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ORIGINAL RESEARCH

Association Between Uric Acid to High-Density Lipoprotein Cholesterol Ratio and Abdominal Aortic Aneurysm: A Single-Center Retrospective Study

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Objective: Uric acid (UA) to high-density lipoprotein cholesterol (HDL-C) ratio (UHR) has been recognized as a novel biomarker for evaluating inflammatory and anti-inflammatory interaction. However, it is not known whether UHR is related to abdominal aortic aneurysm (AAA). The current research aims to explore the potential role of UHR in predicting AAA.

Methods: In this study, 303 AAA patients and 408 normal subjects were retrospectively analyzed. The relationship between UHR and AAA was evaluated using Logistic regression models. Receiver operating characteristic (ROC) curves and restricted cubic spline (RCS) analysis were employed to elucidate the detailed association between UHR and AAA.

Results: UHR value in the AAA group was significantly higher than that in the normal group, and UHA was an independent risk factor for AAA. After adjusting for covariates, each 1-unit increase in UHR was associated with a 12% rise in AAA risk (OR: 1.12, 95% CI: 1.03, 1.21). ROC value of UHR was 0.847 (95% CI, 0.811~0.887, $P < 0.05$), and the optimal critical value of UHR was 17.2%. The incidence of AAA in the UHR≥17.2% group was significantly higher than that in the UHR < 17.2% group. RCS curves revealed a significant nonlinear relationship between UHR and AAA events (p-value < 0.001, p-nonlinear = 0.002).

Conclusion: This study demonstrates that UHR levels are significantly linked to increased AAA risk, which can be widely used as an indicator for dynamic screening of AAA.

Keywords: abdominal aortic aneurysms, UA, HDL-C, lipids, UHR

Introduction

Abdominal aortic aneurysm (AAA) is defined as localized dilatation of the abdominal aorta ≥50% of normal artery diameter, AAA rupture is an important cause of death in adults.[1,2] The treatment of AAA includes surgical treatment and medical treatment, and surgical treatment requires a comprehensive evaluation of the patient's condition.[3] In 2019, the global prevalence of AAA among individuals aged 30 to 79 years was 0.92%, which represents approximately 35.12 million cases.[4] Reports from population-based screening studies indicate a significantly higher prevalence of AAA in men compared to women, with rates ranging from 1.9% to 18.5% in men and from 0.1% to 4.2% in women.[5] AAA are often challenging for patients and medical personnel to detect. A comprehensive meta-analysis of 24 retrospective cohort studies has reported that the fatality rate following a rupture of an AAA is as high as 81%, with approximately one-third of those individuals dying before reaching the hospital.[6] Therefore, early identification of high-risk groups for AAA is crucial.

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Studies have shown that chronic inflammation of the vascular wall, abnormal proliferation and apoptosis of smooth muscle cells, increased expression of matrix metalloproteinases, and degradation of extracellular matrix all promote the course of AAA.[7–9] These pathological processes interact to accelerate the development of AAA.[10] The results of epidemiological and genetic studies show that the increase in serum uric acid level is closely related to atherosclerotic cardiovascular disease.[11,12] Hyperuricemia is associated with inflammatory response and oxidative stress and leads to endothelial dysfunction. High uric acid can lead to the occurrence and development of AAA by promoting an inflammatory response. High-density lipoprotein-cholesterol can reduce atherosclerosis through anti-inflammatory and antioxidant effects and reverse cholesterol transport, and HDL-C level is negatively correlated with the occurrence and development of AAA.[13] Recently, uric acid to high-density lipoprotein cholesterol ratio (UHR) has been recognized as a novel biomarker for evaluating inflammatory and anti-inflammatory interactions.[14] Recent studies have demonstrated a nonlinear relationship between UHR and brachial-ankle PWV.[15] Furthermore, research indicates that an increase in UHR is linked to a heightened risk of major adverse vascular events in patients with chronic total coronary occlusion.[16] Additionally, UHR has been shown to be a valuable predictor of the recurrence of atrial fibrillation.[17] However, as a novel biomarker reflecting the interaction between inflammation and anti-inflammatory, there are few studies on whether UHR is related to AAA.

To address this knowledge gaps, in this study, we intend to examine whether there is an association between AAA patients and the UHR, and our results may provide ideas for improving AAA risk stratification and clinical decision-making.

Materials and Methods

Study Design

This retrospective study complied with the Helsinki Declaration and was approved by The Affiliated Suzhou Hospital of Nanjing Medical University Ethical Committee (KL901463). Human participants' names have been removed from all sections of the manuscript. The Affiliated Suzhou Hospital of Nanjing Medical University Ethical Committee waived the need for informed consent. This study was conducted in accordance with the guidelines of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE).[18] We confirm that all methods were performed according to the relevant guidelines and regulations. The clinical data of patients hospitalized in the Vascular Surgery Center of Suzhou Hospital of Nanjing Medical University from January 2010 to January 2024 were analyzed retrospectively. The following clinical data were obtained: gender, age, diabetes mellitus (DM), body mass index (BMI), smoke, hypertension, statins, antiplatelet, metformin, uric acid (UA), low-density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), total cholesterol (TC), triglycerides (TG), fasting blood glucose (FBG), uric acid to high-density lipoprotein cholesterol ratio (UHR). The criteria for diagnosing an abdominal aortic aneurysm are as follows: (1) The diameter of the aorta is greater than 30 mm. (2) The aneurysm is located within the abdominal aorta.[19] The inclusion criteria for this study included the following: (1) The patient was hospitalized in the vascular surgery center. (2) All patients underwent complete aortic CTA examination with high-quality images. The exclusion criteria for this study included the following: (1) Patients with active inflammation and hematologic diseases. (2) Traumatic aortic aneurysm, syphilitic aortic aneurysm, pseudo aortic aneurysm, aortic dissection. (3) Patients with autoimmune diseases, connective tissue diseases, rheumatic immune diseases, or malignant tumors. (4) Patients with incomplete baseline or laboratory data. (5) Long-term use of corticosteroid drugs was excluded. After strict inclusion and exclusion criteria, the data of 303 patients diagnosed as AAA and 408 normal adults were analyzed retrospectively.

Statistical Analysis

Descriptive statistics are presented as median (interquartile range) or mean ± standard deviation for continuous variables and as frequency (percentage) for categorical variables. For quantitative variables that follow a normal distribution, we used analysis of variance (ANOVA) to assess group differences. When the quantitative variables do not adhere to a normal distribution, the Kruskal–Wallis test is employed to evaluate differences among groups. Categorical variables were analyzed using the Chi-square test.

Logistic proportional hazards regression models were utilized, which calculated odds ratio (ORs) along with 95% confidence intervals (95% CIs). Three models with varying levels of covariate adjustment were developed. Model 1 had no adjustments, Model 2 adjusted for gender and age, and Model 3 further adjusted for BMI, hypertension, statins,

smoke, diabetes, antiplatelet, TC, TG, FBG, and LDL-C. Furthermore, a fully adjusted restricted cubic splines (RCS) analysis was performed to investigate the dose-response relationship between UHR and the risk of AAA.

The "pROC" package is used to draw the receiver operating characteristic (ROC) curve to evaluate the predictive value of UHR for the incidence of AAA. The optimal cut-off point on the ROC curve is selected by the Youden index. The dose-response relationship between UHR and AAA risk was evaluated using restricted cubic splines (RCS) with four knots ("rms" package). Subgroup analyses were carried out to determine whether the relationship between UHR and AAA risk was consistent across various demographic and clinical categories, such as gender, age, BMI, hypertension, diabetes, statins, smoke, and antiplatelet. Interaction analyses were also conducted to examine potential modifications of AAA risk across these subgroups. To ensure the stability of the study findings, we implemented a sensitivity analyses. E-values were calculated based on Model 3 to assess the minimum strength of the link between unmeasured confounders and UHR, which could explain the observed relationship with AAA risk. All statistical analyses were executed employing Stata 17.0 and R version 4.2.2, with P-values < 0.05 deemed statistically significant.

Results

Population Characteristics

All patients' demographic characteristics are summarized in Table 1. Compared with the normal group, the AAA group was more male, relatively older, and had more smoking and hypertension patients.

UHR Is an Independent Predictor of AAA

To investigate whether UHR was useful as a predictor of AAA, Logistic regression analyses for the factors related to AAA were conducted in 711 subjects. The Logistic proportional hazard models confirmed a significant relationship between UHR and the risk of AAA. In Model 1, each 1-unit increase in UHR was linked to a 20% rise in AAA risk (OR: 1.20, 95% CI: 1.15, 1.24). Model 2 indicated that for each additional unit of UHR, there was a 19% increase in AAA risk (OR: 1.19, 95% CI: 1.14, 1.23). Model 3 illustrated a 12% increase in AAA risk for every 1-unit rise in UHR (OR: 1.12, 95% CI: 1.03, 1.21) (Table 2).

Table 1 Comparison of Baseline Characteristics Between AAA Group and Normal Group

Variables	Total (n = 711)	Normal (n = 408)	AAA (n = 303)	*P*
Gender				<0.001
Male	412 (57.95%)	183 (44.85%)	229 (75.58%)	
Female	299 (42.05%)	225 (55.15%)	74 (24.42%)	
Age (years)	61.41 ± 14.35	54.56 ± 12.86	70.64 ± 10.61	<0.001
BMI (kg/m^2)	24.45 ± 3.34	23.80 ± 3.20	25.33 ± 3.32	<0.001
UA (umol/L)	406.68 ± 148.36	358.74 ± 111.96	453.25 ± 164.72	<0.001
TC (mmol/L)	4.01 ± 1.07	3.96 ± 1.01	4.08 ± 1.14	0.154
TG (mmol/L)	2.03 ± 1.25	1.13 ± 0.60	3.25 ± 0.79	<0.001
FBG (mmol/L)	4.92 ± 3.51	4.80 ± 1.14	5.07 ± 5.21	0.322
LDL-C(mmol/L)	2.95 ± 0.93	2.64 ± 0.64	3.37 ± 1.09	<0.001
HDL-C(mmol/L)	1.03 ± 0.31	1.15 ± 0.34	0.92 ± 0.28	<0.001
UHR (%)	24.46 ± 23.82	12.20 ± 4.48	40.96 ± 28.82	<0.001
Hypertension	296 (41.63%)	105 (25.74%)	191 (63.04%)	<0.001
Statins	215 (30.24%)	123 (30.15%)	92 (30.36%)	0.951
Smoke	231 (32.49%)	70 (17.16%)	161 (53.14%)	<0.001
Diabetes	296 (41.36%)	171 (41.91%)	125 (41.25%)	0.860
Antiplatelet	230 (32.35%)	119 (29.17%)	111 (36.63%)	0.036

Abbreviations: BMI, body mass index; UA, uric acid; LDL-C, low-density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; TC, total cholesterol; TG, triglycerides; FBG, fasting blood glucose; UHR, uric acid to high-density lipoprotein cholesterol ratio.

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Table 2 Multivariable Logistic Regression Analysis of the Relationship Between UHR and AAA

Variables	OR	95% CI	P	Pseudo R^2
Adjusted Model I	1.20	1.15, 1.24	<0.001	0.309
Adjusted Model II	1.19	1.14, 1.23	<0.001	0.441
Adjusted Model III	1.12	1.03, 1.21	0.008	0.906

Notes: Model I: unadjusted. Model II: adjusted for gender and age. Model III: adjusted for gender, age, BMI, hypertension, statins, smoke, diabetes, antiplatelet, TC, TG, FBG, and LDL-C.

Abbreviations: OR, odds ratio; CI, confidence interval. BMI, body mass index; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglycerides; FBG, fasting blood glucose; UHR, uric acid to high-density lipoprotein cholesterol ratio; AAA, abdominal aortic aneurysm.

ROC Curve and RCS Regression Analysis

Establishing the ROC curve is integral for evaluating the sensitivity and specificity of AAA as a diagnostic tool. The Youden index selects the optimal cut-off point on the ROC curve. The ROC analysis determined that the predictive cut-off value of the UHR was 17.2%, and the area under the curve was 0.847 (95% CI, 0.811~0.887, $P < 0.05$) (Figure 1). The sensitivity and specificity of AAA were 0.776 and 0. 934 respectively. All 711 patients were divided into UHR<17.2% group and UHR≥17.2% group with the optimal cut-off value of UHR as the dividing line (Table 3), and

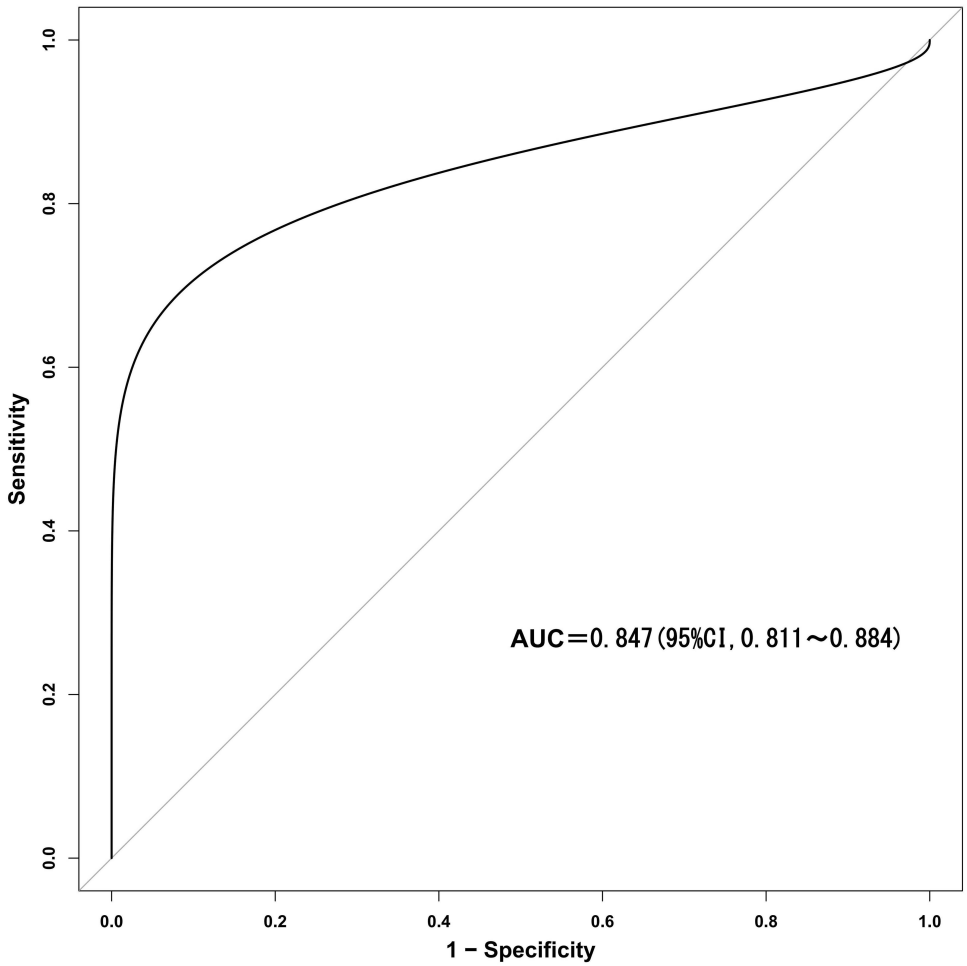

Figure 1 The receiving operating characteristic curve for UHR in predicting AAA.

Abbreviations: AAA, abdominal aortic aneurysm; UHR, uric acid to high-density lipoprotein cholesterol ratio.

Table 3 Baseline Characteristics Based on UHR Group

Variables	Total (n = 711)	UHR<17.2% (n = 449)	UHR≥17.2% (n = 262)	*P*
Gender				<0.001
Male	412 (57.95%)	216 (44.85%)	196 (74.81%)	
Female	299 (42.05%)	233(48.11%)	66 (25.195%)	
Age (years)	61.41 ± 14.35	57.08 ± 13.72	70.64 ± 10.61	<0.001
BMI (kg/m^2)	24.45 ± 3.34	23.98 ± 3.31	25.26 ± 3.23	<0.001
TC (mmol/L)	4.01 ± 1.07	4.00 ± 0.95	4.02 ± 1.24	0.851
TG (mmol/L)	2.03 ± 1.25	1.43 ± 0.92	3.06 ± 1.07	<0.001
FBG (mmol/L)	4.92 ± 3.51	4.75 ± 1.76	5.20 ± 5.30	0.186
LDL-C (mmol/L)	2.95 ± 0.93	2.75 ± 0.71	3.30 ± 1.15	<0.001
Hypertension	296 (41.63%)	148 (32.96%)	148 (56.49%)	<0.001
Statins	215 (30.24%)	134 (29.84%)	81 (30.92%)	0.764
Smoke	231 (32.49%)	93 (20.71%)	138 (52.67%)	<0.001
Diabetes	296 (41.63%)	192 (42.76%)	104 (39.69%)	0.424
Antiplatelet	230 (32.35%)	145 (32.29%)	85 (32.44%)	0.967

Abbreviations: BMI, body mass index; UA, uric acid; LDL-C, low-density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; TC, total cholesterol; TG, triglycerides; FBG, fasting blood glucose; UHR, uric acid to high-density lipoprotein cholesterol ratio.

the incidence of AAA was significantly different between the two groups (Figure 2). Adjusted RCS curves revealed a significant nonlinear relationship between UHR and AAA events (p-value < 0.001, p-nonlinear = 0.002) (Figure 3).

Subgroup Analysis and Interaction Analysis

We also conducted various subgroup analyses to assess whether UHR is consistent in different demographic characteristics. The results revealed that elevated UHR levels were consistent across different subgroups, including age < 65 years, BMI < 24, non-hypertension, and diabetes. To elucidate the potential interactions between these subgroups and the risk of AAA, we also conducted interaction analyses concurrently. Interaction analysis showed that UHR did not interact with these factors, which collectively affected AAA (Figure 4).

Sensitivity Analysis

To assess the stability of the outcomes, we conducted a sensitivity analyses. The E-value for UHR was calculated based on Model 3, revealing a value of 1.483, indicating that only a relatively large unmeasured confounding factor could explain the observed association.

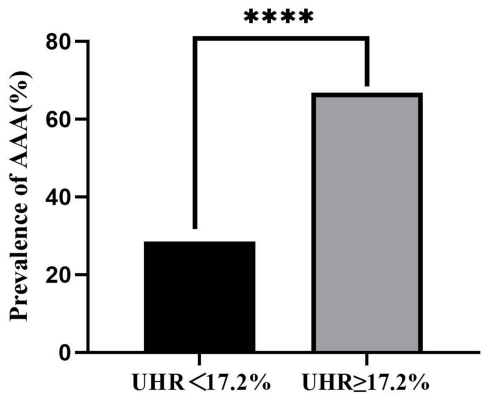

Figure 2 Prevalence of AAA in patients with different UHR classifications. ****, P < 0.0001.
Abbreviations: AAA, Abdominal aortic aneurysm; UHR, uric acid to high-density lipoprotein cholesterol ratio.

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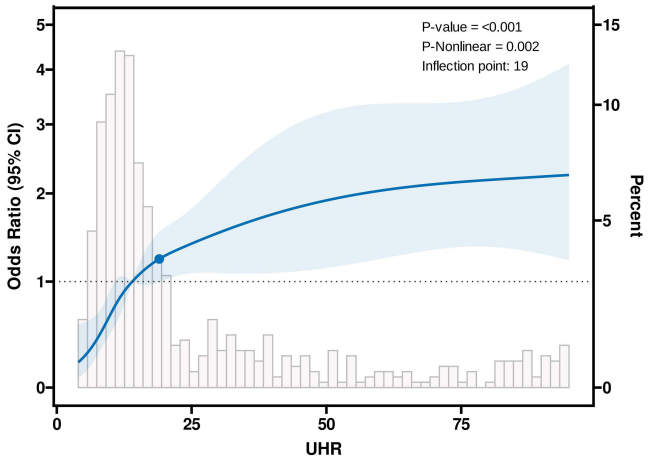

Figure 3 The dose-response relationship between UHR and AAA. Adjusted for gender, age, BMI, hypertension, statins, smoke, diabetes, antiplatelet, TC, TG, FBG, and LDL-C.

Abbreviations: BMI, body mass index; TC, total cholesterol; TG, triglycerides; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; AAA, abdominal aortic aneurysm; UHR, uric acid to high-density lipoprotein cholesterol ratio.

Subgroup	Adjusted OR (95% CI)*	P value	P for interaction
Overall	1.12 (1.03, 1.21)	0.008	
Gender			0.971
Female	1.12 (1.00, 1.26)	0.058	
Male	1.11 (0.97, 1.27)	0.128	
Age			0.576
< 65	1.17 (1.02, 1.36)	0.031	
≥ 65	1.10 (0.97, 1.23)	0.138	
BMI			0.283
< 24	1.21 (1.02, 1.43)	0.026	
≥ 24	1.12 (0.99, 1.27)	0.067	
Hypertension			0.745
No	1.15 (1.02, 1.30)	0.023	
Yes	1.08 (0.93, 1.24)	0.325	
Diabetes			0.848
No	1.11 (0.98, 1.27)	0.107	
Yes	1.17 (1.01, 1.36)	0.037	
Smoke			0.619
No	1.10 (0.99, 1.22)	0.069	
Yes	1.14 (0.97, 1.34)	0.115	

Figure 4 Subgroup analysis and interaction analysis. *, Adjusted for gender, age, BMI, hypertension, statins, smoke, diabetes, antiplatelet, TC, TG, FBG, and LDL-C.

Abbreviations: BMI, body mass index; TC, total cholesterol; TG, triglycerides; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; AAA, abdominal aortic aneurysm; UHR, uric acid to high-density lipoprotein cholesterol ratio.

Discussion

This is the largest sample size study so far on the correlation between UHR and AAA. In our study, we found that the increase in UHR was significantly correlated with the incidence of AAA. After adjusting other covariables, the relationship still existed, suggesting that UHR is an independent risk factor for the occurrence of AAA. The ROC analysis determined that the predictive cut-off value of the UHR AAA was 17.2%, and the area under the curve was 0.847; the AAA calculation rate of the UHR ≥ 17.2% group was significantly higher than that of the UHR < 17.2%

group. These findings underscore the usefulness of this simple and easily calculated measure for early identification of patients with AAA. The dose-response analysis found that this association between UHR and AAA was nonlinear.

Uric acid is the final product of purine catabolism, which is excreted by the kidney with urine and intestine. Hyperuricemia is related to DNA damage, oxidation, inflammatory cytokine production, and even apoptosis.[20,21] The Second Manifestations of Arterial Disease (SMART) study was a case-cohort study of 431 patients. During the follow-up period, 220 patients developed new vascular events, including AAA. The study showed that increased plasma uric acid levels increased the incidence of AAA.[22] Previous studies have shown that the uric acid level in the aortic aneurysm wall is significantly higher than in the normal aortic wall.[23] The relationship between serum uric acid levels and atherosclerosis has been studied for decades, and studies have shown that high uric acid levels promote the occurrence and development of atherosclerosis and, thus, the formation of aneurysms.[24,25] Uric acid can aggravate the occurrence and development of atherosclerosis through ERK/p38 mitogen-activated protein kinase (MAPK) cascade, AMP-activated protein kinase (AMPK), Phosphatidylinositol-3 kinase (PI3K)-Akt Pathway, Inflammasome, and other signal pathways.[26] Inflammation plays a vital role in the occurrence and development of AAA. It has been found that a large number of inflammatory cells infiltrate before the formation of AAA, and uric acid promotes the occurrence and development of aortic disease.[27] Oxidative stress is considered the beginning of vascular injury, and people in the patients with the AAA group detected a large number of reactive oxygen species (ROS).[28] It was reported that KLF11 expression was decreased in endothelial cells from human aneurysms, and it was time-dependently decreased in the aneurysmal endothelium from both elastase- and Pcsk9/AngII-induced AAA mouse models. The deficiency of KLF11 in endothelial cells significantly increased AAA formation, while selective overexpression of KLF11 in endothelial cells significantly inhibited AAA formation.[29] In summary, it is currently believed that uric acid can cause vascular injury through inflammatory response, oxidative stress, reduced availability of nitric oxide, endothelial dysfunction, vasoconstriction and proliferation of vascular smooth muscle cells, insulin resistance, and metabolic disorders, and then cause vascular dilation, leading to the formation of AAA.[30–32]

High-density lipoprotein cholesterol (HDL-C) is synthesized primarily in the liver and is an anti-atherosclerotic lipoprotein that transports cholesterol from extrahepatic tissues to the liver for metabolism and excretion by bile. In a nationwide Danish registry, it was found that AAA patients (n=6560) had lower average HDL-C concentrations than those with aortic and iliac artery occlusive disease (n=23,496).[33] An AAA is usually characterized by cholesterol accumulation and macrophage infiltration in the aortic wall. The ability of HDL-C to promote cholesterol excretion from macrophages is considered the primary vascular protection function of HDL-C, while the removal of cholesterol from macrophages by AAA patients under the promotion of HDL-C is damaged, which may be related to the AAA mechanism.[34] Through the analysis of plasma and tissues of AAA patients, it was found that the anti-HDL level of IgG in AAA patients increased, which indicated that there was a potential immune response against HDL-C in AAA and supported the new role of anti-HDL antibody in AAA.[35] The decrease of HDL-C, its anti-inflammatory, anti-oxidation, and anti-atherosclerosis effects are weakened, which can also lead to AAA.[36,37] It has been reported that the level of HDL-C is negatively correlated with the diameter of AAA, and targeted therapy for HDL-C can prevent the formation of AAA.[38]

In this study, we found that with each 1-unit increase in UHR, the risk of AAA incidence rises by 12%; this significant correlation remained even after adjusting for all potential covariates. More importantly, our study further revealed a significant nonlinear relationship between UHR and AAA events. This clearly demonstrates the critical need to monitor UHR levels to assess AAA risk accurately. The ROC analysis determined that the predictive cut-off value of the UHR AAA was 17.2%, and the area under the curve was 0.847. These findings underscore the usefulness of this simple and easily calculated measure for early identification of patients with AAA. It is worth noting that significant positive associations exist among individuals with age < 65 years, BMI < 24, non-hypertension, and diabetes. These findings emphasize the need for clinicians to focus on these specific subgroups and individuals. Interaction analysis showed that UHR did not interact with these factors in collectively affecting AAA. This suggests the generalizability of our study findings to a broad population.

UHR is considered an index of metabolic imbalance and evaluates the interaction between inflammation and anti-inflammation. Although the exact mechanism of the interaction between UHR and AAA is not fully understood, it can be explained as follows. First, the UHR index is reliable for evaluating the interaction between inflammation and anti-inflammation. Previous evidence shows that inflammation plays a vital role in the occurrence and development of AAA.[27,39] Secondly, UHR is usually related to metabolism, and the abnormality of the UHR index well reflects the

metabolic disorder in the body. Many studies have shown that metabolic disorders will promote the occurrence and development of AAA.[40–42] In addition, a higher UHR index is independently related to atherosclerosis, microvascular injury, and the occurrence and development of various cardiovascular diseases, which are high-risk factors for AAA patients.[15,43–45] Therefore, it seems reasonable to suggest that patients with higher UHR levels are more likely to be experiencing vascular injury, so the incidence of AAA increases. In clinical practice, our findings can guide clinicians to control UHR within a target range, which is beneficial to prevent AAA.

This study clarifies the role of the UHR in predicting AAA, offering a comprehensive approach to clinical decision-making that aligns with existing evidence. The global incidence of AAA patients is on the rise, making it crucial to identify high-risk individuals promptly. In clinical practice, evaluating a patient's UHR index can enhance disease assessment and facilitate the development of more effective, personalized treatment and management strategies. Furthermore, understanding the relationship between UHR and AAA can assist healthcare professionals in better assessing patient risks and in timely identification and management of potential complications.

However, this study also has many limitations. Firstly, this study is a retrospective study, which requires more prospective studies. Secondly, despite the use of multivariate adjustment, confounding factors may still bias the results, but the calculated E value indicates the robustness of the results, and the absence of confounding factors is improbable to alter the conclusions of this investigation. Thirdly, the population in this study may have a higher prevalence of abdominal aortic aneurysms (AAA) compared to the general population. As a result, the findings may not be entirely applicable to the broader population. Further research involving larger and more diverse samples is needed to verify these results. Lastly, the correlation between UHR and AAA diameter has yet to be explored. In the future, prospective studies will be designed to reduce selection bias and study the correlation between UHR and AAA diameter, and further animal experiments will be carried out to reveal the correlation mechanism between UHR and AAA.

Conclusion

In summary, UHR shows potential as an independent predictor of the incidence of AAA, and our study identified possible cut-off values for UHR in predicting AAA. Although further validation in a larger population is essential, the detection of UHR proves to be valuable for the early identification and risk stratification of individuals at high risk for AAA.

Patient Data Confidentiality Statement

This study follows strict data confidentiality and privacy protection protocols, with the following measures:

1. All study data has been anonymized and does not contain any personal information that could identify patients (eg, names, identification numbers, contact details, etc.).
2. The data is stored on secure servers, with access restricted to the research team members, all of whom have signed confidentiality agreements.
3. The use of the data is limited to the purposes of this study and will not be shared with third parties or used for other purposes.
4. During the publication of study results, all reported data will be in aggregated statistical form, ensuring that patient privacy is fully protected.

This study has been approved by the Ethics Committee of Suzhou Hospital Affiliated to Nanjing Medical University (Ethics Approval Number: KL901463), which has granted a waiver of patient informed consent. The reasons for the waiver are as follows:

1. This is a retrospective study that primarily utilizes historical data from the hospital's electronic health records (EHR) system. No additional interventions or treatments were administered to the patients, and the research poses minimal risk to them.
2. The data used in the study has been de-identified to ensure that no personally identifiable information of the patients will be disclosed or compromised.

3. The data collection and analysis strictly adhere to the Declaration of Helsinki and other relevant ethical guidelines to protect patient privacy and data security.

Data Sharing Statement

All data related to this study can be obtained upon reasonable request from the correspondent.

Ethics Approval and Consent to Participate

This paper did not involve the direct use of any human tissue. This study was conducted by the Declaration of Helsinki and was approved by the Ethics Committee of The Affiliated Suzhou Hospital of Nanjing Medical University. Written consent was obtained from all participants in the study.

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Disclosure

The authors of this manuscript declare no relationships with any companies whose products or services may be related to the subject matter of the article.

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