations

AIP: Atherogenic Index of Plasma

AUC: Area Under the Curve

BMI: Body Mass Index

CI: Confidence Interval

CVD: Cardiovascular Disease

FBG: Fasting Blood Glucose

HbA1c: Hemoglobin A1c

HDL-C: High-Density Lipoprotein Cholesterol

HOMA-IR: Homeostatic Model Assessment for Insulin Resistance

LDL-C: Low-Density Lipoprotein Cholesterol

OR: Odds Ratio

ROC: Receiver Operating Characteristic

T2DM: Type 2 Diabetes Mellitus

TG: Triglycerides

VLDL-C: Very-Low-Density Lipoprotein Cholesterol

Wald: Wald chi-square test

WHR: Waist–Hip Ratio

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Author contributions: A.A.S. and A.E. conceptualized, designed the study, and designed the methodology, conducted field sampling, and performed data collection. A.A.S. and A.Y.H. carried out the statistical analysis, generated the figures, and analyzed the ROC curves. A.A.S., A.E., A.M.D., and A.Y.H. drafted the main manuscript text. A.M.D. and A.Y.H. critically reviewed, edited, and revised the intellectual content. All authors reviewed and approved the final manuscript.

Availability of data and materials:

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare no competing interests

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- DataCollectionQuestionnaireSupplementaryFile1.docx
- AIPRawDataSupplementaryFile2.xlsx