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U-shaped association of body roundness index with all-cause and cardiovascular mortality in individuals with chronic kidney disease

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ABSTRACT

Purpose: This study aimed to investigate the association between body roundness index (BRI) and deaths from all causes and cardiovascular disease (CVD) in participants with chronic kidney disease (CKD).

Materials and methods: The data was sourced from the National Health and Nutrition Examination Survey (NHANES) 1999–2018. Cox proportional hazards regression along with restricted cubic splines were applied to assess the associations of BRI with deaths from all causes and CVD in individuals with CKD.

Results: In total, 8,000 individuals were included in the final analysis. In participants with CKD, there was a nonlinear U-shaped association between BRI and deaths from all causes and CVD, with inflection points at 6.29 and 6.04. When BRI was below the inflection points, each unit increment of BRI correlated with a 11% reduction in deaths from all causes [HR: 0.89 (0.84–0.94), $p<0.001$] and a 15% reduction in deaths from CVD [HR: 0.85 (0.75–0.95), $p=0.006$)]. Conversely, when BRI surpassed the inflection points, each unit increment of BRI was linked to a 9% rise in deaths from all causes [HR: 1.09 (1.03–1.14), $p=0.001$] and a 10% rise in deaths from CVD [HR: 1.10 (1.02–1.20), $p=0.019$].

Conclusion: In this nationally representative study, we found a nonlinear U-shaped association between BRI and deaths from all causes and CVD among participants with CKD, with inflection points at 6.29 and 6.04. Further research is required to establish the optimal range of BRI as a measure of visceral fat deposition.

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Introduction

Chronic kidney disease (CKD), characterized by irreversible injury and progressive decline of kidney function, is a global health concern affecting a growing number of individuals [1]. CKD is estimated to impacting over 10% of the global population, amounting to more than 800 million individuals [2,3]. Moreover, even with the optimal current administration, the onset of CKD is frequently accompanied by heightened risks of cardiovascular complications and deaths [4]. Hence, early prevention and effective management are crucial for patients with CKD to improve adverse health outcomes.

Evidence suggests that obesity is a significant risk factor for CKD and cardiovascular disease (CVD) [5–7], as well as for both all-cause and cause-specific mortality [8,9]. As research deepens, visceral obesity has been found to be a stronger predictor of health impairments compared to general obesity, such as renal function decline [10], cancer [11], and mortality [12]. Body mass index (BMI) is a widely used anthropometric index to evaluate obesity, but it fails to distinguish between lean mass and fat mass. BMI cannot

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differentiate people with similar BMI but varying body fat levels. Body roundness index (BRI) is a newly developed measurement index of obesity that integrates height and waist circumference (WC) to predict proportion of visceral fat [13]. BRI has been demonstrated more effective than BMI in predicting CVD [14] and metabolic syndrome [15]. In addition, a U-shaped association has been observed between BRI and death from all causes [16]. As obesity contributes to kidney damage, the prevalence of abdominal obesity in patients with CKD reaches up to 70% and continues to rise in the United States population [17]. Meanwhile, protein-energy wasting, characterized by muscle loss and metabolic imbalance, affects up to 54% of patients with advanced CKD [18] and 25% of those with obesity [19], suggesting the inadequacy of BMI in capturing body composition in this population. Therefore, BRI, which better reflects visceral adiposity, may offer improved risk assessment. However, in patients with CKD, the relationship between BRI and mortality remains unclear and the optimal BRI level for patients with CKD is also unknown.

Thus, this cohort study aimed to explore the association between BRI and deaths from all causes and CVD in participants with CKD in a nationally representative population.

Materials and methods

Study design

The data was sourced from National Health and Nutrition Examination Survey (NHANES) [20], which is a multistage and nationally representative study that evaluates the nutrition and physical health of the non-institutionalized US population. The study procedure was approved by the National Center for Health Statistics Ethics Review Board and written informed consent for participation was obtained from all individuals. The procedures adhered to the ethical standards outlined in the Helsinki Declaration.

Initially, 101,316 participants were included across ten NHANES cycles spanning 1999 to 2018. The exclusion criteria included the following: (1) individuals under 18 years old ($n=42,112$); (2) missing data on serum creatinine or urinary albumin-creatinine ratio ($n=7,393$); (3) those without CKD ($n=42,773$); (4) missing BRI data ($n=671$); (5) not eligible for mortality linkage ($n=7$); and (6) missing values of key covariates ($n=360$). In total, 8,000 patients were included in the final analysis (Figure 1).

Definitions

Urinary albumin creatinine ratio (ACR) and serum creatinine, used to define CKD, were extracted from the laboratory data. Estimated glomerular filtration rate (eGFR) levels were determined using the CKD-EPI equation [21]. CKD was diagnosed as an ACR ≥30mg/g or an eGFR < 60 mL/min/1.73 m^2 [22].

Height (cm) and WC (cm) were obtained from the examination data of 1999–2018 cycles. As reported previously, the following equation was used to calculate BRI: $\text{BRI} = 364.2-365.5\times[1-(\text{WC}/2\pi)^2/(0.5 \times \text{height})^2]^{1/2}$ [13]. Patients were categorized into 3 groups according to BRI tertiles: Tertile 1, ≤4.95; Tertile 2, (4.96–6.76); Tertile 3, > 6.76.

Determination of mortality

The determination of deaths was used the National Death Index mortality data until 31 December 2019 [23]. Death causes were determined using the 10th revision of the International Classification of Diseases. CVD mortality was defined as deaths from heart disease (I00-I09, I11, I13, I20-I51) and cerebrovascular diseases (I60-I69). Follow-up time was calculated from the baseline examination date until death or the end of the mortality period.

Covariates

The covariates of this study include demographic information (age, sex, ethnicity, education level and family income-poverty ratio), lifestyle (smoking and drinking habits and physical activity), medical history (hypertension, diabetes mellitus, dyslipidemia, and CVD), and laboratory tests (ACR, eGFR and uric acid).

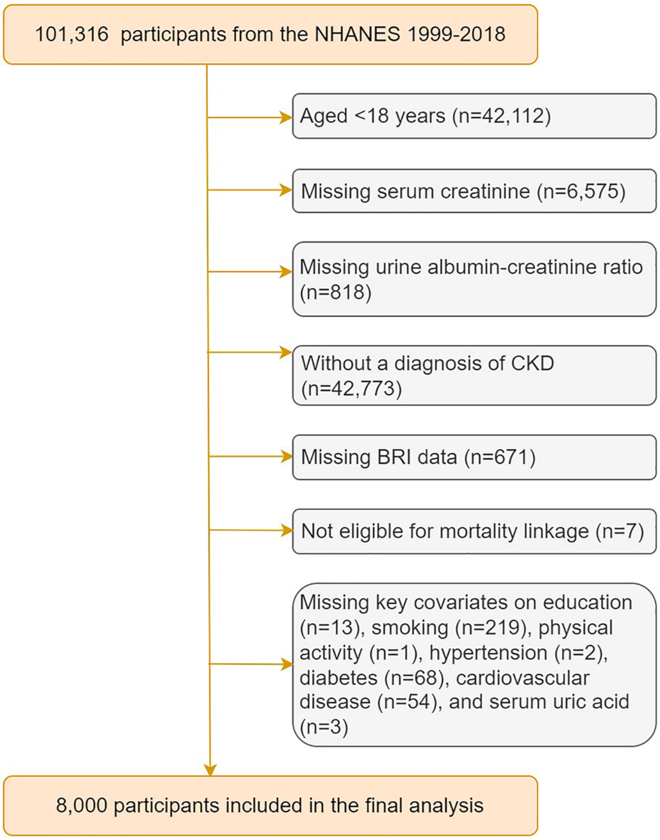

Figure 1. Flow chart of identification of study population. There were 8,000 participants included in the present analysis. CKD, chronic kidney disease; NHANES, National Health and Nutrition Examination Survey; BRI, body mass index.

Statistical analyses

The weighted analysis was performed using sample weights due to the stratified and multistage design of NHANES. Categorical variables are shown as counts and percentages, while continuous variables are reported as means ± standard error (SE). Group comparisons were performed using chi-square tests for categorical variables and one-way ANOVA for continuous variables. Weighted Cox proportional hazards regression was applied to evaluate the association between BRI and deaths from all causes and CVD in patients with CKD, adjusting for potential confounders across three models.

Restricted cubic splines with 4 knots were employed to model the dose-response relationships between BRI and mortality, enabling the assessment of potential non-linear associations. If a non-linear association were detected, a recursive algorithm would be utilized to determine the inflection points. Additionally, piecewise Cox proportional hazards regression would be employed to examine the threshold effect.

Subsequently, subgroup analyses were performed to examine possible differences of associations between BRI and mortality across the categories: age, sex, ACR and eGFR groups. Sensitivity analyses were performed to assess the stability of these associations. A two-sided P-value of less than 0.05 was deemed statistically significant. All analyses were performed using R software (version 4.4.2).

Results

Characteristics of study participants

The weighted distribution of individuals' characteristics by BRI tertiles is presented in Table 1. Of the 8,000 patients with CKD from NHANES 1999–2018, the mean age was 60.40 ± 0.30 years, and the average ACR and eGFR were 177.90 ± 8.51 mg/g, 73.36 ± 0.46 mL/min/1.73 m^2, respectively. Patients in the higher tertile of BRI tended to be older and former smokers, have higher blood pressure, BMI, triglycerides levels, uric acid levels and ACR levels, and suffer from hypertension, diabetes mellitus, dyslipidemia, and CVD (all $p < 0.05$). Furthermore, individuals with higher BRI were less likely to be current drinkers or engage in physical activity (all $p < 0.001$). They were also more likely to have a lower education level, a reduced family income-to-poverty ratio, lower HDL-C levels, and decreased eGFR (all $p < 0.001$). In addition, the distribution of CKD stages varied significantly across BRI tertiles ($p < 0.001$), with a higher proportion of advanced CKD stages (G3–G5) observed in the higher BRI groups.

Association of BRI with all-cause and CVD mortality

The median follow-up time of this prospective study was 10.2 years. Over a follow-up period totaling 64,642 person-years, 2,980 deaths from all causes were recorded, including 1,082 cardiovascular deaths, among individuals with CKD from NHANES 1999–2018.

Table 1. Characteristics of participants categorized by tertiles of body roundness index.

		Tertiles of body roundness index			
Variables	Total	Tertile1 (≤ 4.95)	Tertile 2 (4.96–6.76)	Tertile 3 (> 6.76)	*P* value
Participants	8000	2667	2666	2667	
Age, years	60.40 ± 0.30	56.97 ± 0.52	63.25 ± 0.44	61.41 ± 0.39	<0.001
Sex, %					<0.001
Women	4168 (56.82)	1377 (60.35)	1217 (48.75)	1574 (60.65)	
Men	3832 (43.18)	1290 (39.65)	1449 (51.25)	1093 (39.35)	
Ethnicity, %					<0.001
Non-hispanic white	3951 (70.03)	1355 (71.08)	1309 (69.39)	1287 (69.50)	
Non-hispanic black	1690 (11.71)	602 (11.72)	505 (10.58)	583 (12.76)	
Mexican American	1215 (6.87)	297 (5.17)	449 (7.72)	469 (7.89)	
Other	1144 (11.39)	413 (12.02)	403 (12.31)	328 (9.85)	
Education, %					<0.001
Less than high school	2724 (24.11)	787 (20.23)	950 (25.60)	987 (26.89)	
High school	1927 (26.15)	656 (26.20)	613 (24.40)	658 (27.75)	
More than high school	3349 (49.74)	1224 (53.57)	1103 (50.01)	1022 (45.37)	
Smoking, %					<0.001
Never	3989 (50.30)	1341 (52.76)	1348 (49.35)	1300 (48.55)	
Former	2664 (32.69)	731 (25.51)	944 (35.79)	989 (37.51)	
Now	1347 (17.01)	595 (21.74)	374 (14.86)	378 (13.95)	
Drinking, %					<0.001
Never	1280 (14.17)	390 (12.70)	433 (14.54)	457 (15.40)	
Former	2192 (24.10)	636 (20.28)	732 (24.52)	824 (27.82)	
Now	4078 (56.93)	1481 (61.78)	1340 (55.93)	1257 (52.66)	
Missing data	450 (4.80)	160 (5.25)	161 (5.01)	129 (4.12)	
Physical activity, %	4919 (66.59)	1799 (73.25)	1653 (67.81)	1467 (58.26)	<0.001
Family income-poverty ratio, %					<0.001
≤1	1580 (14.81)	505 (14.56)	487 (12.82)	588 (16.96)	
1-3	3492 (40.16)	1118 (37.24)	1141 (39.49)	1233 (43.94)	
>3	2209 (37.23)	797 (39.83)	799 (39.93)	613 (31.89)	
Missing data	719 (7.80)	247 (8.37)	239 (7.77)	233 (7.22)	
Hypertension, %	5707 (66.22)	1575 (51.49)	1985 (69.44)	2147 (79.03)	<0.001
Diabetes mellitus, %	3040 (32.42)	609 (16.62)	995 (31.70)	1436 (50.13)	<0.001
Dyslipidemia, %	6637 (83.00)	1970 (73.48)	2303 (87.31)	2364 (89.17)	<0.001
Cardiovascular disease, %	2120 (23.13)	585 (18.14)	721 (22.86)	814 (28.75)	<0.001
BMI, Kg/m^2	29.78 ± 0.13	23.53 ± 0.09	29.12 ± 0.08	37.17 ± 0.17	<0.001
Blood pressure, mmHg					
Systolic	132.63 ± 0.35	129.29 ± 0.64	133.37 ± 0.56	135.58 ± 0.55	<0.001
Diastolic	70.29 ± 0.25	69.83 ± 0.38	69.98 ± 0.38	71.09 ± 0.39	0.024
TC, mmol/L	5.11 ± 0.02	5.13 ± 0.03	5.14 ± 0.03	5.05 ± 0.03	0.073
TG, mmol/L	1.75 ± 0.04	1.40 ± 0.05	1.89 ± 0.09	1.99 ± 0.05	<0.001
HDL-C, mmol/L	1.36 ± 0.01	1.54 ± 0.01	1.30 ± 0.01	1.23 ± 0.01	<0.001
LDL-C, mmol/L	2.89 ± 0.02	2.92 ± 0.03	2.93 ± 0.04	2.84 ± 0.04	0.145
Uric acid, mmol/L	353.81 ± 1.56	323.58 ± 2.64	361.31 ± 2.44	379.26 ± 2.42	<0.001
ACR, mg/g	177.90 ± 8.51	153.89 ± 11.12	158.82 ± 11.86	221.82 ± 17.03	0.002
eGFR, mL/ (min*1.73 m^2)	73.36 ± 0.46	76.94 ± 0.73	70.40 ± 0.75	72.32 ± 0.72	<0.001
CKD Stage, %					<0.001
G1	2290 (31.63)	879 (36.62)	649 (27.04)	762 (30.60)	
G2	1847 (21.87)	592 (21.00)	623 (22.07)	632 (22.60)	
G3	3497 (43.03)	1089 (39.51)	1267 (47.19)	1141 (42.89)	
G4-G5	366 (3.47)	107 (2.86)	127 (3.70)	132 (3.91)	

BMI, body mass index; TC, total cholesterol; TG, triglycerides; HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate; CKD, chronic kidney disease.

Table 2 presents hazard ratios (HRs) and 95% confidence intervals (CIs) for the association of BRI with deaths from all causes and CVD. After adjustment for age and sex in Model 1, participants in Tertile 3 of BRI exhibited a markedly higher risk of deaths from all causes compared to those in Tertile 2 [HR: 1.31 (1.17–1.48), $p < 0.001$], while Tertile 1 showed a non-significant trend toward increased risk [HR: 1.10 (0.99–1.22), $p = 0.068$]. After adjusting for variables including age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking habits, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR, and uric acid in Model 3, participants in Tertile 1 had a 20% higher risk of deaths from all causes [HR: 1.20 (1.09–1.33), $p < 0.001$], while those in Tertile 3 showed a 6% increase in risk [HR: 1.06 (0.95–1.19), $p = 0.276$], although the result for Tertile 3 did not reach statistical significance. Analyzing BRI as a continuous variable revealed no linear association with deaths from all causes [HR: 1.00 (0.97–1.03), $p = 0.968$).

The risk of CVD mortality was 13% higher in Tertile 1 [HR: 1.13 (0.95–1.34), $p = 0.159$] and 1% higher in Tertile 3 [HR: 1.01 (0.85–1.20), $p = 0.916$] compared to Tertile 2 in Model 3. However, neither association reached statistical significance. Analyzing BRI as a continuous variable also revealed no significant linear relationship with CVD mortality [HR: 1.00 (0.96–1.05), $p = 0.930$]. These findings raise the possibility of nonlinear associations between BRI and mortality outcomes.

Assessment of potential non-linear associations

The fully adjusted restricted cubic splines show the nonlinear U-shaped association of BRI with deaths from all causes and CVD (Figure 2, all *P*-overall < 0.001, all *P*-nonlinear <0.001). The BRI inflection points were identified as 6.29 for all-cause mortality and 6.04 for CVD mortality.

The piecewise Cox proportional hazards regression was conducted to examine the threshold effect (Table 3). When BRI was below the inflection points, each unit increment of BRI correlated with a 11% reduction in deaths from all causes [HR: 0.89 (0.84–0.94), $p < 0.001$] and a 15% reduction in deaths from CVD [HR: 0.85 (0.75–0.95), $p = 0.006$)]. Conversely, when BRI surpassed the inflection points, each unit increment of BRI was linked to a 9% rise in deaths from all causes [HR: 1.09 (1.03–1.14), $p = 0.001$] and a 10% rise in deaths from CVD [HR: 1.10 (1.02–1.20), $p = 0.019$].

Subgroup analyses of the association

Table 4 summarizes associations between BRI and mortality across different subgroups. The impact of BRI on deaths from all causes and CVD showed consistency across ACR and eGFR subgroups, with all interaction *P*-values >0.05. However, these associations varied by sex and age. In men and patients under 65 years of age, lower BRI demonstrated a stronger negative association with deaths from all causes and

Table 2. Association of body roundness index with mortality in patients with CKD from the NHANES 1999–2018.

	No. of participants	No. of deaths	Model 1		Model 2		Model 3	
			HR (95% CI)	*P* value	HR (95% CI)	*P* value	HR (95% CI)	*P* value
All-cause mortality								
Tertile 1	2667	989	1.10 (0.99, 1.22)	0.068	1.12 (1.01, 1.24)	**0.030**	1.20 (1.09, 1.33)	**<0.001**
Tertile 2	2666	1057	Reference	–	Reference	–	Reference	–
Tertile 3	2667	934	1.31 (1.17, 1.48)	**<0.001**	1.20 (1.07, 1.34)	**0.002**	1.06 (0.95,1.19)	0.276
Per 1 unit increment	8000	2980	1.06 (1.04, 1.09)	**<0.001**	1.04 (1.01, 1.06)	**0.001**	1.00 (0.97, 1.03)	0.968
CVD mortality								
Tertile 1	2010	341	1.06 (0.90, 1.24)	0.505	1.08 (0.92, 1.27)	0.351	1.13 (0.95, 1.34)	0.159
Tertile 2	2019	401	Reference	–	Reference	–	Reference	–
Tertile 3	2073	340	1.34 (1.10, 1.63)	**0.003**	1.20 (0.99, 1.46)	0.061	1.01 (0.85, 1.20)	0.916
Per 1 unit increment	6102	1082	1.08 (1.04, 1.13)	**<0.001**	1.05 (1.01, 1.10)	**0.015**	1.00 (0.96, 1.05)	0.930

Model 1: adjusts for age and sex.
Model 2: model 1 + ethnicity, education, family income-poverty ratio, smoking and drinking habits, and physical activity.
Model 3: model 2 + hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid.
CKD, chronic kidney disease; NHANES, National Health and Nutrition Examination Survey; HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate.

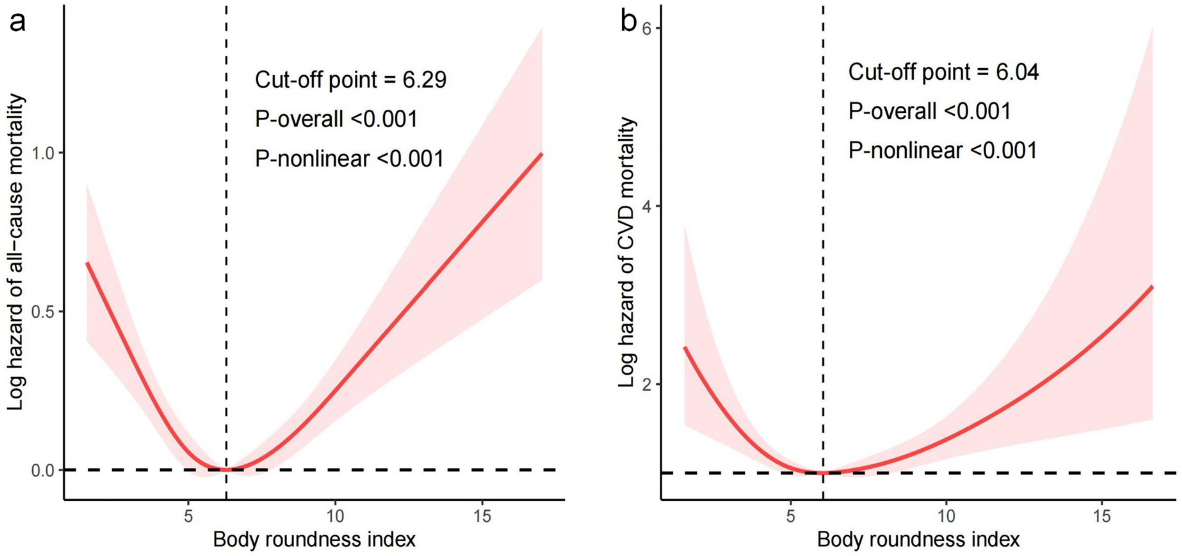

Figure 2. The U-shaped association of BRI with all-cause and CVD mortality. The solid line represents the adjusted log hazards, while the shaded areas indicate the 95% CIs. The vertical dashed line marks the inflection points of BRI. Adjustment for age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking status, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid. BRI, body mass index; CVD, cardiovascular disease; CI, confidence interval; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate.

Table 3. Threshold effect analysis of body roundness index on mortality in patients with CKD from the NHANES 1999–2018.

Body roundness index	HR (95% CI)	*P* value
All-cause mortality		
Weighted cox regression model	1.00 (0.97, 1.03)	0.968
Weighted two-piecewise cox regression model, Cutoff point, 6.29		
<6.29	0.89 (0.84, 0.94)	**<0.001**
≥6.29	1.09 (1.03, 1.14)	**0.001**
CVD mortality		
Weighted Cox regression model	1.00 (0.96, 1.05)	0.930
Weighted two-piecewise cox regression model, Cutoff point, 6.04		
<6.04	0.85 (0.75, 0.95)	**0.006**
≥6.04	1.10 (1.02, 1.20)	**0.019**

Adjusted for age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking habits, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid.
CKD, chronic kidney disease; NHANES, National Health and Nutrition Examination Survey; HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate.

CVD, while higher BRI showed a stronger positive association compared to women and the elderly (all *P* for interaction < 0.05). Similarly, dose-response analyses across age, sex, ACR, and eGFR subgroups indicated stronger associations between BRI and deaths from all causes (Figure 3) and CVD (Figure 4) in men and patients under 65 years of age.

We then performed a sensitivity analysis to assess the stability of the association between BRI and mortality. As shown by Table S1, after excluding 433 deaths that occurred within the initial 2 years, the associations between BRI and all-cause and CVD mortality remained broadly consistent with the primary findings, although the inverse association in the lower BRI range for CVD mortality was attenuated and did not reach statistical significance. In addition, the association between BRI and death persisted after we excluded 58 deaths caused by accidents (Table S2).

We additionally compared the predictive performance of BRI and BMI for 10-year all-cause and CVD mortality. As shown in Figure S1, the two indices demonstrated nearly identical AUCs across both outcomes, suggesting comparable discrimination ability in this population with CKD.

Table 4. Subgroup analyses of the association of body roundness index with mortality in patients with CKD from the NHANES 1999–2018.

Subgroups	Tertile1	Tertile2	Tertile 3	P for interaction
All-cause mortality				
Age				**<0.001**
<65 years	1.29 (0.93, 1.80)	Reference	**1.44 (1.01, 2.05)**	
≥65 years	**1.13 (1.00, 1.27)**	Reference	**0.86 (0.76, 0.97)**	
Sex				**0.006**
Men	**1.22 (1.07, 1.39)**	Reference	**1.31 (1.11, 1.56)**	
Women	**1.18 (1.03, 1.36)**	Reference	0.91 (0.78, 1.06)	
ACR				0.904
<30 mg/g	1.17 (1.00, 1.37)	Reference	1.15 (0.97, 1.35)	
≥30 mg/g	**1.26 (1.08, 1.46)**	Reference	1.02 (0.88, 1.19)	
eGFR				0.831
<60 mL/ (min*1.73 m^2)	**1.21 (1.06, 1.37)**	Reference	1.11 (0.98, 1.25)	
≥60 mL/ (min*1.73 m^2)	1.19 (0.97, 1.44)	Reference	0.98 (0.81, 1.19)	
CVD mortality				
Age				**0.002**
<65 years	1.30 (0.78, 2.17)	Reference	1.67 (0.99,2.82)	
≥65years	1.04 (0.85, 1.26)	Reference	**0.81 (0.66, 0.99)**	
Sex				**0.016**
Men	1.18 (0.92, 1.51)	Reference	**1.37 (1.03, 1.81)**	
Women	1.11 (0.87, 1.42)	Reference	0.84 (0.67, 1.07)	
ACR				0.579
<30 mg/g	1.06 (0.79, 1.41)	Reference	0.99 (0.75, 1.32)	
≥30 mg/g	**1.27 (1.01, 1.60)**	Reference	1.08 (0.85, 1.37)	
eGFR				0.845
<60 mL/ (min*1.73 m^2)	1.17 (0.93, 1.47)	Reference	1.05 (0.85, 1.29)	
≥60 mL/ (min*1.73 m^2)	1.10 (0.82, 1.46)	Reference	0.95 (0.67, 1.33)	

Adjusted for all listed covariates except the stratification variable in each subgroup model: age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking habits, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid.
CKD, chronic kidney disease; NHANES, National Health and Nutrition Examination Survey; HR, hazard ratio; CI, confidence interval; CVD, cardiovascular disease; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate.

Discussion

Using the data of NHANES 1999–2018, we assessed the association of BRI with all-cause and CVD mortality in participants with CKD. In this nationally representative study, we found a nonlinear U-shaped association between BRI and deaths from all causes and CVD in participants with CKD, with inflection points at 6.29 and 6.04, respectively. Both low and high BRI levels were related to increased risks of deaths from all causes and mortality in patients with CKD. Moreover, subgroup analyses indicated stronger associations of BRI with mortality in men and participants under 65 years of age.

Obesity, characterized by an excess of body fat, has become increasingly prevalent worldwide [24]. BMI remains the most widely used indicator for assessing obesity due to its simplicity. However, it does not account for fat distribution, especially visceral adiposity, which is more strongly associated with renal and cardiovascular risks [10,11]. This limitation highlights the importance of assessing body fat distribution rather than total body mass. Although imaging techniques such as computed tomography and magnetic resonance imaging can accurately measure visceral fat [25], their cost and, in the case of CT, radiation exposure limit routine use. To address this, alternative anthropometric indices were developed. Among them, BRI, introduced in 2013, is a noninvasive index for estimating total and visceral fat, with values ranging from 1 to 16 [13].

BRI has been linked to a variety of cardiometabolic and renal outcomes. Prior studies have shown that elevated BRI is positively associated with diabetes, hypertension, heart failure, and CKD in general and high-risk populations [26–30]. Moreover, several studies in general and high-risk populations have demonstrated U-shaped associations between BRI and mortality. For instance, a previous NHANES study (1999–2014) and a more recent update covering 1999–2018 both reported U-shaped relationships between BRI and all-cause mortality in the general U.S. population [16,31]. Similar patterns were observed in a large prospective Chinese cohort, where the lowest risk was noted at a BRI of 3.9 [32], and in participants with hyperglycemia, where critical points for all-cause and CVD mortality were 5.54 and 5.21, respectively [33]. Moreover, among U.S. adults with metabolic syndrome, the association remained U-shaped, with a threshold of 6.89 [34]. Our findings in patients with CKD also demonstrated a similar U-shaped pattern,

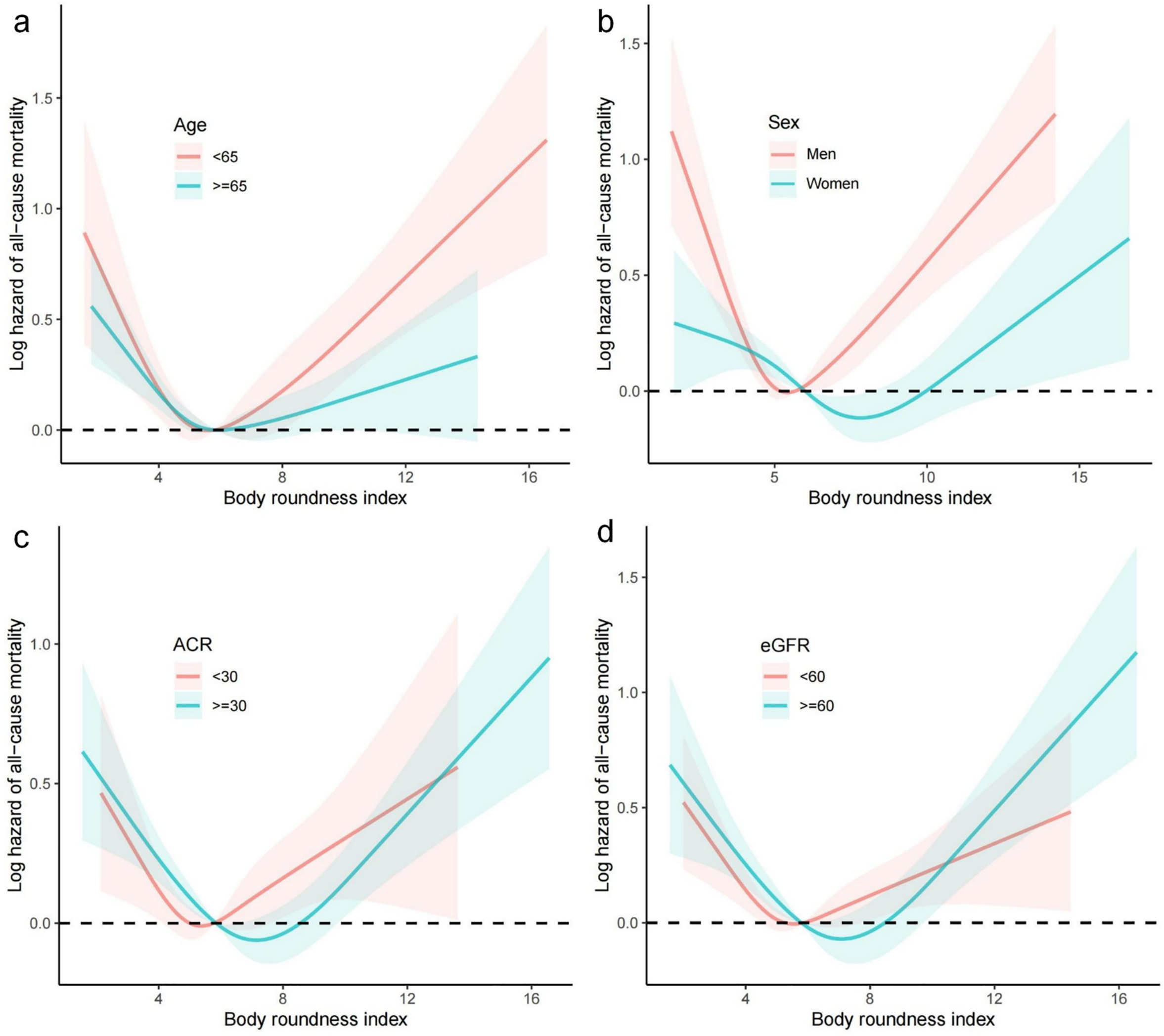

Figure 3. The U-shaped association of BRI with all-cause mortality across age, sex, ACR, and eGFR subgroups. Dose-response analyses across age, sex, ACR, and eGFR subgroups indicated stronger associations of BRI with deaths from all causes in men and patients under 65 years of age. Adjustment for age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking status, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid. BRI, body mass index; ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate; CVD, cardiovascular disease.

with distinct inflection points at 6.29 for all-cause and 6.04 for CVD mortality, suggesting a potentially different risk threshold in this population.

Notably, after full adjustment, the associations between both the highest BRI tertile and all-cause or CVD mortality, as well as the lowest tertile and CVD mortality, were no longer statistically significant. These attenuations may be due to confounding by comorbidities such as hypertension, diabetes, and dyslipidemia, which are more prevalent in individuals with high BRI and may partly mediate the risk. For low BRI, the limited number of CVD deaths and the potential competing risks from non-CVD causes may have weakened the association. In addition, tertile-based analysis may not fully capture the nonlinear relationship, which remained evident in spline and threshold models.

The potential mechanisms underlying the U-shaped association between BRI and mortality may be explained as follows. When BRI levels are excessively high, indicating visceral fat deposition, the risk of mortality in patients with CKD increases. Obesity contributes to abnormal adipose tissue remodeling, chronic low-grade inflammation, and vascular stiffening, which may partially explain the mechanisms linking elevated BRI to increased mortality [35]. The higher odds of mortality related to low BRI may be attributed to factors such as malnutrition and sarcopenia [36,37], which collectively impair

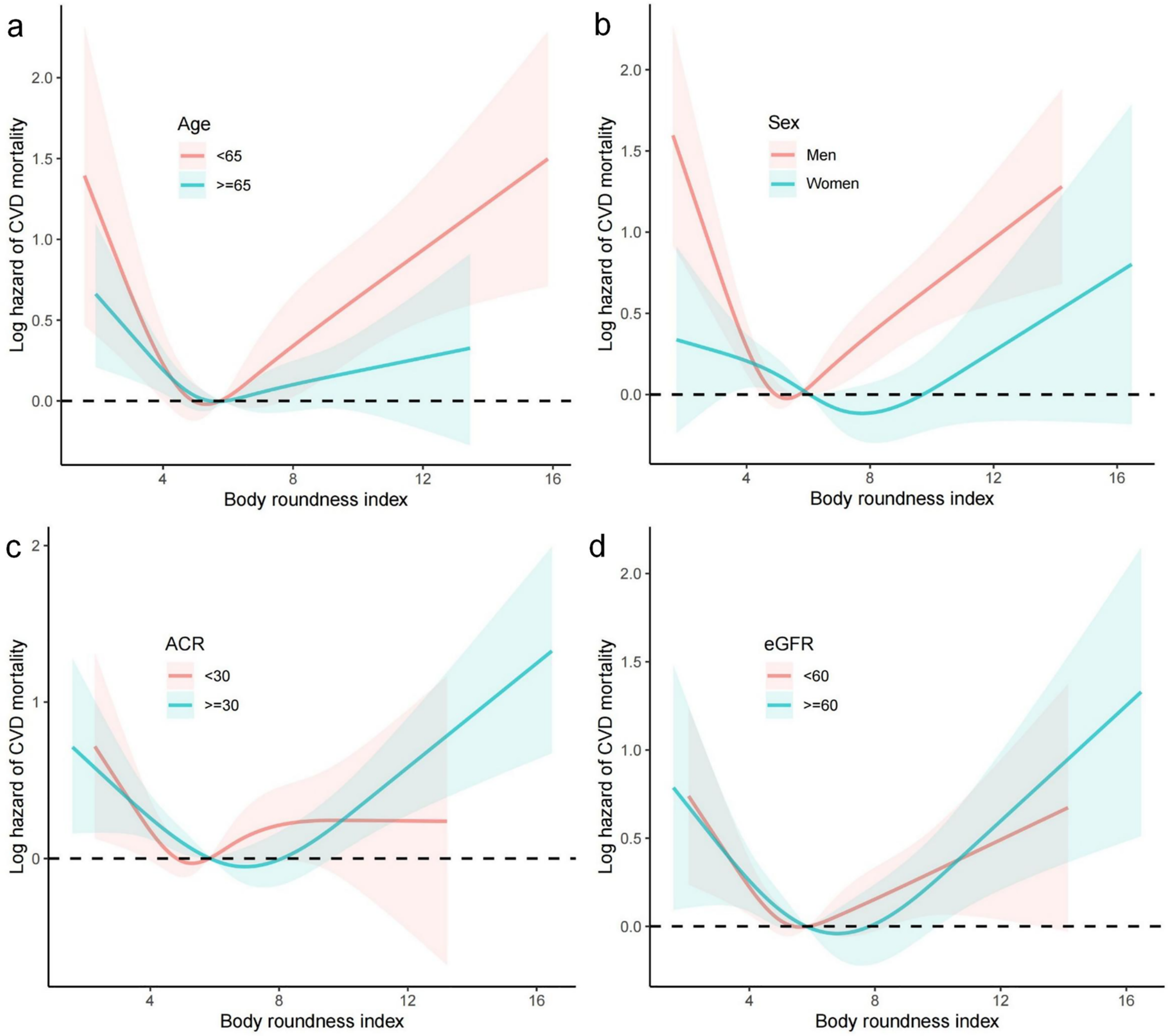

Figure 4. The U-shaped association of BRI with CVD mortality across age, sex, ACR, and eGFR subgroups. Dose-response analyses across age, sex, ACR, and eGFR subgroups indicated stronger associations of BRI with deaths from CVD in men and under 65 years of age. Adjustment for age, sex, ethnicity, education, family income-poverty ratio, smoking and drinking status, physical activity, hypertension, diabetes mellitus, dyslipidemia, CVD, ACR, eGFR and uric acid. BRI, body mass index; CVD, cardiovascular disease, ACR, albumin to creatinine ratio; eGFR, estimated glomerular infiltration rate.

immune function and metabolic capacity, thereby increasing susceptibility to infections and other adverse health outcomes [38]. Additionally, low BRI may reflect a catabolic state caused by chronic diseases, such as malignancies or advanced kidney disease [39], which further exacerbate the odds of death.

To further explore the comparative value of BRI versus BMI in predicting mortality in patients with CKD, we performed a supplementary ROC analysis. BRI and BMI exhibited nearly identical AUCs for predicting 10-year all-cause and CVD mortality, indicating comparable discrimination performance in patients with CKD. However, statistical similarity does not necessarily imply clinical equivalence. Although both BMI and BRI have shown U- or J-shaped associations with mortality in previous studies [9,40], the underlying mechanisms may differ. BMI reflects general body mass and cannot distinguish between fat and lean tissue, which may confound its association with mortality, particularly in patients with CKD where muscle wasting and fluid overload are common [39,41]. In contrast, BRI incorporates waist circumference and better approximates visceral fat, a more metabolically active and pathogenic fat tissue. These differences suggest that BRI may provide additional pathophysiological insight into obesity-related mortality risk in CKD, even if its statistical predictive power is similar to that of BMI.

We also observed stronger associations of BRI with death from all causes and CVD in men and participants under 65 years of age. The stronger association between BRI and mortality observed in men may be influenced by several factors. Men tend to store fat in the abdominal region, primarily as visceral fat [42], which is closely linked to cardiovascular diseases and metabolic disorders. The absence of estrogen's protective effects in men may worsen the impact of BRI, as estrogen helps reduce inflammation and regulate metabolism. Additionally, unhealthy habits like smoking, excessive drinking, and poor diets are more common in men and may further strengthen this relationship. The age difference may partly be explained by the fact that higher levels of body fat in younger adults are associated with accelerated early vascular aging and subsequent cardiovascular disease [6,43], ultimately increasing the odds of mortality.

Furthermore, although the average eGFR values exceeded 70 mL/min/1.73 m^2 across all BRI tertiles, we observed that the proportions of participants with more advanced CKD stages increased with higher BRI levels. To further assess whether the observed associations were confounded by kidney function, we conducted stratified analyses by eGFR level (≥60 vs. <60 mL/min/1.73 m^2). The associations between BRI and mortality remained consistent in both subgroups, suggesting that the relationship may not be entirely explained by differences in renal function. These findings together imply that BRI may serve as a risk indicator across a range of kidney function levels.

Beyond its statistical association with outcomes, BRI may also have practical value in managing CKD. The U-shaped relationship with mortality suggests that both high and low BRI levels are linked to increased risk, making BRI a potential noninvasive tool for risk stratification. For patients with high BRI, lifestyle changes such as weight loss and more physical activity may reduce risk. In contrast, low BRI may signal malnutrition or sarcopenia, warranting nutritional evaluation. Since BRI better reflects visceral fat than BMI, it may help tailor more individualized management strategies in CKD care.

This study has several limitations. First, the use of single measurements for calculating BRI, based on one-time height and waist circumference assessments, does not account for dynamic changes over time, potentially underestimating BRI's impact on mortality. Second, some subgroup analyses had small sample sizes, which may have reduced statistical power and led to non-significant findings in certain subgroups. Third, CKD was defined using a single measurement of eGFR and ACR. Although this approach is consistent with prior NHANES-based studies [44,45], it may not fully meet the Kidney Disease: Improving Global Outcomes (KDIGO) diagnostic criteria requiring persistence over 3 months. Consequently, some individuals may have been misclassified, potentially affecting the precision of CKD-related analyses. Lastly, this study focused on individuals with CKD, and we did not assess whether the association between BRI and mortality differs in those without CKD. Future research could help clarify whether these findings are specific to CKD populations or generalizable to broader populations.

Conclusions

In this nationally representative study, we found a nonlinear U-shaped association between BRI and deaths from all causes and CVD among participants with CKD, with inflection points at 6.29 and 6.04. These findings emphasize the potential application of BRI as a clinical tool for risk stratification in populations with CKD. Further research is required to establish the optimal range of BRI as a measure of visceral fat deposition.

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Ethical statement

The study procedure was approved by the National Center for Health Statistics Ethics Review Board (Protocol No. 98-12, Protocol No. 2005-06, Protocol No. 2011-17, Protocol No. 2018-01) (available at: https://www.cdc.gov/nchs/nhanes/about/erb.html). The procedures adhered to the ethical standards outlined in the Helsinki Declaration.

Consent to participate

Informed consent was obtained from all individual participants included in the study.

Author contributions

CRediT: **Bo Huang**: Data curation, Formal analysis, Methodology, Writing – original draft; **Xinxin Zhang**: Data curation, Formal analysis, Funding acquisition, Methodology, Writing – original draft; **Hongbao Guo**: Data curation, Methodology, Writing – original draft; **Cheng Meng**: Data curation, Formal analysis, Methodology; **Jingqiu Cui**: Conceptualization, Supervision, Validation, Writing – review & editing; **Junya Jia**: Conceptualization, Supervision, Validation, Writing – review & editing.

Disclosure statement

The authors report there are no competing interests to declare.

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Data availability statement

The data that support the findings of this study are openly available online (https://wwwn.cdc.gov/nchs/nhanes/default.aspx).

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