Original Research

OPEN ACCESS

Association between Uric Acid-to-High-Density Lipoprotein Cholesterol Ratio and Diabetic Kidney Disease in US Adults with Diabetes: A Cross-Sectional Study

Yang Yang  ***, Xin Yang, Keye Ding, Huan Shao****

Wuxi No.2 People's Hospital, Wuxi214016, Jiangsu, China

ARTICLE INFO

Article history:
Received 10 April 2026
Accepted 22 April 2026

Keywords:
Diabetic kidney disease
NHANES
Cross-sectional study

ABSTRACT

Background This study aimed to investigate the association between the uric acid (UA) to high-density lipoprotein cholesterol (HDL-C) ratio (UHR) and diabetic kidney disease (DKD) among American adults with diabetes. **Methods** Data from NHANES 2007-2016 were analyzed. Logistic regression and restricted cubic spline (RCS) models were used to evaluate the association between UHR and DKD risk. Additionally, the diagnostic performance of UHR for DKD was assessed using receiver operating characteristic (ROC) curves. Subgroup analyses and interaction tests were performed to examine the reliability and robustness of the findings. **Results** A total of 4,312 participants were included in the analysis. As a continuous variable, UHR was positively associated with an increased risk of DKD (OR 1.07, 95%CI 1.05–1.09, (P<0.05)). When stratified by quartiles, the fully adjusted model showed that the third and fourth UHR quartiles were associated with significantly higher odds of DKD compared with the lowest quartile (Q3: OR 1.70, 95%CI 1.31-2.22, (P<0.001); Q4: OR 2.44, 95%CI 1.86-3.18, (P<0.001)). RCS analysis indicated a linear relationship between UHR and DKD risk (P for nonlinearity = 0.502). The area under the ROC curve (AUC) of UHR for discriminating DKD was 0.708. Subgroup analyses revealed consistent associations across all subgroups, and interaction tests identified significant effect modifications by age, sex, and smoking status (all P for interaction <0.05). **Conclusion** UHR is significantly associated with DKD in US adults with diabetes. An elevated UHR may serve as a valuable biomarker for assessing DKD risk in adult patients with diabetes.

1. Introduction

Diabetes is a metabolic disorder characterized by hyperglycemia and multiple organ complications. In 2021, 529 million people of all ages were living with diabetes worldwide, and this number is projected to reach 1.31 billion by 2050 [1]. Microvascular complications represent a major category of chronic diabetic complications. Diabetic kidney disease (DKD) is a common microvascular complication affecting nearly 40% of patients with diabetes. DKD is the leading cause of chronic kidney disease (CKD) globally. DKD increases the risk of kidney failure, heart failure, atherosclerotic cardiovascular disease, and premature death. Therefore, several studies have emphasized that trajectories of function in diabetic adults can differ from the classical DKD phenotype, which is manifested by a decrease in glomerular filtration rate (eGFR) and the appearance of proteinuria [2]. Therefore, identifying potential biomarkers for DKD has become increasingly important.

Metabolic alterations due to diabetes result in glomerular hypertrophy, glomerulosclerosis, and inflammation and fibrosis of the tubulointerstitial area [3]. DKD is closely linked to both systemic and renal local inflammation [4]. Renal and systemic inflammation contribute to proteinuria, matrix deposition, and a progressive decline in the glomerular filtration rate (GFR) [5]. The uric acid (UA) to high-density lipoprotein cholesterol (HDL-C) ratio (UHR) serves as a significant biomarker, offering valuable insights into metabolic health and chronic inflammation [6]. Previous studies have demonstrated that high UHR is associated with an increased risk of type 2 diabetes, thyroiditis and metabolic syndrome [7][8][9]. A recent study further confirmed that UHR has favorable predictive value for CKD [10].

Emerging evidence suggests that UHR is associated with DKD. A Turkish study of 287 patients reported a significant relationship between UHR and DKD risk [11]. Another two studies conducted in China reached similar

* *Corresponding author: Huan Shao.*
ISSN: 3071-4265/ This work is licensed under a Creative Commons Attribution 4.0 International License.
DOI: 10.64904/20260678

conclusions [12][13]. However, the incidence and clinical presentation of DKD vary across countries [14]. Moreover, racial disparities in UHR levels may modify the association between UHR and DKD [15]. This study fills critical research gaps with three key innovations: first, it uses a large, nationally representative US sample (NHANES 2007–2016) to validate the UHR-DKD association across diverse ethnic groups; second, it includes 4,312 participants, markedly larger than prior small-sample studies (e.g., 287 cases in the Turkish study); third, it employs RCS analysis for the first time to confirm a linear dose–response relationship between UHR and DKD risk. Accordingly, this study aimed to examine the relationship between UHR and DKD in US adults with diabetes.

2. Material and methods

2.1. Study population

NHANES is an ongoing nationwide program designed to evaluate the nutritional and health status of the US population. The survey was approved by the Research Ethics Review Board of the National Center for Health Statistics, and all adult participants provided written informed consent. This study included data from five NHANES cycles (2007–2016), covering 50,588 participants. Data on demographics, physical examinations, laboratory tests, and questionnaires were collected. Participants were excluded if they were aged <20 years, did not have diabetes, had missing UHR data, or lacked complete covariate information. Finally, 4,312 participants were included in the analysis (Fig. 1).

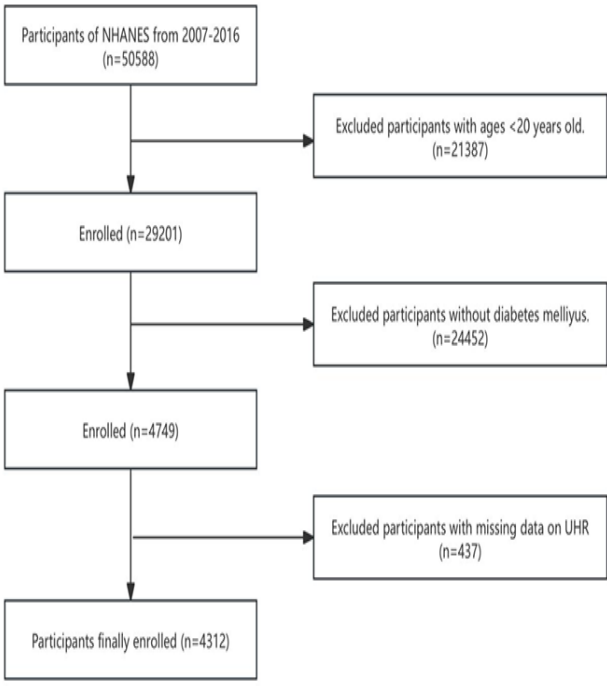

Fig.1 - Study Participant Selection Chart: A flow chart of participants selected from the NHANES study conducted from 2007 to 2016.

2.2. Definition of DKD

Diabetes was defined as: (1) self-reported physician diagnosis and use of oral antidiabetic agents or insulin; or (2) hemoglobin A1c (HbA1c) ≥6.5% or fasting blood glucose (FBG) ≥7.0 mmol/L [16]. DKD was defined as urinary albumin-to-creatinine ratio (UACR) ≥30 mg/g or eGFR <60 ml/min/1.73 m^2 [17]. The eGFR was calculated using the CKD-EPI equation [18].

- Female: $eGFR = 144 \times (sCr/0.7)^{-0.329} \times 0.993^{Age}$ if $sCr \leq 0.7$ mg/dL;
- $eGFR = 144 \times (sCr/0.7)^{-1.209} \times 0.993^{Age}$ if $sCr > 0.7$ mg/dL
- Male: $eGFR = 144 \times (sCr/0.9)^{-0.411} \times 0.993^{Age}$ if $sCr \leq 0.9$ mg/dL;
- $eGFR = 144 \times (sCr/0.9)^{-1.209} \times 0.993^{Age}$ if $sCr > 0.9$ mg/dL

2.3. UHR assessment

In this study, UHR was treated as the main exposure variable. UHR is calculated by dividing UA (mg/dL) by HDL-C (mg/dL), then multiplying by 100.

$$UHR\ (\%) = UA / HDL\text{-}C \times 100$$

2.4. Covariates assessment

Covariates included age, gender, race/ethnicity, education level, poverty income ratio (PIR), and body mass index (BMI). According to the Physical Activity Guidelines, we categorized participants into three groups: active (meeting or exceeding the suggested level), less active (below the suggested level) and inactive (absence of physical activity) [19]. Participants were classified as drinkers if they drank at least 12 different kinds of alcoholic beverages per year. Smoking status was defined as having ever smoked more than 100 cigarettes [20]. Hypertension was defined as self-reported hypertension or average systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg [21]. Total cholesterol (TC), aspartate aminotransferase (AST), and alanine aminotransferase (ALT) were also measured.

2.5. Statistical analysis

Sampling weights, strata, and primary sampling units (PSUs) from NHANES were applied to account for the complex multistage stratified sampling design. Continuous variables were expressed as mean ± standard deviation (SD) or median (interquartile range, IQR). Categorical variables were presented as percentages with 95% confidence interval (CI). UHR was divided into quartiles with the first quartile (Q1) as the reference group. Univariate and multivariable logistic models (Model 1, 2, and 3) were constructed. Model 1 was unadjusted; Model 2 adjusted for age, gender, and race/ethnicity; Model 3 adjusted for age, gender, race/ethnicity, education, physical activity, BMI, PIR, smoking, drinking, hypertension, ALT, AST, and TC. ROC curve analysis was used to assess the diagnostic performance of UHR. RCS regression was used to explore the linear relationship. Stratified analyses and interaction tests were performed using Model 3. EmpowerStats 4.2 and Stata 17.0 were used for analyses. A two-sided $P<0.05$ was considered statistically significant.

3. Results

3.1. Baseline characteristic

A total of 4,312 eligible participants were included, among whom 36.7% had DKD (Table 1). The mean age was 59.2 years, and 51.9% were aged ≥60 years. Males accounted for 51.7% of the study population. UHR quartiles were Q1:7.1%, Q2:10.8%, Q3:14.1%, Q4:20.8%. Significant differences were observed in gender, race, BMI, PIR, smoking, drinking, hypertension, DKD, ALT, AST, and TC across UHR quartiles (all (P<0.001). Higher UHR was associated with lower eGFR (P<0.001) (Table 1).

Table1 - Baseline characteristics of all study participants (n=4312) in the NHANES 2007-2016.

Variable	Total (n=4312)	Q1 (n=1077)	Q2 (n=1076)	Q3 (n=1080)	Q4 (n=1079)	P-value
Age, years	59.2±13.6	58.7±14.1	58.7±13.7	59.9±12.7	59.2±13.8	0.168
Age, %						0.422
＜60	48.1 (46.0–50.2)	49.7 (45.6–53.9)	49.1 (45.0–53.3)	46.7 (42.5–50.9)	47.2 (43.1–51.4)	
≥60	51.9 (49.8–54.0)	50.3 (46.1–54.4)	50.9 (46.7–55.0)	53.3 (49.1–57.5)	52.8 (48.6–56.9)	
Gender, %						＜0.001
Male	51.7 (49.6–53.7)	31.7 (27.9–35.7)	45.9 (41.8–50.1)	60.5 (56.5–64.4)	65.9 (61.8–69.8)	
Female	48.3 (46.3–50.4)	68.3 (64.3–72.1)	54.1 (49.9–58.2)	39.5 (35.6–43.5)	34.1 (30.2–38.2)	
Race/ethnicity, %						＜0.001
Mexican American	10.3 (9.5–11.1)	13.0 (11.2–15.1)	11.7 (10.0–13.6)	8.9 (7.5–10.6)	7.8 (6.5–9.4)	
Other Hispanic	5.9 (5.3–6.6)	7.5 (6.2–9.1)	6.5 (5.3–7.9)	5.0 (4.0–6.2)	4.9 (3.9–6.1)	
Non-Hispanic White	59.6 (57.7–61.4)	53.0 (49.0–57.0)	55.3 (51.4–59.2)	63.6 (60.0–67.0)	65.3 (61.7–68.7)	
Non-Hispanic Black	15.4 (14.4–16.5)	18.4 (16.2–20.9)	16.2 (14.1–18.4)	14.4 (12.5–16.5)	13.0 (11.3–15.0)	
Other Racea	8.9 (7.8–10.0)	8.0 (6.4–10.0)	10.3 (8.3–12.8)	8.1 (6.2–10.5)	9.0 (6.8–11.7)	
Education Level, %						0.618
Less than high school	25.5 (24.0–27.1)	25.1 (22.2–28.2)	27.0 (23.9–30.3)	25.1 (22.1–28.4)	24.9 (21.9–28.1)	
High school or equivalent	23.6 (21.9–25.4)	25.0 (21.4–28.9)	21.3 (18.1–24.9)	23.0 (19.8–26.7)	25.0 (21.6–28.8)	
More than high school	50.9 (48.8–52.9)	49.9 (45.7–54.0)	51.7 (47.5–55.8)	51.9 (47.7–56.0)	50.0 (45.9–54.2)	
Not recorded	0.1 (0.0–0.1)	0.1 (0.0–0.3)	0.1 (0.0–0.2)	0.0	0.1 (0.0–0.3)	
BMI, kg/m², %						＜0.001
＜25	10.9 (9.7–12.1)	20.1 (17.0–23.6)	13.0 (10.4–16.0)	7.5 (5.8–9.6)	4.1 (3.1–5.4)	
≥25–＜30	24.6 (22.9–26.3)	31.6 (27.9–35.6)	25.2 (22.0–28.7)	23.7 (20.4–27.3)	18.7 (15.9–21.8)	
≥30	62.7 (60.7–64.6)	47.1 (43.0–51.3)	60.9 (56.8–64.7)	67.3 (63.4–70.9)	73.5 (70.0–76.7)	
Not recorded	1.9 (1.5–2.5)	1.2 (0.6–2.2)	1.0 (0.5–1.8)	1.6 (0.9–2.6)	3.8 (2.6–5.4)	
PIR, %						＜0.001
≤1	16.7 (15.4–18.0)	17.5 (15.1–20.1)	16.5 (14.3–19.1)	16.6 (14.1–19.4)	16.2 (13.8–18.9)	
1.1–3.0	38.8 (36.8–40.8)	38.5 (34.6–42.6)	37.3 (33.5–41.3)	36.0 (32.3–39.9)	43.1 (39.1–47.2)	
＞3	36.9 (34.8–39.1)	35.0 (30.9–39.4)	36.9 (32.7–41.3)	40.5 (36.3–45.0)	35.2 (31.0–39.6)	
Not recorded	7.6 (6.7–8.7)	9.0 (6.8–11.8)	9.3 (7.2–11.9)	6.9 (5.2–9.0)	5.6 (4.4–7.1)	
Smoking status, %						＜0.001
Yes	51.6 (49.5–53.7)	44.7 (40.6–48.9)	46.2 (42.1–50.3)	54.9 (50.7–59.0)	59.5 (55.4–63.5)	
No	48.3 (46.3–50.4)	55.2 (51.1–59.3)	53.8 (49.6–57.9)	45.1 (41.0–49.3)	40.4 (36.4–44.5)	
Not recorded	0.0 (0.0–0.1)	0.1 (0.0–0.5)	0.0 (0.0–0.2)	0.0	0.0 (0.0–0.4)	
Physical activity, %						0.113
Inactive	45.4 (43.3–47.5)	43.2 (39.2–47.3)	44.9 (40.8–49.0)	43.5 (39.4–47.6)	49.7 (45.5–53.9)	
Less active	40.9 (38.9–43.0)	43.4 (39.2–47.6)	41.2 (37.2–45.4)	42.6 (38.4–46.8)	37.0 (33.0–41.1)	
Active	13.5 (12.1–14.9)	13.3 (10.8–16.3)	13.8 (11.1–16.9)	13.6 (11.2–16.5)	13.2 (10.5–16.5)	
Not recorded	0.2 (0.1–0.5)	0.2 (0.0–0.7)	0.1 (0.0–0.4)	0.3 (0.0–2.0)	0.2 (0.0–0.8)	
Drinking status, %						＜0.001
Yes	62.6 (60.6–64.5)	56.7 (52.6–60.7)	59.6 (55.6–63.6)	65.7 (61.8–69.3)	67.6 (63.8–71.1)	
No	30.8 (29.0–32.7)	34.7 (31.0–38.6)	35.1 (31.3–39.1)	28.5 (25.0–32.2)	25.7 (22.5–29.3)	
Not recorded	6.6 (5.7–7.5)	8.6 (6.6–11.0)	5.3 (3.9–7.1)	5.9 (4.4–7.7)	6.7 (5.1–8.8)	
Hypertension, %						＜0.001
Yes	70.6 (68.7–72.5)	64.6 (60.4–68.6)	65.8 (61.6–69.8)	72.7 (68.9–76.2)	78.3 (74.9–81.4)	
No	27.2 (25.4–29.1)	33.5 (29.5–37.6)	31.7 (27.8–36.0)	25.2 (21.8–28.9)	19.4 (16.5–22.7)	
Not recorded	2.2 (1.7–2.8)	1.9 (1.2–3.2)	2.4 (1.5–4.0)	2.1 (1.2–3.6)	2.2 (1.3–3.9)	
ALT, U/L	28.0±30.9	24.1±14.3	27.4±21.0	28.9±29.2	31.1±46.6	＜0.001
AST, U/L	27.3±20.6	25.2±13.1	26.7±15.3	27.4±16.8	29.5±30.9	＜0.001
TC, mg/dL	183.2±46.9	188.6±44.4	183.2±44.4	182.3±47.9	179.2±49.8	＜0.001
UA, mg/dL	5.7±1.6	4.3±1.0	5.3±1.0	6.1±1.0	7.1±1.5	＜0.001

HDL-C, mg/dL	46.8±14.0	61.9±15.1	49.0±9.4	43.0±7.3	35.1±7.2	<0.001
UHR, %	13.4±5.7	7.1±1.5	10.8±0.9	14.1±1.1	20.8±4.6	<0.001
eGFR, mL/min/1.73m²	82.4±25.1	88.0±23.3	85.6±24.1	81.3±24.7	75.8±26.3	<0.001
CKD, %						<0.001
Yes	36.7 (34.8–38.7)	28.4 (25.1–32.0)	32.4 (28.9–36.1)	38.0 (34.1–42.0)	46.8 (42.7–50.9)	
No	63.3 (61.3–65.2)	71.6 (68.0–74.9)	67.6 (63.9–71.1)	62.0 (58.0–65.9)	53.2 (49.1–57.3)	

3.2. Association between UHR and DKD

As a continuous variable, UHR was positively associated with DKD risk after full adjustment (OR=1.07, 95%CI 1.05-1.09, P<0.001). As a categorical variable, Q2 (OR=1.30, 95%CI 1.01-1.66, P=0.042), Q3 (OR=1.70, 95%CI 1.31-2.22, P<0.001) and Q4 (OR=2.44, 95%CI 1.86-3.18, P<0.001) had significantly higher DKD risk than Q1, with a significant dose–response trend (P<0.001) (Table 2). RCS analysis confirmed a linear relationship (P for nonlinearity=0.502) (Figure 2).

Table 2 Weighted correlation of UHR levels with DKD

Exposure	Model 1 OR (95%CI); P-value	Model 2 OR (95%CI); P-value	Model 3 OR (95%CI); P-value
UHR	1.06 (1.05–1.08); <0.001	1.07 (1.06–1.09); <0.001	1.07 (1.05–1.09); <0.001
UHR index quartile			
Q1	Reference	Reference	Reference
Q2	1.21 (0.95–1.53); 0.117	1.27 (1.00–1.63); 0.052	1.30 (1.01–1.66); 0.042
Q3	1.54 (1.21–1.96); <0.001	1.68 (1.30–2.17); <0.001	1.70 (1.31–2.22); <0.001
Q4	2.22 (1.75–2.81); <0.001	2.56 (1.99–3.30); <0.001	2.44 (1.86–3.18); <0.001
P for trend	<0.001	<0.001	<0.001

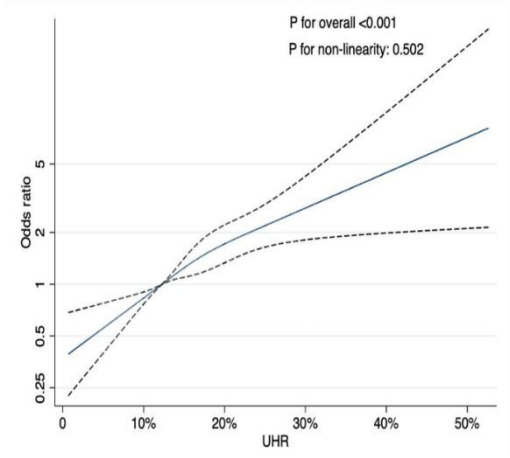

Fig. 2 RCS plots to assess the relationship between UHR levels and DKD rates in adults with diabetes in Model 3.

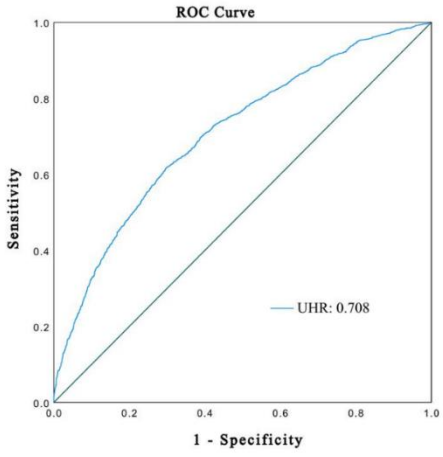

Fig. 3 ROC curve for UHR and DKD using logistic regression models.

3.3. ROC curve analyses of UHR for DKD

The ROC curve of UHR for predicting DKD is shown in Figure 3. The AUC of UHR for discriminating DKD was 0.708 (95% CI: 0.692–0.724), indicating that UHR is a moderate predictor of DKD.

3.4. Subgroup analysis and interaction tests

With UHR treated as a continuous variable, multiple subgroup analyses were conducted using different covariates to assess the consistency of the UHR–DKD relationship and to explore possible differences across populations (Fig. 4). A consistent positive association was observed across all subgroups (all P<0.05), regardless of age, sex, race, education level, physical activity, BMI, PIR, smoking status, drinking status, hypertension, ALT, AST, or TC. Furthermore, age, sex, and smoking status significantly modified the association (all P for interaction <0.05), whereas race, education, PIR, BMI, physical activity, drinking status, and hypertension did not.

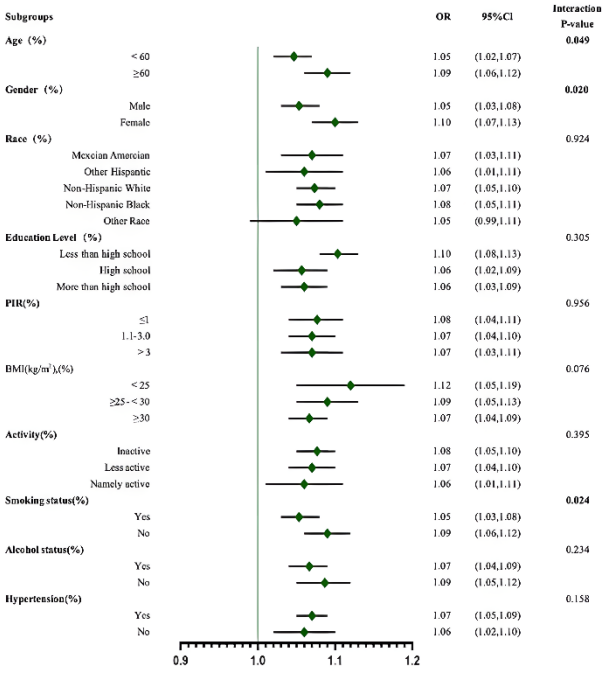

Fig.4 - Stratified analysis of the correlation between UHR and DKD. Stratification of covariates adjusted according to model III. OR, odds ratio; 95%CI, confidence intervals; BMI, body mass index; PIR, poverty income ratio.

4. Discussion

This study demonstrates that elevated UHR is positively associated with a higher risk of DKD in US adults with diabetes. Each 1-unit increase in UHR was associated with a 7% increase in DKD prevalence. These results are consistent with previous small-sample studies [10][11][12], and our study provides three key advancements: validation in a large, nationally representative US sample with diverse ethnicities, a much larger sample size (4,312 vs. 287), and the first confirmation of a linear UHR-DKD relationship via RCS analysis.

Hyperuricemia promotes renal tubular injury, oxidative stress, and interstitial fibrosis by activating the NLRP3 inflammasome and increasing pro-inflammatory cytokines such as IL-1β and TNF-α [22][23]. Meanwhile, reduced HDL-C loses its anti-inflammatory and antioxidant properties, exacerbating endothelial dysfunction and renal lipid accumulation [24][25]. The combined effect of high UA and low HDL-C amplifies metabolic disturbance and renal inflammation, forming a vicious cycle that accelerates DKD progression [26][27]. UHR integrates these two metabolic abnormalities, making it a sensitive indicator of renal inflammatory and fibrotic processes in diabetes [28].

Purine metabolism results in uric acid as its final product. Hyperuricemia is connected to increased pro-inflammatory cytokines, boosting UA production and creating an inflammation-UA cycle [22]. Hyperuricemia increases oxidative stress, a key driver of metabolic disturbances [23]. Diabetes is linked to serum uric acid, an independent CKD risk facto [24][25]. Diabetic dyslipidemia features hypertriglyceridemia and low HDL-C [26]. HDL-C has antioxidant and anti-inflammatory functions that may be lost in CKD [27]. UHR is a novel inflammatory and metabolic marker reflecting metabolic changes and predicting CKD [28]. Our study found highest UHR quartiles had highest UA, highest DKD rate, and lowest HDL-C, supporting UHR as a DKD predictor.

The AUC of 0.708 indicates moderate predictive power for UHR alone. While UHR cannot serve as a standalone diagnostic tool for DKD, it can be integrated into a multivariable predictive model alongside traditional markers (eGFR, UACR, HbA1c) to improve risk stratification accuracy in clinical practice. Subgroup analysis showed consistent associations, with age, gender and smoking status as significant effect modifiers, consistent with prior reports on diabetic renal progression [29][30].

5. Strengths and limitations

This research has a lot of benefits. Firstly, the NHANES database was utilized to ensure sample diversity and representativeness, with strict compliance with its guidelines. Secondly, this study examined the relationship between UHR and DKD using logistic regression models. Thirdly, covariates were meticulously adjusted according to relevant previous research. Fourthly, the RCS and ROC curve demonstrated that UHR is a moderate predictor of DKD. Fifthly, the use of subgroup analysis and interaction tests significantly strengthened the study's findings in terms of validity and reliability.

However, there are a number of restrictions on this research. Firstly, the evidence connecting UHR and DKD may be weaker due to the cross-sectional design. Further prospective studies should be taken to confirm these findings. Secondly, even though some confounding variables were considered, not every potential confounder was accounted for. Thirdly, using features from the NHANES database to diagnose DKD might lead to some bias.

6. Conclusions

UHR is significantly associated with increased DKD risk in US adults with diabetes. Elevated UHR may be a reliable biomarker for DKD risk assessment. Future prospective studies are needed to confirm causality, and intervention studies are required to explore whether UHR reduction can lower DKD risk, providing new strategies for DKD prevention and treatment.

A.1. Author contributions

DKY used the software for data analysis, tabulation, and graphing. YY interpreted the results and wrote the manuscript. YX provided conceptual and methodological guidance. SH collected data and reviewed the manuscript. All authors approved the final version.

A.2. Funding

Supported by Wuxi Pharmacists Association – Wuxi Pharmaceutical Research Project (No.202501012).

REFERENCES

[1] GBD 2021 Diabetes Collaborators. Global, regional, and national burden of diabetes from 1990 to 2021, with projections of prevalence to 2050: a systematic analysis for the Global Burden of Disease Study 2021. Lancet. 2023;402(10397):203-234.

[2] Oshima M, Shimizu M, Yamanouchi M, et al. Trajectories of kidney function in diabetes: a clinicopathological update. Nat Rev Nephrol. 2021;17(11):740-750.
[3] Alicic RZ, Rooney MT, Tuttle KR. Diabetic kidney disease: challenges, progress, and possibilities. Clin J Am Soc Nephrol. 2017;12(12):2032-2045.
[4] Taslamacioglu Duman T, Ozkul FN, Balci B. Could systemic inflammatory index predict diabetic kidney injury in type 2 diabetes mellitus? Diagnostics (Basel). 2023;13(12):2063.
[5] MacIsaac RJ, Jerums G, Koschinsky T, et al. Diabetic kidney disease. Nat Rev Dis Primers. 2015;1:15018.
[6] Aktas G, Khalid A, Kurtkulagi O, et al. Poorly controlled hypertension is associated with elevated serum uric acid to HDL-cholesterol ratio: a cross-sectional cohort study. Postgrad Med. 2022;134(3):297-302.
[7] Kocak MZ, Aktas G, Erkus E, et al. Serum uric acid to HDL-cholesterol ratio is a strong predictor of metabolic syndrome in type 2 diabetes mellitus. Rev Assoc Med Bras. 2019;65(1):9-15.
[8] Aktas G, Kocak MZ, Bilgin S, et al. Uric acid to HDL cholesterol ratio is a strong predictor of diabetic control in men with type 2 diabetes mellitus. Aging Male. 2020;23(5):1098-1102.
[9] Kurtkulagi O, Tel BMA, Kahveci G, et al. Hashimoto's thyroiditis is associated with elevated serum uric acid to high density lipoprotein-cholesterol ratio. Rom J Intern Med. 2021;59(4):403-408.
[10] Cheng Y, Zhang H, Zheng H, et al. Association between serum uric acid/HDL-cholesterol ratio and chronic kidney disease: a cross-sectional study based on a health check-up population. BMJ Open. 2022;12(12):e066243.
[11] Aktas G, Yilmaz S, Kantarci DB, et al. Is serum uric acid-to-HDL cholesterol ratio elevation associated with diabetic kidney injury? Postgrad Med. 2023;135(5):519-523.
[12] Han R, Duan L, Zhang Y, et al. Serum uric acid is a better indicator of kidney impairment than serum uric acid-to-creatinine ratio and serum uric acid-to-high-density lipoprotein ratio: a cross-sectional study of type 2 diabetes mellitus patients. Diabetes Metab Syndr Obes. 2023;16:2695-2703.
[13] Xuan Y, Zhang W, Wang Y, et al. Association between uric acid to HDL cholesterol ratio and diabetic complications in men and postmenopausal women. Diabetes Metab Syndr Obes. 2023;16:167-177.
[14] Parving HH, Lewis JB, Ravid M, et al. Prevalence and risk factors for microalbuminuria in a referred cohort of type II diabetic patients: a global perspective. Kidney Int. 2006;69(11):2057-2063.
[15] Zhou X, Xu J. Association between serum uric acid-to-high-density lipoprotein cholesterol ratio and insulin resistance in an American population: a population-based analysis. J Diabetes Investig. 2024;15(6):762-771.
[16] American Diabetes Association Professional Practice Committee. 2. Diagnosis and classification of diabetes: Standards of Care in Diabetes—2025. Diabetes Care. 2025;48(Suppl 1):S27-S49.
[17] Kidney Disease: Improving Global Outcomes (KDIGO) Glomerular Diseases Work Group. KDIGO 2021 clinical practice guideline for the management of glomerular diseases. Kidney Int. 2021;100(4 Suppl):S1-S276.
[18] Rognant N, Lemoine S, Laville M, et al. Performance of the chronic kidney disease epidemiology collaboration equation to estimate glomerular filtration rate in diabetic patients. Diabetes Care. 2011;34(6):1320-1322.
[19] Piercy KL, Troiano RP, Ballard RM, et al. The Physical Activity Guidelines for Americans. JAMA. 2018;320(19):2020-2028.
[20] Qi X, Wang S, Huang Q, et al. The association between non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio (NHHR) and risk of depression among US adults: a cross-sectional NHANES study. J Affect Disord. 2024;344:451-457.
[21] McEvoy JW, McCarthy CP, Bruno RM, et al. 2024 ESC Guidelines for the management of elevated blood pressure and hypertension. Eur Heart J. 2024;45(38):3912-4018.
[22] Sun S, Qin F. The association between dietary antioxidant quality score and uric acid related mortality in patients with chronic kidney disease. Front Nutr. 2024;11:1408898.
[23] Fan Z, Li Z, Guo A, et al. The association of low serum uric acid with mortality in older people is modified by kidney function: National Health and Nutrition Examination Survey (NHANES) 1999-2018. BMC Nephrol. 2024;25(1):108.
[24] Ames BN, Cathcart R, Schwiers E, et al. Uric acid provides an antioxidant defense in humans against oxidant- and radical-caused aging and cancer: a hypothesis. Proc Natl Acad Sci U S A. 1981;78(11):6858-6862.
[25] Wei YH, Lee HC. Oxidative stress, mitochondrial DNA mutation, and impairment of antioxidant enzymes in aging. Exp Biol Med (Maywood). 2002;227(9):671-682.
[26] Kane JP, Pullinger CR, Goldfine ID, et al. Dyslipidemia and diabetes mellitus: role of lipoprotein species and interrelated pathways of lipid metabolism in diabetes mellitus. Curr Opin Pharmacol. 2021;61:21-27.
[27] Krusinska B, Wadolowska L, Slowinska MA, et al. Associations of dietary patterns and metabolic-hormone profiles with breast cancer risk: a case-control study. Nutrients. 2018;10(12):2013.
[28] Liu R, Peng Y, Wu H, et al. Uric acid to high-density lipoprotein cholesterol ratio predicts cardiovascular mortality in patients on peritoneal dialysis. Nutr Metab Cardiovasc Dis. 2021;31(2):561-569.
[29] Aktas G, Kocak MZ, Bilgin S, et al. Uric acid to HDL cholesterol ratio is a strong predictor of diabetic control in men with type 2 diabetes mellitus. Aging Male. 2020;23(5):1098-1102.
[30] Afghahi H, Cederholm J, Eliasson B, et al. Risk factors for the development of albuminuria and renal impairment in type 2 diabetes—the Swedish National Diabetes Register (NDR). Nephrol Dial Transplant. 2011;26(4):1236-1243.