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Nonlinear association between the serum uric acid-to-creatinine ratio and all cause mortality in patients with hypertension: a ten-year cohort study using the NHANES database

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The serum uric acid-to-creatinine ratio (UCR) may be a simple method for assessing xanthine oxidase overactivation, which may contribute to an increase in serum uric acid production and oxidative stress. In this study, we investigated the nonlinear association between the UCR and long-term mortality in patients with hypertension. Data were acquired from the National Health and Nutrition Examination Survey database, and a total of 11,346 patients with hypertension were included. We explored the nonlinear link between the UCR and all-cause mortality via spline smoothing, threshold saturation, and log-likelihood ratio tests. The results were validated through a competing risk model. A nonlinear pattern emerged between the UCR and all-cause mortality in hypertensive patients, with an inflection point at 4.3. Below this point, an increased UCR was associated with a decreased mortality risk (*OR* = 0.80, 95% *CI*: 0.68–0.94, *P* = 0.008), whereas above this point, the risk increased (*OR* = 1.21, 95% *CI*: 1.07–1.36, *P* = 0.004). The competing risk model yielded similar findings for cardiovascular and chronic kidney disease-related deaths. In patients with hypertension, the UCR nonlinearly predicted all-cause mortality, with a notable inflection at 4.3. These findings suggest that the UCR is a valuable prognostic indicator for assessing long-term outcomes in patients with hypertension.

Keywords Hypertension, Uric acid-to-creatinine ratio, Mortality, Spline smoothing, Competing risk model

The global prevalence of hypertension, a condition characterized by persistently high blood pressure, has increased from 648 million to 1.278 billion individuals between 1990 and 2019[1]. The development of hypertension often leads to a series of complications, including stroke, renal failure, cardiac hypertrophy, myocardial infarction, and heart failure[2,3]. Hypertension is often accompanied by cardiovascular disease (CVD), which affects more than one billion people worldwide[4,5]. The latest Global Burden of Disease study revealed that ischaemic heart disease was the leading cause of age-standardized deaths in 2021[6]. Among these diseases, coronary artery disease (CAD) is the most common, and hypertension is a major contributor to its development, resulting in a significant disease burden[7]. The arterial walls of hypertensive patients are subjected to physical forces that promote increased pressure and arterial stiffness, potentially leading to myocardial structural changes and coronary microvascular dysfunction, further causing ischaemia and fibrosis[8]. Sustained hypertension also contributes to arteriosclerosis, affecting normal blood flow and reducing blood flow to the kidneys, thereby affecting kidney health[9]. Renal injury commonly manifests 5–10 years after hypertension onset, with hypertension being the primary driver of renal disease progression[10]. Therefore, early health assessment of patients with hypertension is essential prior to the onset of complications. Simple biomarkers for monitoring disease risk are valuable for the early assessment, management, and treatment of patients with hypertension.

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Uric acid (UA), the end product of purine metabolism, is widely present in intracellular and extracellular fluids and is primarily excreted by the kidneys[11]. Previous studies have suggested that elevated UA levels are an important risk marker for the development of hypertension from a normal high blood pressure[12]. UA can increase intracellular oxidative stress by activating the renin–angiotensin system, thereby contributing to hypertension[13]. Serum creatinine (Cr) is one of the most common biomarkers for assessing renal insufficiency and is associated with hypertension and cardiovascular risk[14,15]. The causal relationship between abnormal renal function or failure and the development of hyperuricaemia remains unclear in hypertensive populations[16–18]. The serum uric acid-to-creatinine ratio (UCR) may be a simple method of assessing xanthine-oxidase overactivation, which may contribute to an increase in UA production and oxidative stress[19]. Many studies have suggested that the UCR is associated with metabolic disorders and has predictive value in studying the progression and prognosis of metabolic syndrome, CVD, fatty liver disease, diabetes, chronic kidney disease, preeclampsia and, in general, hypertensive disorders during pregnancy[20–27].

However, evidence regarding the correlation between the UCR and hypertension-related mortality risk in prospective cohort studies is limited. Therefore, the aim of this study was to explore the relationship between the UCR and mortality risk in hypertensive individuals via the National Health and Nutrition Examination Survey (NHANES) database to provide a basis for the early assessment of hypertensive patients.

Methods

Data source and study population

In the present study, we performed a cohort analysis via publicly available NHANES data. The NHANES is a nationally representative survey that assesses the health and nutritional status of the civilian population in the United States. All the data are available on the official website (https://www.cdc.gov/nchs). The Ethical Review Committee of the National Center for Health Surveys (NCHS) approved the NHANES study protocol, and all participants provided informed consent. All the experiments described in this paper were performed in accordance with the relevant guidelines and regulations.

From 1999 to 2008, a total of 28,852 adult patients from different races and different regions were included in the NHANES database. Among them, 12,109 participants suffered from hypertension. Hypertension status was assessed via computer-assisted personal interviews and questionnaires. A total of 757 participants were excluded because of missing information on UA and creatinine levels. Six participants were excluded from the study because of a lack of sufficient information to link with the National Death Index (NDI) data and thus a lack of subsequent survival status data. A total of 11,346 participants were included in this study (Fig. 1).

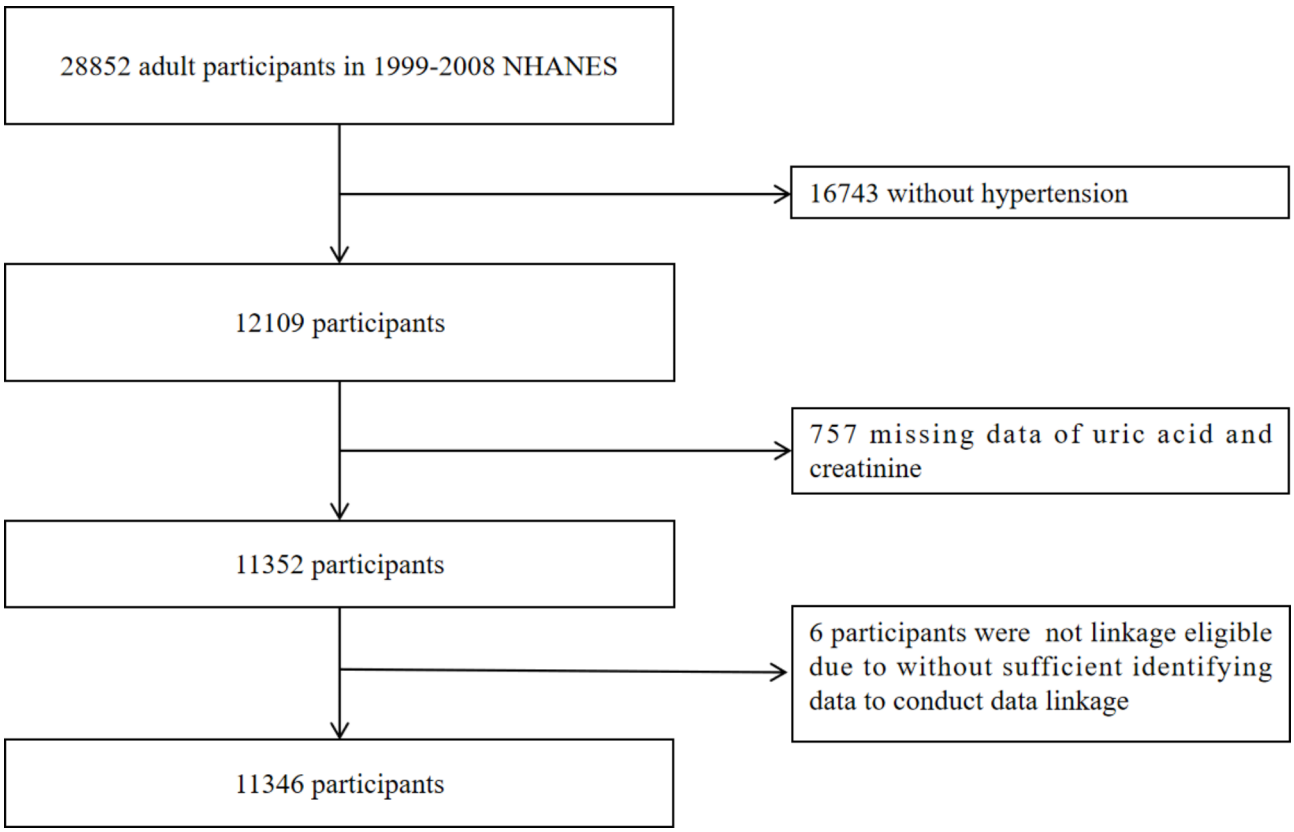

Fig. 1. Flowchart of participant selection.

Survival status and follow-up duration

The NCHS, led by the U.S. government, links data from various surveys with death certificate records from the NDI to investigate the relationships between various health factors and mortality. All the data were obtained from publicly available documents on the NCHS website (https://www.cdc.gov/nchs/nhanes/index.htm).

Definition of hypertension

The participants were diagnosed with hypertension if they met any of the following three criteria: (1) systolic blood pressure (SBP) ≥ 130 mmHg or diastolic blood pressure (DBP) ≥ 80 mmHg[28]; (2) currently taking antihypertensive drugs. All blood pressure measurements were performed at mobile examination centres.

Baseline characteristics

Baseline data were obtained through computer-assisted personal interviews, questionnaires, and blood tests. These included sex, age, race, educational level, the ratio of family income to poverty, SBP, DBP, body mass index (BMI), smoking status, alcohol consumption status, hypercholesterolemia status, diabetes status, heart failure status, coronary artery disease status, stroke status, alanine aminotransferase (ALT) levels, aspartate aminotransferase (AST) levels, albumin levels, Cr levels, and UA levels. Smokers were defined as participants who had smoked at least 100 cigarettes in their lifetimes. Drinkers were defined as participants who had consumed at least 12 alcoholic beverages in the past 12 months. Hypertension, hypercholesterolemia, diabetes, heart failure, and stroke status were defined on the basis of self-reported diagnosis by a health care provider. Biochemical markers were measured via serum testing. The estimated glomerular filtration rate (eGFR) was calculated with a modified renal disease equation[29]. Renal insufficiency was defined as an eGFR < 60 (ml/min/1.73 m^2). The UCR was calculated by dividing UA by Cr and expressed as a continuous variable.

Statistical analysis

The weight of the sample was considered in this study. Categorical and continuous data are represented by survey-weighted percentages (95% *CI*s) and survey-weighted means (95% *CI*s), respectively.

In the present study, we applied spline smoothing to explore the linear relationship between the UCR and all-cause mortality in patients with hypertension. Threshold saturation effect modelling and log-likelihood ratio tests based on logistic regression were used to analyse the inflection point of the effect of UCR on all-cause mortality. A competing risk model based on Cox regression was used to examine the associations between the UCR and cardiovascular mortality, renal disease mortality, and mortality from other causes. Collinearity screening of the covariates was performed before logistic regression analysis, and the results indicated that the variance inflation factor of all covariates did not exceed 5. Three logistic regression models were constructed to adjust for confounders. Model 1 was adjusted for age, sex and race. Model 2 was adjusted for age, sex, race, educational level, the ratio of family income to poverty, BMI, smoking status, and alcohol consumption status. Model 3 was additionally adjusted for factors selected on the basis of covariate screening principles, i.e., factors that changed the p value by more than 10% when included in the regression model: age, sex, race, educational level, the ratio of family income to poverty, BMI, smoking status, alcohol consumption status, ALT levels, albumin levels, hypercholesterolemia status, diabetes status, heart failure status, CAD status, stroke status, and renal failure status.

EmpowerXYS 6.0 and StataSE 18.5 were used for data analysis, and *P* values < 0.05 were considered to indicate statistical significance.

Results

Baseline characteristics

A total of 11,346 adult patients with hypertension were included in the study. The basic characteristics of the participants are shown in Table 1.

Relationship between the UCR and long-term all-cause mortality risk in patients with hypertension

Spline smoothing revealed one inflection point in the relationship between the UCR and all-cause mortality in patients with hypertension (Fig. 2). Therefore, threshold saturation effect analysis was used to explore whether the nonlinear relationship was statistically significant. The results revealed that when the UCR was less than 4.3, the mortality risk gradually decreased with increasing UCR. When the UCR was greater than or equal to 4.3, the mortality risk gradually increased with increasing UCR. Similar results were obtained for all three models. In Model 3, after adjusting for various confounding factors, the results before and after the inflection point were UCR < 4.3 (*OR* = 0.80, 95% *CI*: 0.68–0.94, *P* = 0.008) and UCR ≥ 4.3 (*OR* = 1.21, 95% *CI*: 1.07–1.36, *P* = 0.004), respectively, with statistically significant differences before and after the inflection point (*P* < 0.001) (Table 2).

Subgroup analysis and interaction testing

Subgroup and interaction analyses were performed to verify whether the results differed when the different diseases were combined. The results suggest that no effect modifiers were present (Fig. 3).

Relationship between the UCR and mortality risk in patients with hypertension

A competing risk model was used to investigate the effect of the UCR on mortality. When the UCR was < 4.3, the risk of death from kidney disease and other causes gradually decreased with increasing UCR (*SHR* = 0.44, 95% *CI*: 0.28–0.69, *P* < 0.001) (*SHR* = 0.87, 95% *CI*: 0.77–0.98, *P* = 0.024). When the UCR was ≥ 4.3, the risk of death

Survey-weighted mean (95% CI)/survey-weighted percentage (95% CI)	Total	UCR < 4.3	UCR ≥ 4.3	
	11,346	5,748	5,598	*P*
Time, months	166.3 (164.4, 168.1)	158.9 (156.9, 160.9)	173.3 (171.0, 175.5)	< 0.001
SBP	135.2 (134.7, 135.7)	136.1 (135.5, 136.7)	134.4 (133.7, 135.0)	< 0.001
DBP	76.2 (75.7, 76.7)	75.2 (74.6, 75.9)	77.1 (76.5, 77.6)	< 0.001
Age, years	53.8 (53.2, 54.5)	56.1 (55.3, 56.9)	51.7 (51.0, 52.3)	< 0.001
Sex				0.007
Male	52.8 (51.7, 53.9)	54.4 (52.7, 56.0)	51.3 (49.8, 52.9)	
Female	47.2 (46.1, 48.3)	45.6 (44.0, 47.3)	48.7 (47.1, 50.2)	
Race				< 0.001
Mexican American	5.3 (4.2, 6.6)	4.0 (3.1, 5.2)	6.4 (5.2, 8.0)	
Other Hispanic	4.0 (2.8, 5.6)	3.2 (2.3, 4.4)	4.8 (3.2, 7.0)	
Non-Hispanic White	74.1 (71.3, 76.8)	75.9 (73.2, 78.5)	72.4 (69.2, 75.4)	
Non-Hispanic Black	11.9 (10.0, 14.0)	13.5 (11.5, 15.9)	10.3 (8.5, 12.4)	
Other Race	4.7 (3.9, 5.7)	3.3 (2.7, 4.1)	6.1 (4.9, 7.5)	
Education level				0.031
High school	22.0 (20.7, 23.3)	20.8 (19.1, 22.6)	23.1 (21.7, 24.5)	
High school graduate or general equivalency diploma	27.8 (26.5, 29.1)	27.4 (25.8, 29.0)	28.1 (26.2, 30.1)	
High school	50.2 (48.2, 52.2)	51.7 (49.3, 54.1)	48.8 (46.3, 51.2)	
Unknown	0.1 (0.1, 0.1)	0.1 (0.0, 0.1)	0.1 (0.0, 0.1)	
Ratio of family income to poverty				0.018
< 1	10.9 (9.8,12.1)	10.2 (9.1, 11.4)	11.6 (10.2, 13.1)	
1–3	35.3 (33.4, 37.3)	34.5 (32.4, 36.7)	36.1 (33.7, 38.6)	
≥ 3	47.1 (44.7, 49.4)	48.9 (46.3, 51.6)	45.3 (42.6, 48.0)	
Unknown	6.7 (5.8, 7.7)	6.4 (5.5, 7.4)	7.1 (5.8, 8.5)	
BMI				< 0.001
< 30 kg/m^2	47.0 (45.3, 48.6)	50.5 (48.4, 52.7)	43.6 (41.6, 45.6)	
≥ 30 kg/m^2	30.8 (29.2, 32.4)	22.2 (20.6, 23.9)	38.9 (36.8, 41.1)	
Unknown	22.3 (20.1, 24.5)	27.3 (24.5, 30.2)	17.5 (15.5, 19.6)	
Smoking				0.033
No	49.8 (48.4, 51.1)	51.1 (49.3, 52.9)	48.5 (46.7, 50.3)	
Yes	50.2 (48.9, 51.6)	48.9 (47.1, 50.7)	51.5 (49.7, 53.3)	
Alcohol use				< 0.001
No	33.0 (31.1, 35.0)	34.6 (32.3, 37.1)	31.5 (29.7, 33.5)	
Yes	67.0 (65.0, 68.9)	65.4 (62.9, 67.7)	68.5 (66.5, 70.3)	
Hypercholesterolemia				0.152
No	61.6 (60.3, 63.0)	60.9 (59.1, 62.6)	62.4 (60.7, 64.0)	
Yes	38.4 (37.0, 39.7)	39.1 (37.4, 40.9)	37.6 (36.0, 39.3)	
Diabetes				0.013
No	88.3 (87.5, 89.0)	87.5 (86.4, 88.4)	89.0 (88.0, 90.0)	
Yes	11.7 (11.0, 12.5)	12.5 (11.6, 13.6)	11.0 (10.0, 12.0)	
Heart failure				0.006
No	96.2 (95.8, 96.6)	95.7 (95.0, 96.2)	96.7 (96.2, 97.2)	
Yes	3.8 (3.4, 4.2)	4.3 (3.8, 5.0)	3.3 (2.8, 3.8)	
CAD				< 0.001
No	90.2 (89.2, 91.0)	89.0 (87.9, 90.1)	91.3 (90.1, 92.3)	
Yes	9.8 (9.0, 10.8)	11.0 (9.9, 12.1)	8.7 (7.7, 9.9)	
Stroke				< 0.001
No	95.6 (95.1, 96.1)	94.6 (93.8, 95.3)	96.6 (95.9, 97.1)	
Yes	4.4 (3.9, 4.9)	5.4 (4.7, 6.2)	3.4 (2.9, 4.1)	
eGFR < 60				< 0.001
No	87.3 (86.2, 88.4)	78.1 (76.3, 79.8)	96.1 (95.3, 96.7)	
Yes	12.7 (11.6, 13.8)	21.9 (20.2, 23.7)	3.9 (3.3, 4.7)	
eGFR	85.4 (84.4, 86.4)	74.2 (73.3, 75.2)	96.0 (94.9, 97.1)	< 0.001
ALT, U/L	27.7 (26.9, 28.4)	25.4 (24.2, 26.7)	29.8 (29.1, 30.5)	< 0.001
AST, U/L	26.4 (25.9, 26.8)	25.1 (24.8, 25.5)	27.5 (26.9, 28.2)	< 0.001
Albumin, g/L	4.3 (4.3, 4.3)	4.3 (4.2, 4.3)	4.3 (4.3, 4.3)	< 0.001
Continued				

Survey-weighted mean (95% CI)/survey-weighted percentage (95% CI)	Total	UCR < 4.3	UCR ≥ 4.3	
	11,346	5,748	5,598	*P*
Uric Acid, umol/L	342.2 (340.0, 344.5)	306.4 (303.5, 309.3)	376.3 (373.3, 379.2)	< 0.001
Creatinine, umol/L	81.9 (80.9, 82.9)	93.0 (91.5, 94.5)	71.3 (70.7, 71.9)	< 0.001
UCR	4.4 (4.4, 4.5)	3.5 (3.4, 3.5)	5.4 (5.3, 5.4)	< 0.001

Table 1. Basic characteristics of the participants (weighted) ($n = 11,346$).

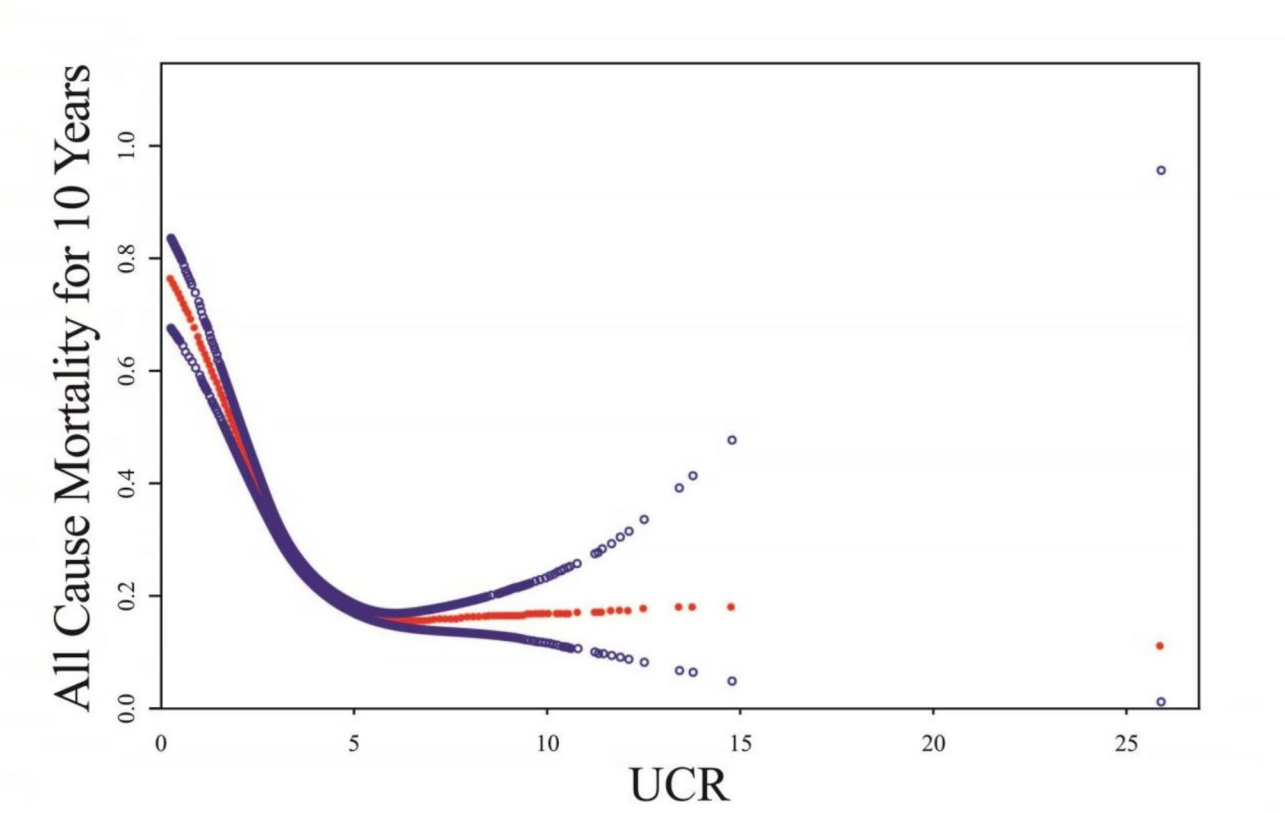

Fig. 2. Spline interpolation between the UCR and all-cause mortality in patients with hypertension.

	Model 1	Model 2	Model 3
	OR (95% CI) *P*	OR (95% CI) *P*	OR (95% CI) *P*
UCR < 4.3	0.66 (0.58, 0.74) < 0.001	0.66 (0.56, 0.78) < 0.001	0.80 (0.68, 0.94) 0.008
UCR ≥ 4.3	1.21 (1.13, 1.30) < 0.001	1.17 (1.04, 1.32) 0.011	1.21 (1.07, 1.36) 0.004
P for Logarithmic likelihood ratio test	< 0.001	< 0.001	< 0.001

Table 2. Threshold saturation analysis between the UCR and all-cause mortality risk in patients with hypertension (weighted). Model 1: Adjusted for age, sex and race. Model 2: Adjusted for age, sex, race, education level, the ratio of family income to poverty, BMI, smoking status, and alcohol consumption status. Model 3: Adjusted for age, sex, race, education level, the ratio of family income to poverty, BMI, smoking status, alcohol consumption status, ALT levels, albumin levels, hypercholesterolemia status, diabetes status, heart failure status, CAD status, stroke status, and renal failure status.

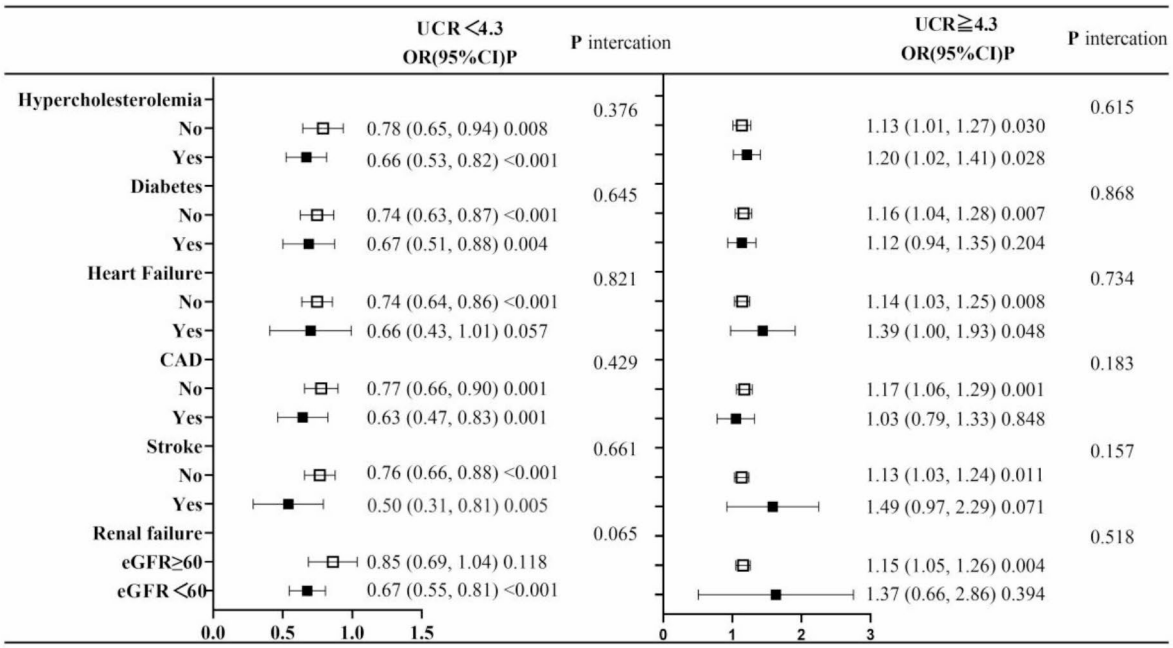

Fig. 3. Subgroup analysis and interaction testing. Adjusted for age, sex, race, education level, the ratio of family income to poverty, BMI, smoking status, alcohol consumption status, ALT levels, albumin levels, hypercholesterolemia status, diabetes status, heart failure status, CAD status, stroke status, and renal failure status in addition to grouping covariate confounding factors.

	Death from cardiovascular disease	Death from kidney disease	Death from all other cause
	SHR (95% CI) *P*	SHR (95% CI) *P*	SHR (95% CI) *P*
UCR < 4.3	0.91 (0.77, 1.08) 0.267	0.44 (0.28, 0.69) < 0.001	0.87 (0.77, 0.98) 0.024
UCR ≥ 4.3	1.06 (0.94, 1.20) 0.316	1.15 (0.81, 1.65) 0.432	1.09 (1.01, 1.18) 0.020

Table 3. Competing risk models for the relationships between the UCR and cardiovascular disease and kidney disease mortality. Adjusted for age, sex, race, education level, the ratio of family income to poverty, BMI, smoking status, alcohol consumption status, ALT levels, albumin levels, hypercholesterolemia status, diabetes status, heart failure status, CAD status, stroke status, and renal failure status.

from kidney disease and other causes gradually increased with increasing UCR (*SHR* = 1.15, 95% *CI*: 0.81–1.65, *P* = 0.432) (*SHR* = 1.09, 95% *CI*: 1.01–1.18, *P* = 0.020), although the *P* value did not change significantly (Table 3).

Discussion

The UCR is the ratio of UA to Cr, which reflects the activity of xanthine oxidase and can represent the oxidation status and metabolism level of the body. Using the NHANES database, this study revealed a nonlinear relationship between the UCR and all-cause mortality in hypertensive patients. With an increasing UCR, the mortality rate of hypertensive patients first decreases but then gradually increases, with an inflection point at 4.3. When the UCR was less than 4.3, the risk of renal disease-related mortality in patients with hypertension gradually decreased with the increase of UCR.

Under hydrophilic conditions, UA scavenges free radicals to inhibit lipid peroxidation at the lipid–water interface[30]. However, in hydrophobic environments, UA loses its antioxidant capacity and becomes a strong pro-oxidant, inducing oxidative stress and leading to the onset of various diseases and an increased risk of mortality[31]. Creatinine is a compound produced during muscle energy production, is metabolized from phosphocreatine and normal renal function filters, and is excreted from the blood, reflecting renal filtration function[32]. The use of the UCR excludes the influence of renal function on UA levels and was initially used to assess the prognostic status of patients with fulminant hepatitis[33]. The UCR has subsequently been shown to be associated with adverse health outcomes[19,34–36]. The association between the UCR and all-cause mortality in the general adult population has been confirmed[37], and this study further explored the relationship between the UCR and all-

cause mortality in hypertensive patients. Through smooth curve fitting and threshold saturation effect analysis, this study revealed a nonlinear relationship between the UCR and mortality in patients with hypertension, with mortality first decreasing but then gradually increasing with an increasing UCR. The observed associations remained statistically significant, even when a variety of potential confounding factors were adjusted for. The findings of this decade-long cohort study suggest that maintaining the UCR below the inflection point of 4.3 in hypertensive patients can reduce mortality risk. One study detected a critical UCR value (UCR = 5.4) via ROC curves and showed that participants whose UCR exceeded this value presented a greater prevalence of cardiovascular events during follow-up[38]. A cohort study involving 2,017 hypertensive patients revealed that an elevated UCR increased the risk of all-cause mortality in patients with hypertension[39]. The findings of this study indicate that in the quartiles with UCR ≥ 4.3 g/dl, all subgroups were identified as risk factors, and the risk of death was elevated in hypertensive patients in the Q4 group. Conversely, in the quartiles with UCR < 4.3 g/dl, all subgroups were identified as protective factors. A high UCR value indicates an elevated risk of all-cause mortality in hypertensive patients. Therefore, in hypertensive populations with a high UCR of more than 4.3, UA-lowering treatment may be beneficial. In the hypertensive population, treatment when the UCR exceeds 4.3 may be of benefit to the patient. The inconsistencies in these results may be related to differences in sample sources and sample sizes. Currently, there is no consensus on the critical inflection point between the UCR and hypertension-related mortality risk. However, the results of this study suggest that hypertensive patients should pay attention to controlling the UCR within a certain range to reduce long-term risk.

When uric acid levels reach a certain threshold, uric acid crystal deposition occurs, leading to the development of gout and affecting the metabolic health of the body[40,41]. Hyperuricaemia (UA > 6.1 mg/dL) has been demonstrated to be associated with the development of a range of diseases[42–44]. The relevant mechanisms can be summarized as follows: increased UA levels induce increased oxidative stress, reduced nitric oxide availability, promotion of local and systemic inflammation, vasoconstriction, vascular smooth muscle cell proliferation, insulin resistance, and metabolic disorders, which have harmful effects on cardiovascular health[45]. Simultaneously, several studies have indicated that hypouricaemia may also have adverse effects[46]. The pathogenesis of hypouricaemia may be attributed to either excessive excretion or insufficient secretion of renal UA, which may be influenced by both genetic and medical factors[47,48]. Previous research has revealed a U-shaped association between UA levels and all-cause mortality, with both elevated and low UA levels increasing the risk of death[49–51].

This study also revealed a nonlinear relationship between the UCR and renal disease mortality and mortality due to other causes in patients with hypertension. The kidney helps regulate blood pressure and is most susceptible to damage under ischaemic conditions. Therefore, sustained hypertension can lead to structural changes within the kidneys, causing injury and dysfunction[52]. In contrast, when the body is in a state of chronically high UA levels, it stimulates the renin–angiotensin system, leading to renal vasoconstriction and potentially elevated blood pressure[53]. Sustained renal vasoconstriction may lead to the development of arteriosclerosis and salt-sensitive hypertension even after hyperuricemia is resolved[54]. An umbrella review revealed UA lowering treatment have been demonstrated to exert a beneficial effect on a number of intermediate traits or biomarkers (e.g., blood pressure, endothelial function, and renal function) that are associated with cardiovascular and renal disease[55,56]. While the potential of UA lowering treatment to reduce the risk of death from the disease remains to be elucidated, UA lowering treatment can still confer benefits to patients, such as the lowering of blood pressure levels, which in turn reduces the risk of an adverse prognosis. Patients with hyperlipidaemia, diabetes, CAD, and renal failure may benefit from controlling the UCR to ≤ 4.3. Therefore, controlling UA levels within a certain range can help reduce the mortality risk in hypertensive patients. These efforts confirmed the stability of the relationship between the UCR and hypertension-related mortality risk. These results provide a reference for clinical interventions at UCR levels to reduce the risk of long-term adverse outcomes in patients with hypertension.

This is the first decade-long cohort study on the correlation between the UCR and hypertension-related mortality risk, establishing the UCR as a novel indicator for predicting adverse outcomes in patients with hypertension. These findings suggest that the UCR is associated not only with all-cause mortality in hypertension patients but also with mortality due to renal disease or other causes, potentially serving as an observational indicator for long-term prognosis in hypertensive patients and supporting early intervention.

However, this study has several limitations. First, the target population originates from the NHANES, which is representative of the United States but may not be directly generalizable to other countries. Second, this study did not include dietary data, which may have influenced UA. In addition, the analysis included a number of confounding factors that were not controlled for or measured, such as antihypertensive, antihyperlipidaemic, uric acid-lowering, and antidiabetic medications.

Conclusions

Our study revealed that, in patients with hypertension, there is a nonlinear relationship between the UCR and all-cause mortality, with an initial decrease followed by an increase and an inflection point at 4.3. These findings suggest that the UCR has value in predicting the long-term prognosis of patients with hypertension, providing evidence-based medicine for preventing long-term complications of hypertension.

Adjusted for age, sex, race, education level, the ratio of family income to poverty, BMI, smoking status, alcohol consumption status, ALT levels, albumin levels, hypercholesterolemia status, diabetes status, heart failure status, CAD status, stroke status, and renal failure status in addition to grouping covariate confounding factors.

Data availability

The study data were derived from the NHANES and can be obtained online (https://www.cdc.gov/nchs/nhanes/index.htm).

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Author contributions

Y.M.Z. interpreted the data and drafted the manuscript. Y.Y.C. analysed the data, interpreted the data and prepared the figures. J.L. designed the present study, analysed the data and interpreted the data. L.C. interpreted the data and revised the article critically for important intellectual content. All the authors read and approved the final manuscript.

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