Association Between Atherogenic Index of Plasma and New-Onset Chronic Kidney Disease: Postprint

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Abstract

Background Chronic kidney disease (CKD) represents an increasingly serious public health challenge globally, with its incidence rising annually and being closely associated with the development of atherosclerosis. The atherogenic index of plasma (AIP), as a simple lipid-based indicator, has been demonstrated to effectively predict cardiovascular event risk. However, studies examining the relationship between AIP and CKD development risk remain insufficient and warrant further investigation.

Objective To investigate the association between AIP and new-onset CKD.

Methods A prospective cohort study was conducted using employees from the Kailuan Group in Tangshan City, Hebei Province, who underwent health examinations between June 2006 and October 2007 (n=101,510, including 81,110 males and 20,400 females, aged 18-98 years) as the study population. After applying inclusion and exclusion criteria, 85,253 individuals were included. Participants were stratified into four groups based on baseline AIP quartiles: Q1 (AIP <-0.58), Q2 (-0.58≤AIP <-0.17), Q3 (-0.17 ≤ AIP <0.29), and Q4 (AIP ≥ 0.29). Follow-up continued until December 31, 2021, with new-onset CKD as the endpoint. Cumulative incidence curves were generated using the Kaplan-Meier method, with intergroup differences assessed by Log-rank test. The association between AIP and CKD was analyzed using Cox proportional hazards regression models.

Results During a median follow-up of 13.97 (13.53, 14.17) years, 18,175 participants developed CKD. With increasing AIP, cumulative CKD incidence rates in Q1-Q4 were 16.87%, 21.49%, 22.31%, and 24.47%, respectively, with incidence densities of 13.48, 17.83, 18.56, and 20.77 per 1,000 person-years. After

adjusting for relevant confounders, Cox regression analysis revealed that compared with Q1, the HRs (95% CIs) for CKD in Q2-Q4 were 1.24 (1.18-1.29), 1.26 (1.21-1.33), and 1.51 (1.43-1.59), respectively (P<0.001). Sensitivity analyses excluding participants with CKD events in the first 2 years, those with all-cause mortality, those on antihypertensive, hypoglycemic, or lipid-lowering medications at baseline, and those with incident myocardial infarction or stroke yielded similar Q4 vs Q1 risk estimates, confirming robustness. Subgroup analyses revealed significant interactions between AIP and age, sex, BMI, hypertension history, and smoking status (P for interaction <0.001), with more pronounced AIP-associated risks observed in subgroups aged <60 years, males, BMI \$ $28kg/m^{2}$\$, and smokers (P<0.05).

Conclusion Elevated AIP is an independent risk factor for new-onset CKD and may enable earlier prediction of CKD development risk.

Full Text

Association between the Atherogenic Index of Plasma and New-onset Chronic Kidney Disease

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Abstract

Background: Chronic kidney disease (CKD) represents a major global public health challenge, with its prevalence rising annually and closely linked to atherosclerosis. The atherogenic index of plasma (AIP), a simple lipid-based indicator, has demonstrated predictive value for cardiovascular events. However, studies examining the association between AIP and CKD risk remain limited and require further investigation.

Objective: To investigate the association between AIP and new-onset CKD.

Methods: This prospective cohort study utilized data from 101,510 employees of the Kailuan Group in Tangshan, Hebei Province, who underwent health examinations between June 2006 and October 2007 (81,110 men and 20,400 women,

aged 18–98 years). After applying inclusion and exclusion criteria, 85,253 participants were enrolled. Participants were stratified into four groups based on baseline AIP quartiles: Q1 (AIP < -0.58), Q2 (-0.58 ≤ AIP < -0.17), Q3 (-0.17 ≤ AIP < 0.29), and Q4 (AIP ≥ 0.29). Follow-up continued until December 31, 2021, with new-onset CKD as the primary endpoint. Cumulative incidence curves were plotted using the Kaplan-Meier method, and intergroup differences were assessed using the log-rank test. The association between AIP and CKD was evaluated using Cox proportional hazards regression models.

Results: During a median follow-up period of 13.97 (13.53, 14.17) years, 18,175 participants developed CKD. With increasing AIP levels, the cumulative incidence of CKD in the Q1–Q4 groups was 16.87%, 21.49%, 22.31%, and 24.47%, respectively, with incidence densities of 13.48, 17.83, 18.56, and 20.77 per 1,000 person-years. After adjusting for relevant confounders, Cox proportional hazards regression analysis showed that compared with the Q1 group, the hazard ratios (HR) (95% CI) for incident CKD in the Q2–Q4 groups were 1.24 (1.18–1.29), 1.26 (1.21–1.33), and 1.51 (1.43–1.59), respectively (P < 0.001). Further analysis revealed that after excluding participants who developed CKD within the first 2 years of follow-up, those who died from any cause during follow-up, those taking antihypertensive, hypoglycemic, or lipid-lowering medications at baseline, and those who experienced myocardial infarction or stroke during follow-up, the risk in the Q4 group remained similar to the main analysis results, demonstrating robust findings. Subgroup analyses showed significant interactions between AIP and age, sex, BMI, hypertension history, and smoking history (P-interaction < 0.001), with more pronounced risks observed in subgroups of age < 60 years, men, BMI ≥ 28 kg/m^2, and those with smoking history (P < 0.05).

Conclusion: Elevated AIP is an independent risk factor for new-onset CKD and can serve as an early predictor of CKD risk.

Keywords: Renal insufficiency, chronic; Chronic kidney disease; Atherogenic index of plasma; Risk factors; Cox models

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1. Methods

1.1 Study Population

This study utilized 101,510 employees of the Kailuan Group in Tangshan, Hebei Province, who underwent health examinations at Kailuan General Hospital and its 11 affiliated hospitals between June 2006 and October 2007 (81,110 men and 20,400 women, aged 18–98 years) as the study population. **Inclusion criteria:** (1) participation in the 2006–2007 (first) health examination; (2) normal cognitive ability to complete questionnaires independently; (3) agreement to participate and provide informed consent. **Exclusion criteria:** (1) missing or extreme values for triglycerides (TG) or high-density lipoprotein cholesterol

(HDL-C); (2) previous CKD diagnosis; (3) history of malignant tumors. After excluding 1,472 participants with missing data or extreme values, 14,484 with previous CKD, and 301 with cancer history, 85,253 participants were ultimately included. This study complied with the Declaration of Helsinki and was approved by the Ethics Committee of Kailuan General Hospital (Approval No.: [2006] Medical Ethics No. 5). All participants provided written informed consent.

1.2 Data Collection

The collection process for demographic characteristics, clinical data, and laboratory measurements followed protocols detailed in our previously published literature [9]. **Laboratory data collection:** All participants provided 5 mL of fasting venous blood from the antecubital vein, collected in EDTA anticoagulant tubes. Samples were left at room temperature for 30 minutes, then centrifuged at 3,000 rpm for 10 minutes. Serum was collected and analyzed within 4 hours using a Beckman automated biochemical analyzer.

1.3 Diagnostic Criteria and Grouping

1.3.1 CKD Diagnostic Criteria According to the *Clinical Practice Guidelines for Chronic Kidney Disease and Dialysis* [10], CKD was diagnosed when estimated glomerular filtration rate (eGFR) < 60 mL $\cdot$ min^{-1} $\cdot$ (1.73 m^2)$^{-1}$. Serum creatinine (Scr) was measured using the enzymatic method [11], and eGFR was calculated using the CKD Epidemiology Collaboration equation [12] as shown in .

1.3.2 AIP Calculation and Grouping AIP was defined as the log-transformed ratio of TG to HDL-C, calculated as AIP = log(TG/HDL-C) [13,14]. Participants were stratified into four groups based on baseline AIP quartiles: Q1 (AIP $<$ -0.58), Q2 (-0.58 $\leq$ AIP $<$ -0.17), Q3 (-0.17 $\leq$ AIP $<$ 0.29), and Q4 (AIP $\geq$ 0.29).

1.3.3 Definitions of Covariates Diagnoses of hypertension, diabetes, and dyslipidemia followed Chinese guidelines [15-17]. Income level was categorized as $\geq$ 800 or $<$ 800 RMB per capita monthly income. Education level was dichotomized as high school or above versus junior high school or below. Physical exercise was defined as $\geq$ 3 sessions per week, each lasting $\geq$ 30 minutes. Smoking history was defined as $\geq$ 1 cigarette per day for $\geq$ 1 year. BMI was calculated as weight (kg)/height (m)^2.

1.4 Endpoint Ascertainment

The baseline examination (2006–2007) served as the starting point. Follow-up endpoints were new-onset CKD, death, or study end (December 31, 2021). For participants without endpoint events, follow-up ended on December 31, 2021;

for those who died without developing CKD, follow-up ended at the date of death.

1.5 Statistical Analysis

Data were analyzed using SAS 9.4 software. Normally distributed continuous variables were expressed as mean ± standard deviation and compared using one-way ANOVA; non-normally distributed variables were expressed as median (P25, P75) and compared using non-parametric rank-sum tests. Categorical variables were expressed as percentages and compared using ^{2} tests. Kaplan-Meier curves were plotted to show cumulative incidence, with log-rank tests for intergroup comparisons. The association between AIP and CKD was analyzed using Cox proportional hazards regression models. A two-sided $P < 0.05$ was considered statistically significant.

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2. Results

2.1 Baseline Characteristics

Among the 85,253 participants, 68,563 (80.42%) were men and 16,690 (19.58%) were women, with a mean age of 50.75 ± 12.15 years. Significant differences were observed across the four AIP groups in terms of AIP values, sex distribution, age, BMI, uric acid (UA), systolic blood pressure (SBP), diastolic blood pressure (DBP), fasting blood glucose (FBG), TG, total cholesterol (TC), HDL-C, LDL-C, high-sensitivity C-reactive protein (Hs-CRP), education level, per capita monthly income, physical exercise, smoking history, alcohol consumption, hypertension history, diabetes history, dyslipidemia, and use of antihypertensive, hypoglycemic, and lipid-lowering medications ($P < 0.05$) .

2.2 CKD Incidence

During a median follow-up of 13.97 (13.53, 14.17) years, 18,175 participants developed CKD, yielding an overall incidence rate of 26.51%. With increasing AIP levels, the proportion of CKD cases, incidence density, and cumulative incidence showed progressive increases across the Q1–Q4 groups (P-trend < 0.05) [TABLE:3, FIGURE:1].

2.3 Cox Proportional Hazards Regression Analysis

After adjusting for age, sex, smoking history, physical exercise, income level, education, diabetes history, dyslipidemia history, hypertension history, use of antihypertensive, hypoglycemic, and lipid-lowering medications, UA, BMI, SBP, DBP, TG, LDL-C, HDL-C, and Hs-CRP, the HRs (95% CI) for incident CKD in the Q2–Q4 groups compared with Q1 were 1.24 (1.18–1.29), 1.26 (1.21–1.33), and 1.51 (1.43–1.59), respectively (P-trend < 0.001) .

Restricted cubic spline analysis revealed a non-linear association between AIP and CKD risk (P-overall < 0.001, P-nonlinear < 0.001), with CKD risk increasing significantly when AIP $>$ -0.12 [Figure 2: see original paper].

2.4 Sensitivity Analysis

After sequentially excluding participants who developed CKD within the first 2 years of follow-up (n = 4,542), those who died during follow-up (n = 12,362), those taking antihypertensive, hypoglycemic, or lipid-lowering medications at baseline (n = 9,944), and those who experienced myocardial infarction or stroke during follow-up (n = 8,886), the risk estimates for the Q4 group remained consistent with the main analysis, demonstrating robust results .

2.5 Subgroup Analysis

Subgroup analyses were conducted by age ($\geq$ 60 vs. $<$ 60 years), sex (men vs. women), BMI ($\geq$ 28 vs. $<$ 28 kg/m^2), hypertension status, diabetes status, smoking history, and alcohol consumption history. Significant interactions were observed between AIP and age, sex, BMI, hypertension history, and smoking history (P-interaction < 0.001), but not for diabetes history or alcohol consumption history (P-interaction > 0.05). AIP showed more pronounced risks in subgroups of age $<$ 60 years, men, BMI $\geq$ 28 kg/m^2, and those with smoking history ($P < 0.05$) .

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3. Discussion

AIP is derived from TG and HDL-C values, reflecting the relationship between atherogenic and protective lipoproteins while also correlating with pre-atherogenic and anti-atherogenic lipoprotein particle size [7]. As major components of blood lipids, TG and HDL-C significantly influence atherogenesis. Previous research including 64,574 participants examined TG' s impact on acute myocardial infarction (AMI) over 10.92 (4.75, 12.83) years of follow-up, demonstrating that elevated TG is an independent risk factor for AMI in middle-aged and young populations. Even in the lowest and middle tertiles where TG levels were within the normal reference range ($\leq$ 1.66 mmol/L), prolonged cumulative exposure increased AMI risk, confirming TG' s atherogenic effect in real-world settings. TG's atherogenic properties stem from remnant lipoprotein cholesterol, which includes intermediate-density lipoprotein cholesterol, very low-density lipoprotein cholesterol remnants, and chylomicron remnants. The entire lipid metabolism process is a continuous, interrelated system where smaller, TG-rich lipoprotein cholesterol particles are hydrolyzed by lipoprotein lipase into denser, more atherogenic lipoprotein cholesterol particles [18]. Recent studies confirm that increased cholesterol in TG-rich lipoproteins correlates with higher ASCVD risk [19]. Another study of 92,297 participants examining HDL-C and

ASCVD over 7.9 years found that low HDL-C (< 1.23 mmol/L) increased ASCVD risk [20]. These findings demonstrate that elevated TG and/or reduced HDL-C are positively associated with ASCVD development. Their combined application as AIP better reflects the impact of lipid levels on atherosclerotic risk and may predict ischemic cardiovascular and cerebrovascular events earlier than atherosclerosis itself [7-8, 21].

Arterial stiffness increases new-onset CKD risk [4], consistent with our findings that higher AIP is associated with increased CKD risk. Compared with the Q1 group, the HRs (95% CI) for CKD in the Q2–Q4 groups were 1.24 (1.18–1.29), 1.26 (1.21–1.33), and 1.51 (1.43–1.59), respectively (P-trend < 0.001). Arterial stiffness promotes CKD development through various mechanisms. Post-stiffness endothelial dysfunction and reduced arterial wall elasticity significantly impair the buffering capacity of large arteries, leading to dramatic blood pressure fluctuations during systole and diastole, increased pulse pressure, and enhanced microcirculatory pulsatility. The kidney, characterized by low resistance and high flow, is particularly vulnerable to injury from high pulsatility, resulting in glomerular damage, hypoxia, and fibrosis that ultimately reduce glomerular filtration rate and accelerate CKD progression [22]. Elevated blood pressure exerts repeated minor mechanical stress on arterial walls, representing a major risk factor for atherosclerosis. However, brachial-ankle pulse wave velocity (BaPWV) predicts ASCVD risk better than blood pressure alone. A study of 47,659 participants with median 3.27-year follow-up found that after multivariable adjustment, the HRs for ASCVD were 1.29 (95% CI = 1.22–1.37) for elevated BaPWV, 1.28 (95% CI = 1.20–1.37) for SBP, and 1.26 (95% CI = 1.17–1.34) for DBP, indicating that hypertension as a risk factor for arterial stiffness is less direct and precise than BaPWV as a risk factor for ASCVD [23].

While BaPWV better predicts ASCVD risk than blood pressure, this also suggests that atherosclerosis results from multiple coexisting risk factors. Atherosclerotic plaque formation and progression are intimately linked to lipid levels, with plaques arising from lipid deposition in vessel walls. Although LDL-C, particularly small dense LDL-C (sdLDL-C), is the established primary pathogenic component of ASCVD, residual ASCVD risk persists even when LDL-C is controlled to normal ranges [24]. This highlights TG' s role throughout the entire lipid metabolism process, particularly in cholesterol metabolism. TG may promote atherosclerosis by serving as a carrier for remnant lipoprotein formation; these remnants are hydrolyzed by lipoprotein lipase into sdLDL-C, which penetrates arterial walls more readily than LDL-C, promoting atherosclerotic development [19, 25]. Our AIP measure aligns perfectly with this pathophysiology.

Elevated TG and reduced HDL-C with increased sdLDL represent the characteristic dyslipidemia pattern in diabetes, which reduces insulin sensitivity, promotes insulin resistance (IR), and increases residual ASCVD risk in diabetic patients [26]. A cross-sectional study of 7,369 participants using Chinese health and nutrition survey data found that diabetic individuals had higher AIP values

than non-diabetic individuals, and that AIP correlated with IR [27]. This may also explain why high BMI increases CKD risk [28]. The triglyceride-glucose (TyG) index, recognized as an IR surrogate marker that also incorporates TG, correlates with arterial stiffness. A meta-analysis of 16 observational studies published through January 14, 2024, found a strong correlation between TyG index and BaPWV, with higher TyG index indicating greater arterial stiffness and AS risk, suggesting TyG index may facilitate early AS diagnosis [30]. IR promotes inflammation and atherosclerosis through AIP [31], consistent with our finding that increased AIP can predict new-onset CKD risk earlier.

Clinical Value and Limitations: This cohort study' s large sample size and long follow-up period enhance the scientific robustness of our findings. Additionally, this study pioneers the use of a lipid-derived index reflecting atherosclerotic burden to assess CKD risk. Lipid testing is simple and widely available, making AIP highly feasible and reproducible for clinical practice, with results that can universally guide clinical decision-making. However, several limitations exist. First, the cohort had an imbalanced sex ratio, which we addressed through statistical adjustment. Second, AIP values were based on single measurements, which may introduce some bias; future studies could use average or cumulative AIP values to further strengthen scientific validity.

In conclusion, AIP, as a calculated index from routine lipid measurements, effectively predicts new-onset CKD risk, significantly enhancing risk assessment tools for CKD primary prevention and identifying high-risk individuals at an earlier, more actionable stage.

Author Contributions: ZHANG Xuechao and HAN Quanle conceived the study, designed the protocol, and drafted the manuscript. QI Qi and WU Xinyu conducted feasibility analysis, data collection, and statistical analysis. WU Shouling, LI Kangbo, LI Lei, DENG Jie, LI Cangtuo, and YUE Bocheng provided critical input on the study protocol. HAN Quanle critically reviewed the intellectual content, performed quality control, revised the final manuscript, and takes responsibility for the overall work.

Conflict of Interest: The authors declare no conflicts of interest.

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