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The association between body roundness index and new-onset hyperuricemia in Chinese population: the Kailuan cohort study

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Abstract

Background This study aimed to investigate the potential relationship between the newly defined adiposity metric, the Body Roundness Index (BRI), which assesses central obesity, and the development of new-onset hyperuricemia.

Methods In the Kailuan cohort study from 2006 to 2019, 91,804 eligible participants were included. A multivariate Cox regression model was used to test the correlation between BRI and hyperuricemia. At the same time, the restricted cubic spline was applied to solve the dose-response relationship between BRI and the risk of hyperuricemia. Then, stratified analysis was carried out using multivariate Cox regression according to age, sex, hs-CRP level, TG level, education level, smoking status and hypertension status.

Results The results showed that the risk of new-onset hyperuricemia was significantly increased in the highest quartile compared with the lowest quartile. After adjusting for confounders, compared with Q1, the HR (95% CI) for new-onset hyperuricemia was 1.24 (1.18–1.30), 1.32 (1.25–1.40), and 1.40 (1.29–1.52) for Q2, Q3, and Q4, respectively. Restricted cubic spline analysis showed a J-curve relationship between baseline BRI levels and new-onset hyperuricaemia. Age, sex, hs-CRP level, TG level, income level, education level, smoking, and hypertension each had a multiplicative interaction with BRI at baseline.

Conclusion We found that elevated BRI increased the risk of developing new-onset hyperuricaemia. In addition, the association between elevated BRI and the risk of new-onset hyperuricemia showed dependency on age, sex, hs-CRP level, TG level, education level, smoking status and hypertension status.

Keywords Body roundness index, Obesity, Hyperuricemia

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Background

The prevalence of hyperuricemia, as a metabolic disorder, is increasing significantly. Previous studies have shown that the prevalence of hyperuricemia in Chinese adults increased from 11.1 to 14.0% between 2015 and 2019 [1]. Long-term elevation of serum uric acid (SUA) can trigger adverse outcomes such as gouty arthritis, gouty nephropathy and cardiovascular disease, thus increasing the burden of hyperuricemia on society [2–4]. Hyperuricemia is usually attributed to chronic high-purine, high-fat and high-fructose diets and alcohol abuse [5]. However, studies have shown that obesity, rather than poor dietary habits, has a more significant effect on hyperuricemia [6, 7]. Therefore, a valid and accurate assessment of obesity is essential.

A commonly used indicator for obesity assessment in clinical practice is body mass index (BMI). However, it has some limitations that it confuses the proportion of fat and muscle in the human body and cannot accurately assess the proportion of fat [8–10]. Normal weight central obesity is another independent risk factor for hyperuricemia [11]. Specifically, increased visceral fat, the main feature of central obesity, significantly raises the risk of hyperuricemia [12]. However, CT-based visceral fat detection method is so expensive that it cannot be widely used in clinical practice. The body roundness index (BRI) is a new obesity-related anthropometric index based on waist circumference and height, which reflects the ratio of body fat to visceral fat and is more economical and convenient [13, 14].

However, the correlation between BRI and the risk of new-onset hyperuricemia has not been explored. Therefore, the present study, based on the Kailuan cohort study (Registration number: ChiCTR-TNRC-11001489, Registration Date: 20150719), aimed to investigate the relationship between BRI and new-onset hyperuricemia.

Methods

Study participants

The Kailuan Study which started in 2006 is a large prospective cohort study of active and retired workers of Kailuan Group. For this study, physical examinations including measurements of weight, waist circumference, height and SUA, were performed every 2 years. Participant was selected as the observation population for this study based on the following inclusion criteria: (1) they had a valid health report from the health check-up in 2006; (2) their data on waist circumference, height and SUA were availabe; (3) they agreed to participate in this study and signed the informed consent form. Exclusion criteria were: (1)they had hyperuricemia at the time of physical examination in 2006; (2) they had a history of malignant tumor before the health check-up in 2006 (Fig. 1). This study was approved by the Ethics Committee of Kailuan General Hospital (approval number: 200605).

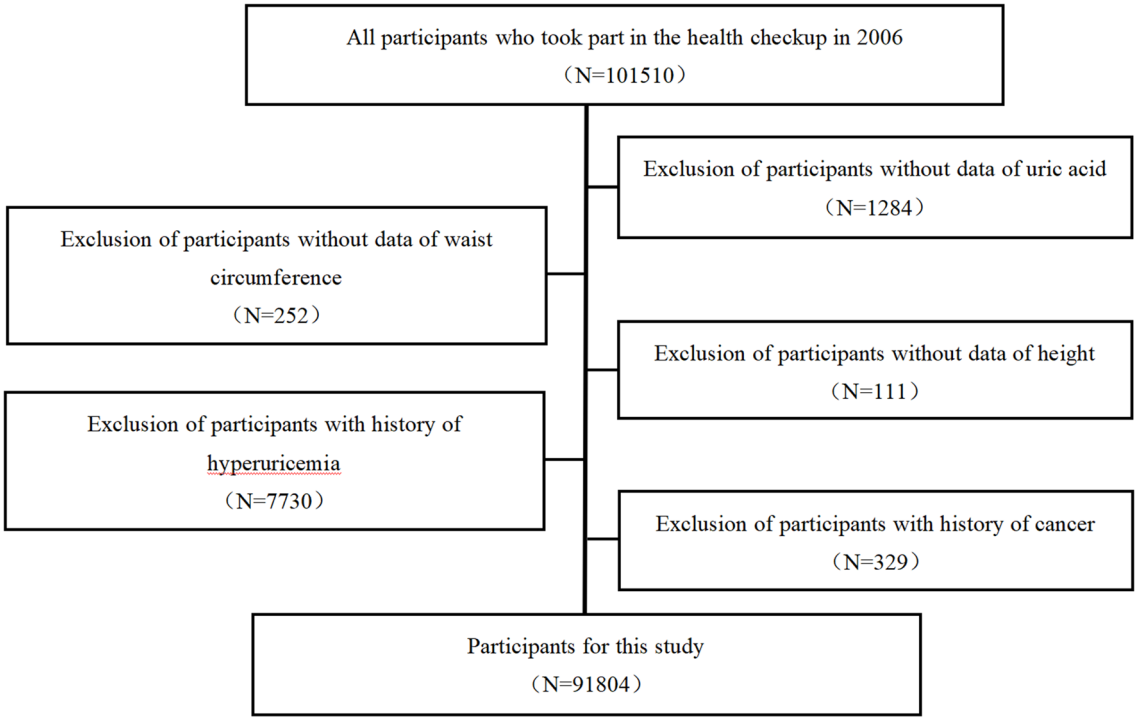

Fig. 1 Flow chart of the selection of study participants

Data collection

Height, weight and waist circumference: the observation subjects took off their shoes, hats and light single clothes, waist circumference was measured at the midpoint between the lower edge of the ribs and the upper edge of the sternum, and height and weight were measured using a calibrated RGZ-120 body mass scale.

SUA collection method: 5mL of venous blood from the elbow of the examinee was drawn on an empty stomach at 07:00–09:00 a.m. on the day of the checkup, and the SUA level was measured by Hitachi 7600 automatic biochemical analyzer, using the oxidase method. The SUA kit was provided by Shanghai Kewa Bioengineering Co.

Covariates

Anthropometric indicators, biochemical determinations and epidemiologic investigations are described in the published papers of our team [15, 16]. Anthropometric indicators included systolic and diastolic blood pressure. Biochemical indices included fasting blood glucose (FBG), triglycerides (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), serum creatinine (SCr), and ultrasensitive C-reactive protein (hs-CRP). Epidemiological information included demographic information (gender, age, education level, income level), health behavioral habits (smoking, alcohol consumption, physical activity), disease history (history of hypertension, history of diabetes mellitus), and medication history (whether or not they were taking antihypertensive, hypoglycemic, or lipid-lowering medications). Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). Estimated glomerular filtration rate (eGFR) was calculated using the Chronic Kidney Disease Epidemiology Collaborative Study formula method [17]. Hypertension was defined as a systolic blood pressure ≥ 140 mmHg and/or a diastolic blood pressure ≥ 90 mmHg, or as systolic blood pressure < 140 mmHg and a diastolic blood pressure < 90 mmHg but with a history of definitively diagnosed hypertension or current use of antihypertensive medication [18]. Diabetes mellitus was diagnosed as fasting blood glucose ≥ 7.0 mmol/L or fasting blood glucose < 7.0 mmol/L with a history of clearly diagnosed diabetes mellitus or current use of hypoglycemic medication [19]. Physical activity was defined as exercising at least 3 times per week for ≥ 30 min per session. Smoking was defined as an average consumption of at least 1 cigarette per day in the past year. Alcohol consumption was defined as drinking an average of about 100 ml of alcohol (with ≥ 50% alcohol content) per day for at least 1 year in the past year.

Variable

BRI = 364.2–365.5 × (1 - [waist circumference (m)/2π]^2/[0.5 × height (m)]^2)$^{1/2}$ [14]. The 25th, 50th, and 75th percentile values of BRI were calculated and divided into quartile 1 (Q1), quartile 2 (Q2), quartile 3 (Q3), and quartile 4 (Q4). Q1: BRI < 2.97; Q2: 2.97 ≤ BRI < 3.65; Q3: 3.65 ≤ BRI < 4.45; Q4: BRI ≥ 4.45.

Outcome and follow-up time

New-onset hyperuricemia was defined as of SUA > 420 μmol/L (7.0 mg/dL) for men and SUA > 360 μmol/L (6.0 mg/dL) for women at a checkup, as recommended in the Uric Acid Right for Heart Health (URRAH) Project [20, 21]. Outcomes included new-onset hyperuricemia and death. The time of completing the 2006 health check-up was used as the starting point for follow-up, the time of the first occurrence of hyperuricemia or death was used as the endpoint for follow-up. The endpoint for follow-up was December 31, 2019 for those who did not have an event.

Statistical analysis

Normally distributed measures were expressed as mean ± standard deviation ($\bar{x}$ ± s), and comparisons between groups were conducted using analysis of variance (ANOVA). Measures with skewed distribution were presented as median (P25, P75), and comparisons between groups were conducted using the nonparametric rank sum test (Kruskal-Wallis). Count data were presented as frequencies and percentages, and comparisons between groups were conducted using the χ^2 test.

The cumulative incidence of hyperuricemia across different groups was calculated using the Kaplan-Meier method, and comparisons between groups were performed using the Log-rank test. The association of BRI on new-onset hyperuricemia was analyzed using a Cox proportional hazards regression model using the lowest quartile of BRI as the reference group. Model 1 was adjusted for age (continuous) and sex (male or female); Model 2 further adjusted for smoking (yes or no), alcohol consumption (yes or no), physical activity (yes or no), education (high school and above or below), hypertension (yes or no), diabetes mellitus (yes or no), and HDL-C (< 1.16 mmol/L in men and < 1.29 mmol/L in women or ≥ 1.16 mmol/L for men and ≥ 1.29 mmol/L for women), LDL-C (> 3.12 mmol/L or ≤ 3.12 mmol/L), eGFR (< 60 ml/min/1.73 m^2 or ≥ 60 ml/min/1.73 m^2); Model 3 Further adjusted for medication use based on Model 2, the including antihypertensive drugs (yes or no), hypoglycemic drugs (yes or no), and lipid-lowering drugs (yes or no). Model 4 further adjusted for height (continuous) and waist circumference (continuous) based on Model 3.

The dose-response relationship between BRI and risk of hyperuricemia was analyzed using restricted cubic spline plots (RCS) with nodes placed at the 30th, 60th, and 90th percentiles of BRI, respectively.

The multiplicative interactions of age, sex, hs-CRP, TG, income level, education level, smoking, and hypertension with BRI were explored using Cox proportional hazrds regression models, stratified by age (≥ 60 years or < 60 years), sex (male or female), hs-CRP (≥ 3 mg/L or < 3 mg/L), TG (≥ 1.70 mmol/L or < 1.70 mmol/ L), educational status (high school and above or below), smoking (yes or no), and hypertension (yes or no).

To test the robustness of the results, we performed 5 sensitivity analyses: the Cox proportional regression model analysis was repeated after (1) excluding those who had an event within 2 years of the start of follow-up (i.e., either the occurrence of hyperuricemia or death), (2) excluding those who used antihypertensive medication, (3) excluding those who used lipid-lowering medication, (4) excluding those who used glucose-lowering medication, and (5) further adjusted for the change of BRI (formula: change of BRI = follow-up BRI - baseline BRI).

Data were analyzed using SAS 9.4 (sas Institute, Cary, North Carolina) statistical software. Two-sided tests were performed with $P < 0.05$ indicating statistical difference.

Results

Baseline characteristics

A total of 101,510 cases participated in the 2006 health check-up of Kailuan Group. After excluding 1647 cases with missing data on height, waist circumference and SUA, 7730 cases with hyperuricaemia, and 329 cases with cancer, 91,804 cases met the inclusion criteria (Fig. 1). The mean age of the study subjects was 51.50 ± 12.46 years, and 73,062 cases (79.58%) being male. With the increase of BRI level from Q1 to Q4, age, TC, TG, FBG, SBP, BMI, hs-CRP, and waist circumference gradually increased, while height, eGFR, and HDL-C gradually decreased. The proportions of hypertension, diabetes mellitus and individuals taking antihypertensive, hypoglycemic, and lipid-lowering drugs showed an increasing trend, whereas the proportions of those educated in high school level or higher showed a decreasing trend. The differences in comparisons between the groups were all statistically significant (all P values < 0.0001) (Table 1).

The association of BRI on new-onset hyperuricaemia

In this study, the mean follow-up duration was 10.77 ± 3.21 years, during which 21,302 new-onset hyperuricemia events occurred. The incidence rate of new-onset hyperuricemia in different BRI quartiles were

Table 1 Baseline characteristics of the study population according to body roundness index quartiles

	Overall *n* = 91,804	Q1 (< 2.97) *n* = 22,753	Q2 (2.97–3.65) *n* = 22,949	Q3 (3.65–4.45) *n* = 23,151	Q4 (≥ 4.45) *n* = 22,951	*P* value
Age (year)	51.50 ± 12.46	46.72 ± 12.98	50.62 ± 11.71	52.74 ± 11.64	55.88 ± 11.64	<0.0001
Male [n (%)]	73,062 (79.58)	17,120 (75.24)	19,000 (82.79)	19,075 (82.39)	17,867 (77.85)	<0.0001
TC (mmol/L)	4.92 ± 1.11	4.81 ± 1.04	4.93 ± 1.10	4.95 ± 1.14	5.01 ± 1.15	<0.0001
TG (mmol/L)	1.24 (0.88–1.87)	0.99 (0.72–1.38)	1.21 (0.88–1.76)	1.36 (0.97–2.05)	1.51 (1.06–2.27)	<0.0001
LDL-C (mmol/L)	2.34 ± 0.89	2.36 ± 0.79	2.40 ± 0.83	2.36 ± 0.91	2.25 ± 1.01	<0.0001
HDL-C (mmol/L)	1.55 ± 0.39	1.58 ± 0.38	1.56 ± 0.39	1.54 ± 0.39	1.52 ± 0.41	<0.0001
FBG (mmol/L)	5.47 ± 1.64	5.22 ± 1.31	5.39 ± 1.51	5.54 ± 1.71	5.75 ± 1.92	<0.0001
SBP (mmol/L)	130.59 ± 20.85	122.83 ± 19.12	129.55 ± 19.90	132.78 ± 20.56	137.11 ± 21.12	<0.0001
BMI (kg/m^2)	24.93 ± 3.43	22.12 ± 2.59	24.26 ± 2.48	25.66 ± 2.60	27.64 ± 3.39	<0.0001
hs-CRP (mg/L)	0.80 (0.30–2.13)	0.50 (0.20–1.27)	0.70 (0.28–1.70)	0.90 (0.34–2.28)	1.30 (0.50–3.60)	<0.0001
eGFR (ml/min/1.73m^2)	83.77 ± 22.46	86.73 ± 22.54	83.12 ± 22.98	82.59 ± 21.38	82.66 ± 22.66	<0.0001
WC (cm)	86.71 ± 9.99	75.27 ± 5.33	83.69 ± 3.71	89.21 ± 4.05	98.55 ± 7.43	<0.0001
Height (cm)	167.36 ± 6.96	168.38 ± 6.98	168.20 ± 6.59	167.18 ± 6.76	165.69 ± 7.18	<0.0001
Current smoker [n (%)]	30,397 (33.11)	8090 (35.56)	7959 (34.68)	7799 (33.69)	6549 (28.53)	<0.0001
Current drinker [n (%)]	32,065 (35.52)	8618 (37.88)	8536 (37.20)	8433 (36.43)	7018 (30.58)	<0.0001
Physical activity [n (%)]	83,169 (90.59)	20,364 (89.50)	20,752 (90.43)	21,018 (90.79)	21,035 (91.65)	<0.0001
High school and above [n (%)]	11,024 (12.01)	4404 (19.36)	2624 (11.43)	2304 (9.95)	1692 (7.37)	<0.0001
Hypertension [n (%)]	39,823 (43.38)	6281 (27.61)	9537 (41.56)	11,042 (47.70)	12,963 (56.48)	<0.0001
Diabetes [n (%)]	8633 (9.40)	1000 (4.40)	1757 (7.66)	2460 (10.63)	3416 (14.88)	<0.0001
Antihypertensive drugs [n (%)]	21,859 (23.81)	2923 (12.85)	4649 (20.26)	6245 (26.98)	8042 (35.04)	<0.0001
Hypoglycemic drugs [n (%)]	5031 (5.48)	458 (2.01)	893 (3.89)	1519 (6.56)	2161 (9.42)	<0.0001
Lipid-lowering drugs [n (%)]	2786 (3.03)	354 (1.56)	578 (2.52)	828 (3.58)	1026 (4.47)	<0.0001

Note:

Abbreviation: BMI, body mass index; BRI, body roundness index; eGFR, estimated glomerular filtration rate; FBG, fasting blood glucose; hs-CRP, high sensitivity C reactive protein; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglyceride; WC, waist circumference

Table 2 HR and 95%CI of new-onset hyperuricemia according to quartiles of BRI

	Case/total	Incidence rate (per 1000 person-years)	Model 1	Model 2	Model 3	Model 4
Q1	4357/22,753	17.15	Ref.	Ref.	Ref.	Ref.
Q2	5243/22,949	20.96	1.37 (1.31–1.43)	1.34 (1.29–1.40)	1.32 (1.27–1.38)	1.24 (1.18–1.30)
Q3	5696/23,151	23.01	1.60 (1.54–1.67)	1.53 (1.47–1.59)	1.48 (1.42–1.54)	1.32 (1.25–1.40)
Q4	6006/22,951	25.35	1.97 (1.89–2.05)	1.77 (1.69–1.85)	1.69 (1.61–1.77)	1.40 (1.29–1.52)
P for trend			<0.0001	<0.0001	<0.0001	<0.0001

Note:

Model 1: adjusted for age, gender

Model 2: included variables in model 1 and further adjusted for smoking status, drinking status, physical activity, body mass index, education level, hypertension, diabetes mellitus, eGFR, HDL-C and LDL-C

Model 3: included variables in model 2 and further adjusted for hypoglycemic drugs, anti-hypertensive drugs and lipid-lowering drugs

Model 4: included variables in model 3 and further adjusted for height and waist circumference at baseline

Abbreviation: BRI, body roundness index; HR, hazard rate; CI, confidence interval

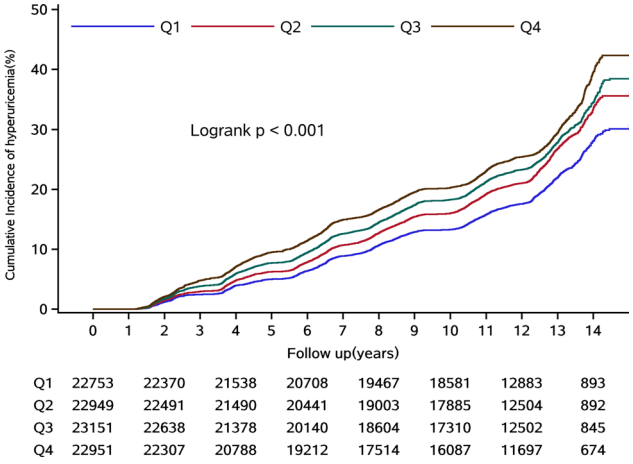

Fig. 2 Kaplan–Meier incidence rate of new-onset hyperuricemia according to quartiles of BRI

17.15, 20.96, 23.01, and 25.35 per 1000 person-years, respectively (Table 2). The cumulative incidence of new-onset hyperuricemia tended to increase with increasing BRI levels, and the difference in the cumulative incidence of new-onset hyperuricemia was statistically significant (Log-Rank $P < 0.001$) between the quartiles (Fig. 2).

Cox proportional hazards regression analyses using different BRI levels as the independent variable and new-onset hyperuricemia as the dependent variable showed a significantly increased in the highest quartile compared with the lowest quartile. After adjusting for confounders, the HR (95% CI) for new-onset hyperuricemia was 1.24 (1.18–1.30), 1.32 (1.25–1.40), and 1.40 (1.29–1.52) for Q2, Q3, and Q4, respectively, compared with Q1 (Table 2). Restricted cubic spline analysis showed a J-curve relationship between baseline BRI levels and new-onset hyperuricaemia, with significant overall ($P < 0.0001$) and non-linear associations ($P < 0.0001$) (Fig. 3).

Stratified analyses

Age, sex, hs-CRP, TG, income level, education level, smoking, and hypertension each showed a multiplicative interaction with BRI at baseline (all interaction P values ≤ 0.001). Cox regression analyses stratified by age (≥ 60 years or < 60 years), sex (male or female), hs-CRP (≥ 3 mg/L or < 3 mg/L), TG (≥ 1.70 mmol/L or < 1.70 mmol/L), educational status (high school and above or below), smoking (yes or no), and hypertension (yes or no) revealed a significant interaction with BRI. The effect of elevated BRI on new-onset hyperuricemia was more pronounced in those aged ≥ 60 years, female, hs-CRP ≥ 3 mg/L, TG < 1.70 mmol/L, income level ≥ 800 yuan/month, high school education or highter, non-smokers, and non-hypertensive individuals (Table 3).

Sensitivity analyses

To rule out the possibility of reverse causation, medication effects and impact of change of BRI during follow-up, the Cox regression analyses described above were repeated after excluding those individuals with an event within 2 years of follow-up ($n = 1998$) and those using antihypertensive ($n = 21859$), lipid-lowering ($n = 2786$), glucose-lowering ($n = 5031$) medications, and further adjusted for the change of BRI, respectively. Results were consistent with the main findings (Supplement Table 1).

Discussion

In this study, elevated BRI was found to be an independent risk factor for new-onset hyperuricaemia. The risk of developing new-onset hyperuricemia was significantly higher with increasing BRI levels and showed a non-linear correlation. In addition, the association between elevated BRI and the risk of new-onset hyperuricemia was influenced by age, sex, hs-CRP level, TG level, education level, smoking status and hypertension status.

The primary finding of this study was that elevated BRI independently increased the rist of new-onset hyperuricaemia. There was a progressive rise in hyperuricemia

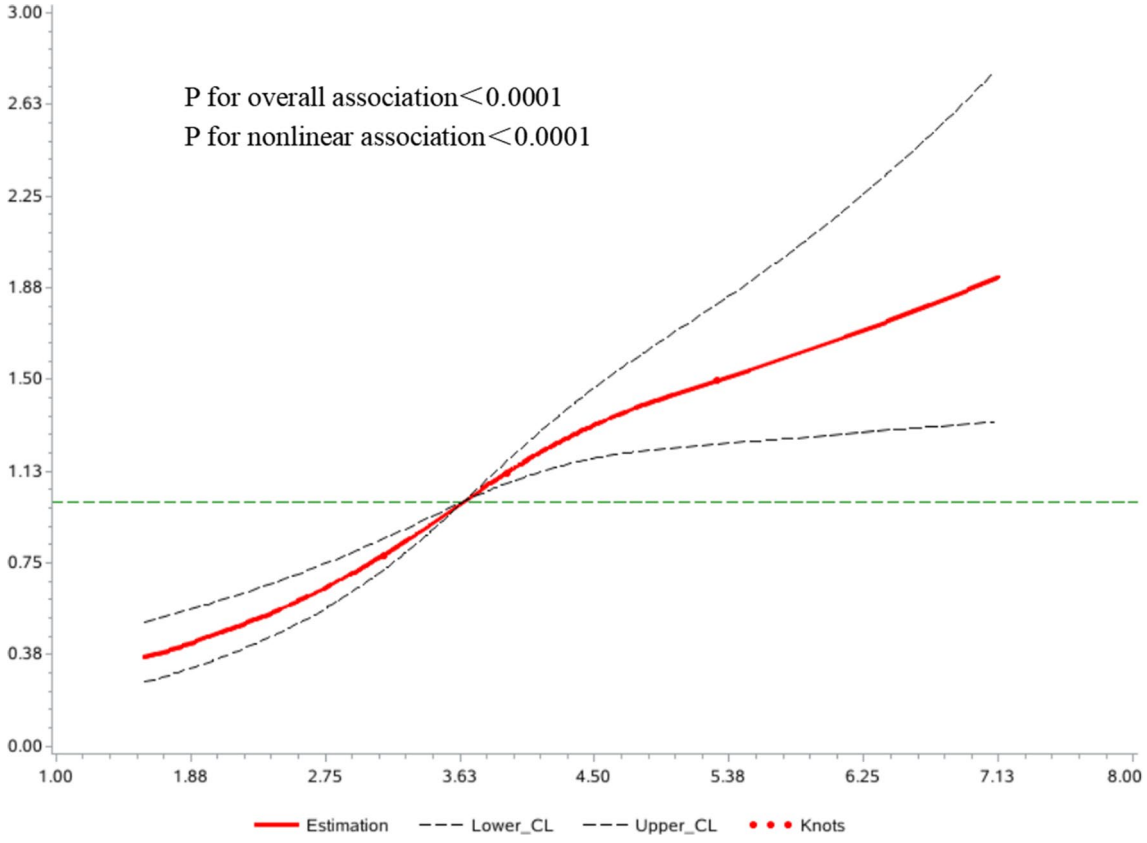

Fig. 3 Multivariable-adjusted hazard ratios for new-onset hyperuricemia based on restricted cubic spines with 3 knots at 30th, 60th and 90th percentiles of BRI

risk from Q2 to Q4 compared to Q1, with an increase of up to 69% in Q4. Although the risk decreased after adjusting for baseline height and waist circumference, it still increased by 40%, a result that suggests that the association between BRI and the risk of hyperuricemia is independent of waist circumference and height. To our knowledge, this is the first study that has observed such an association of BRI with new-onset hyperuricemia. In two previous studies in the United States and Spain, the risk of hyperuricemia has been found to increase by 177% and 66% in Q4 compared to Q1 [22, 23]. Our results not only confirm but also extend these findings.

In addition, we found that the factos of age, sex, hs-CRP level, TG level, education level, smoking habits, and hypertension status interacted with the effects of BRI on new-onset hyperuricaemia. Stratified analyses revealed that compared to young and middle-aged adults, males, low hs-CRP level, high TG level, low education, smokers, and hypertensives, the risk of new-onset hyperuricemia due to high BRI was significantly increased in older adults, females, high hs-CRP level, low TG level, high education, non-smokers and non-hypertensives (Table 3). These results suggest that the relationship between elevated BRI and the risk of new-onset hyperuricemia depends on the factors including age, sex, hs-CRP level, TG level, education level, smoking status, and hypertension status.

Regarding the potential effects of age, elderly people may have a reduced metabolic rate, making them more susceptible to an increased risk of new-onset hyperuricemia associated with elevated BRI. As for gender differences, females face an increased risk of developing hyperuricemia due to the decline in oestrogen levels after menopause, which subsequently affects insulin sensitivity and fat distribution [24]. Hyperuricemia may be exacerbated in individuals with high hs-CRP levels due to the chronic inflammatory state in the body. In addition, highly educated individuals may have an abnormal body fat distribution and increased purine intake due to their sedentary work environment and high-purine diet, making the effect of BRI on their risk of hyperuricemia more prominent [25–27]. The risk of new-onset hyperuricemia due to high BRI also significantly increased in people with low TG levels, non-smokers and no hypertension, suggesting that elevated BRI remains a significant independent risk factor for new-onset hyperuricemia even in individuals with low traditional risk factors.

Although BMI is an important measure of obesity, the results of this study showed that BRI was still significantly associated with the risk of developing new-onset

Table 3 Stratified analysis for HR and 95%CI of new-onset hyperuricemia according to quartiles of BRI

	Q1	Q2	Q3	Q4	*p* for interaction
	HR(95%CI)	HR(95%CI)	HR(95%CI)	HR(95%CI)	
Age (years)					<0.0001
<60	Ref.	1.33 (1.27–1.39)	1.48 (1.42–1.55)	1.69 (1.60–1.77)	
≥ 60	Ref.	1.41 (1.22–1.62)	1.57 (1.37–1.79)	1.74 (1.52–1.98)	
Gender					<0.0001
Female	Ref.	1.38 (1.25–1.52)	1.60 (1.45–1.76)	1.79 (1.61–1.98)	
Male	Ref.	1.28 (1.22–1.34)	1.41 (1.35–1.48)	1.61 (1.53–1.70)	
hs-CRP (mg/L)					0.0007
<3	Ref.	1.34 (1.28–1.40)	1.47 (1.40–1.54)	1.61 (1.53–1.69)	
≥ 3	Ref.	1.24 (1.12–1.39)	1.44 (1.30–1.59)	1.72 (1.55–1.91)	
TG (mmol/L)					0.0003
<1.7	Ref.	1.30 (1.24–1.36)	1.44 (1.37–1.52)	1.64 (1.55–1.72)	
≥ 1.7	Ref.	1.19 (1.10–1.29)	1.27 (1.18–1.37)	1.44 (1.33–1.57)	
Education level					<0.0001
Below high school	Ref.	1.27 (1.21–1.33)	1.40 (1.34–1.48)	1.66 (1.58–1.75)	
High school and above	Ref.	1.45 (1.34–1.56)	1.67 (1.54–1.80)	1.68 (1.52–1.86)	
Smoke					<0.0001
No	Ref.	1.34 (1.27–1.41)	1.51 (1.43–1.59)	1.77 (1.68–1.88)	
Yes	Ref.	1.29 (1.21–1.37)	1.42 (1.33–1.52)	1.52 (1.41–1.65)	
Hypertension					0.001
No	Ref.	1.39 (1.32–1.46)	1.53 (1.46–1.62)	1.73 (1.63–1.84)	
Yes	Ref.	1.18 (1.10–1.26)	1.34 (1.25–1.43)	1.53 (1.43–1.65)	

Note:

Model adjusted for age, gender, smoking status, drinking status, physical activity, body mass index, education level, hypertension, diabetes mellitus, eGFR, HDL-C, LDL-C, hypoglycemic drugs, anti-hypertensive drugs and lipid-lowering drugs

hyperuricaemia, even in models adjusted for BMI. It suggests that elevated BRI is an independent risk factor apart from BMI. In addition, previous studies have shown that the use of antihypertensive, lipid-lowering and glucose-lowering medications may increase the risk of hyperuricemia [28–30]. To exclude the effect of medications as confounders, we repeated the Cox proportional hazard regression model analysis after separately excluding antihypertensive, lipid-lowering and glucose-lowering medications. However, the results showed that these confounders had no significant effect on the association between BRI and new-onset hyperuricemia. Nevertheless, the risk of new-onset hyperuricemia was not reduced by eliminating these associated risk factors. These findings suggest that BRI is an independent risk factor for new-onset hyperuricemia and is not influenced by other risk factors.

Although the underlying mechanism between BRI and new-onset hyperuricemia is unclear, relevant studies have found that increased BRI reflects abnormal distribution and dysfunction of adipose tissue, particularly visceral fat accumulation, which leads to increased uric acid secretion and inhibits uric acid excretion. Firstly, excessive visceral fat deposition affects purine metabolism in the liver and increases uric acid production [31]. Secondly, large accumulations of visceral fat lead to the release of large amounts of pro-inflammatory factors, such as leptin and aldosterone, which cause insulin resistance and increase the risk of renal damage, thereby affecting the kidney's ability to excrete uric acid [32]. Therefore, in order to assess the risk of developing new-onset hyperuricemia, an accurate assessment of obesity status is necessary [33]. In addition, previous studies have shown that obesity and hyperuricemia share several lifestyle-related common risk factors, such as sedentary lifestyle and poor dietary habits. Prolonged periods of sedentary work or recreational activities significantly reduce daily energy expenditure, leading to excess energy and storage in adipose tissue, particularly visceral fat. At the same time, a sedentary lifestyle lowers the overall metabolic rate, further increasing the risk of hyperuricemia. And unhealthy eating habits, such as high-calorie, high-fat, high-sugar and high-purine diets, directly result in energy intake exceeding expenditure, leading to weight gain and fat accumulation. Also, a long-term diet high in sugar and purines directly increases the risk of hyperuricemia.

This study reveals that elevated BRI is an independent risk factor for new-onset hyperuricemia with clear public health implications. Firstly, BRI, as a more comprehensive indicator for assessing fat distribution, may help to identify people at high risk of new-onset hyperuricemia, especially those with normal BMI but abnormal body fat distribution. Secondly, previous studies have shown that lifestyle interventions in obese populations

can effectively prevent and/or delay the onset of hyperuricemia, as well as reduce the incidence of gout and cardiovascular disease and lower all-cause mortality [6, 34, 35]. Understanding the impact of BRI on new-onset hyperuricemia can provide a scientific basis for developing public health policies that promote healthy lifestyles and implement interventions to reduce the incidence and associated burden of hyperuricemia. Finally, this study found that the association between elevated BRI and the risk of new-onset hyperuricemia showed dependency on age, gender, hs-CRP level, TG level, education level, smoking status and hypertension status dependency, emphasizing the importance of individualised prevention strategies to provide precise interventions for different risk factors and improve the effectiveness of public health management.

The present study has several strengths. First, there is a lack of large prospective cohort studies exploring the association between BRI and the risk of developing new-onset hyperuricemia. The present study is the first to observe the association. Second, the data in this study were obtained from the Kailuan study, a cohort study with a large sample size, a long follow-up period, and a standardised protocol used for data collection. Finally, stratified analyses were performed in this study to ensure that the relationship between BRI and new-onset hyperuricemia remained stable across populations and to ensure that the results were robust.

However, this study also has some limitations that need to be noted. Firstly, although confounders were adjusted during the multivariate Cox proportional hazard regression model analysis, potential residual confounders may still exist, such as the use of uric acid-lowering medications and dietary habits. However, the uric acid-lowering treatment and detailed dietary information were not collected in our study. Second, the study population consisted of a Chinese community-based population, which is not representative of all populations. Given these limitations, future studies are needed to take these factors into consideration. For example, future studies could investigate varied populations and explore the impacts of uric acid-lowering medication or dietary habits on the association between elevated BRI and hyperuricemia risks.

Conclusions

In this large community-based prospective study, we examined the relationship between BRI and new-onset hyperuricaemia. We found that elevated BRI increased the risk of developing new-onset hyperuricaemia. In addition, the association between elevated BRI and the risk of new-onset hyperuricemia showed dependency on age, sex, hs-CRP level, TG level, education level, smoking status and hypertension status.

Abbreviations

BMI	Body mass index
BRI	Body roundness index
eGFR	Estimated glomerular filtration rate
FBG	Fasting blood glucose
hs-CRP	High sensitivity C reactive protein
HDL-C	High-density lipoprotein cholesterol
LDL-C	Low-density lipoprotein cholesterol
SBP	Systolic blood pressure
TC	Total cholesterol
TG	Triglyceride
WC	Waist circumference

Supplementary Information

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Supplementary Material 1

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Author contributions

Liufu Cui and Shouling Wu had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Liufu Cui, Shouling Wu, Xuemei Yang, Yicheng Liao, and Xi Cai contributed to the study design. Na Li, Naihui Zhao and Shouling Wu have accessed data and Liufu Cui, Shouling Wu, Xi Cai, Jiajia Ma, Yajing Liang, and Naihui Zhao have verified the data. Xi Cai wrote the manuscript. Ruiyue Liu, Xinran Wen, Shuohua Chen, Guodong Wang, Liufu Cui and Shouling Wu reviewed and edited the manuscript. All authors made important contributions to editing and critically revising the manuscript for important intellectual content. All authors have read and approved the final manuscript. Liufu Cui, Shouling Wu, Xi Cai were responsible for the decision to submit the manuscript.

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Data availability

The datasets generated and/or analyzed during the current study are not publicly available but are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

This study was approved by the ethics committees of Kailuan General Hospital (Approve No.: 2006-5) and conducted in accordance with the Declaration of Helsinki. A written informed consent form was obtained from all participants.

Consent for publication

None.

Competing interests

The authors declare no competing interests.

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