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Association between body roundness index and risk of osteoarthritis: a cross-sectional study

Xudong Wang[1†], Zijian Guo[1†], Meng Wang[1,2] and Chuan Xiang[1*]

Abstract

Background The link between body roundness index (BRI) and osteoarthritis (OA) has yet to be validated. Our aim was to explore this connection between BRI and OA risk.

Methods This cross-sectional study utilized the 1999–2018 National Health and Nutrition Examination Survey retrieved data. To assess the association between BRI and OA risk, we performed weighted multivariable regression analysis (MVRA), with smooth curve fitting for potential nonlinear association and subgroup analysis and interaction tests for relationships in specific subgroups. A 7:3 ratio was adopted for the random division of the acquired data into training and validation sets. Subsequently, least absolute shrinkage and selection operator regression, along with MVRA, were conducted for the training set to isolate variables for a prediction model. This model was visualized using the nomogram and was followed by evaluation. Finally, the validation set was utilized to validate the model.

Results This study enrolled 12,946 individuals. Following the adjustment for all covariables, OA risk increased by 18% with every unit rise in BRI (odd ratio [OR] = 1.18; 95% confidence interval [CI]: 1.13–1.23; $P < 0.0001$). Upon regarding BRI as a categorical variable, it was divided into quartiles for subsequent analysis. In comparison to quartile 1, the risk of OA was increased in quartile 2 (OR = 1.58; 95% CI: 1.22–2.03; $P = 0.0006$), quartile 3 (OR = 1.83; 95% CI: 1.40–2.40; $P < 0.0001$) and quartile 4 (OR = 2.70; 95% CI: 1.99–3.66; $P < 0.0001$). Smooth curve fitting revealed no non-linear relationships. None of the subgroups showed a statistically significant interaction (all $P > 0.05$). After selecting the variables, a prediction model was developed. The prediction model exhibited favorable discriminatory power, high accuracy, and potential clinical benefits in training and validation sets.

Conclusions The BRI was positively associated with OA risk. Our predictive model demonstrated that combining BRI with other easily accessible factors was helpful in assessing and managing high-risk OA groups.

Keywords Body roundness index, Osteoarthritis, Cross-sectional study, National Health and Nutrition Examination Survey

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Introduction

Osteoarthritis (OA) is marked by cartilage deterioration, bone remodeling, osteophyte formation, and synovial inflammation [1, 2]. More than 360 million individuals are afflicted by OA, and its incidence continues to rise [3]. The disease can affect several joints, with the hip, knees, and hands being the most frequently afflicted [4]. Osteoarthritis is a primary factor of disability in individuals aged > 60 years and has a greater impact on women than on men [5, 6]. Individuals suffering from OA have a higher all-cause mortality risk and are more prone to mortality related to cardiovascular disease [7, 8]. Furthermore, OA is correlated with poor quality of life among elderly individuals and results in an immense individual and societal financial burden [9–11]. Therefore, prevention of OA and identification of those at high risk are crucial strategies for easing these burdens.

There are two primary categories of OA risk factors: person-level and joint-level [12]. Age, sex, genes, and obesity are factors at the person level, whereas injury, malalignment, and abnormal loading are factors at the joint level [12]. Some factors can be addressed through treatments or lifestyle changes, while others cannot. For example, age cannot be changed, while obesity can be more effectively managed through medical or behavioral therapies [13, 14]. The body mass index (BMI) represents a common obesity indicator, with its heightened values being strongly related to OA development [15–17]. The body roundness index (BRI) constitutes a novel anthropometric marker used to measure obesity [18]. Unlike BMI, the BRI accounts for both height and waist circumference (WC) and presents a more accurate indication of the ratio between body fat and visceral adipose tissue [19].

Currently, the link between BRI and OA has yet to be validated. Accordingly, this cross-sectional study aimed to uncover the connection between BRI and OA risk and evaluate BRI suitability as a predictor for high-risk OA groups.

Methods

Study population

Data were acquired from the National Health and Nutrition Examination Survey (NHANES), a nationwide survey using a randomized, stratified, multistage method. Among the 101,316 participants in the 1999–2018 NHANES, those with (1) missing OA data (N=55,809), (2) missing WC or height data (N=4,643), and (3) missing covariable data (N=27,918) were excluded. Ultimately, 12,946 participants were enrolled (Fig. 1).

Definition of BRI and OA

We used the following equation to define BRI: $BRI = 364.2\text{-}365.5 \times (1\text{-}[WC(m)/2\pi]^2/[0.5 \times height(m)]^2)^{½}$ [19]. Additionally, OA diagnosis was self-reported; if participants responded in the affirmative to "Has a doctor or other health professional ever told you that you had arthritis?", they were next asked, "Which type of arthritis was it?". Participants could be classified as having OA depending on their answer to the second question.

Covariables

The following covariables were selected according to prior studies and clinical experience: (1) demographic data (age, sex, race, education level, marital status, and family poverty-income ratio), (2) questionnaire data (diabetes, hypertension, smoking [minimum of 100 cigarettes over one's lifetime], and alcohol status [consuming alcohol once a month or more], as well as moderate or vigorous activity), (3) laboratory data (albumin, alanine and aspartate aminotransferase, alkaline phosphatase, blood urea nitrogen, total calcium, phosphorus, total protein, uric acid, triglyceride, low- and high-density lipoprotein cholesterol, and total cholesterol), (4) dietary data (energy and protein take).

Statistical analysis

Complete data were accessible for all participants, after individuals with absent data about OA status and covariables, together with those for whom the BRI could not be computed from WC and height, were eliminated. An appropriate sample weight was calculated, and the analysis considered the complex multistage survey design procedures. Continuous and categorical variables are represented as weighted mean [95% confidence interval (CI)] and proportions (95% CI), respectively. To compare the BRI quartile groupings, either survey-weighted Chi-square or survey-weighted linear regression was used. Because of their potential to provide inaccurate inferential results, traditional regression approaches are unsuitable [20]. Particularly, the standard error and CI of parameter estimations might be drastically understated. Therefore, to determine the link between BRI and OA risk, weighted multivariable regression analysis (MVRA) was conducted. Models 1–3 progressively adjusted the covariables (details in Supplementary Material). The results were reported as an odd ratio (OR) and 95% CI. A trend test was used to enhance the reliability of the regression analysis results. Potential nonlinear associations were assessed by smooth curve fitting. Furthermore, subgroup analysis and interaction tests were deployed to identify associations in particular subgroups. Subgroup analysis was stratified by age, sex, race, education level, and moderate or vigorous activity, as well as marital, diabetes, hypertension, and smoking status.

A 7:3 ratio was adopted for the random division of the acquired data into training and validation sets. Least absolute shrinkage and selection operator (LASSO)

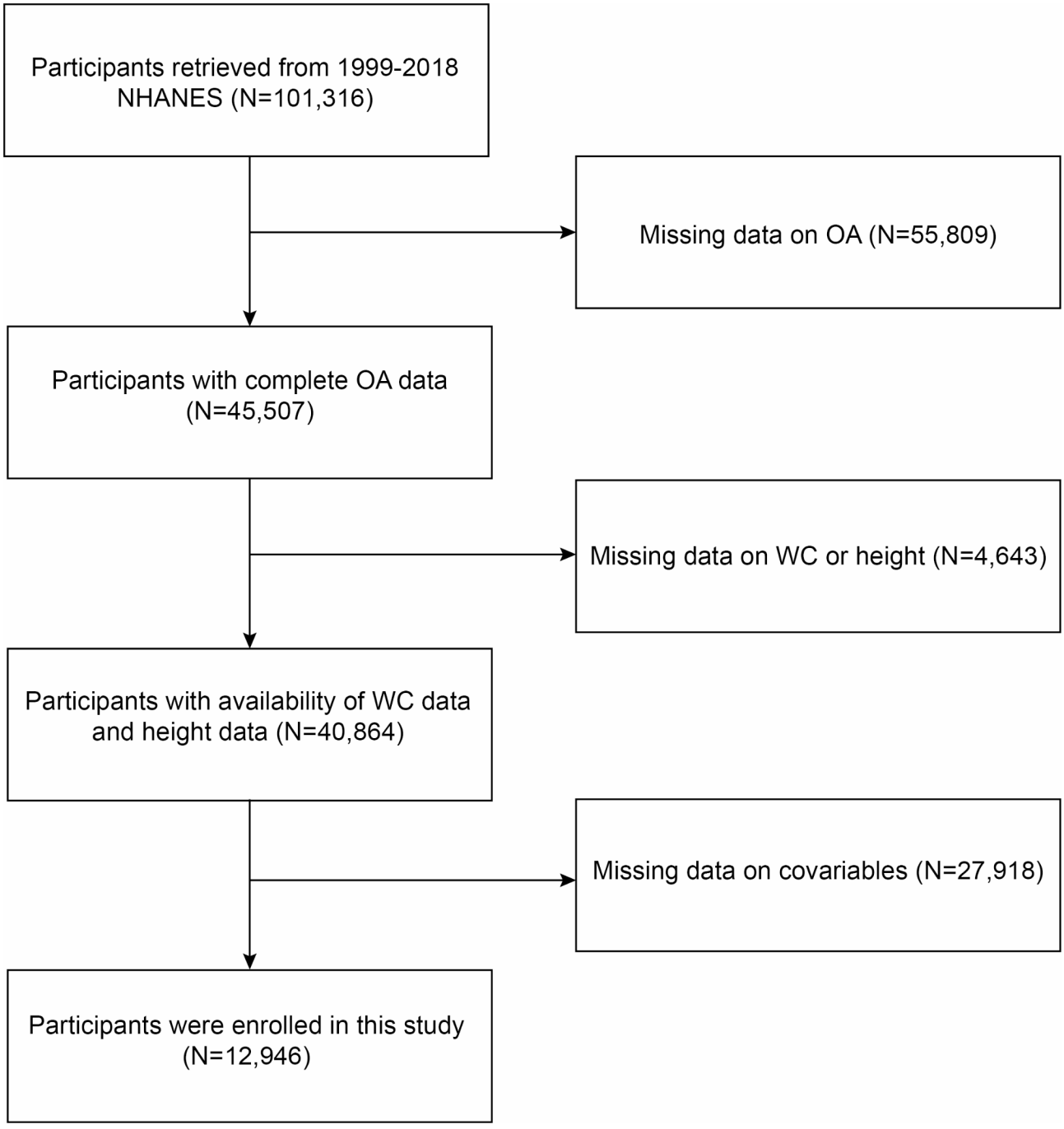

Fig. 1 Flowchart

regression is a linear regression technique utilizing lambda penalty coefficients to pick out variables that did not have 0 regression coefficients and eliminate those that did [21, 22]. The selected variables were regarded as the most relevant. LASSO regression along with MVRA were employed for the training set, aiming at identifying variables for a prediction model construction. Afterward, the model was visualized using the nomogram and assessed with the receiver operator characteristic (ROC) curve, calibration curve, and decision curve analysis (DCA). Finally, the validation set was utilized to validate the model. The statistical analyses were conducted utilizing R software (http://www.R-project.org) and EmpowerStats (www.empowerstats.com), considering $P<0.05$ as statistically significant.

Results

Baseline characteristics

Overall, 12,946 individuals were enrolled. Significant differences were observed among the BRI quartile groups in all investigated variables (all $P<0.05$), except for smoking status and aspartate aminotransferase (Table 1).

Association between BRI and OA risk

The weighted MVRA results indicated that a greater BRI was correlated with an elevated OA risk (Table 2). A statistically significant positive association was shown in models 1 (OR=1.23; 95% CI: 1.20–1.27; $P<0.0001$) and 2 (OR=1.18; 95% CI: 1.14–1.23; $P<0.0001$), which was consistent (OR=1.18; 95% CI: 1.13–1.23; $P<0.0001$) even after adjustment for all covariables. This indicates that OA risk increased by 18% with every unit rise in BRI. Then, BRI was regarded as a categorical variable and divided into quartiles for subsequent analysis. In comparison with the risk in quartile 1, OA risk increased in quartiles 2 (OR=1.58; 95% CI: 1.22–2.03; $P=0.0006$), 3 (OR=1.83; 95% CI: 1.40–2.40; $P<0.0001$) and 4 (OR=2.70; 95% CI: 1.99–3.66; $P<0.0001$). The risk of OA was exacerbated within increased BRI quartile groups (P-for-trend<0.0001). Furthermore, smooth curve fitting revealed no non-linear associations (Supplementary Fig. 1). Sensitivity analysis showed that BRI was preferred over BMI in predicting OA (area under the curve 0.652 vs. 0.594, $P<0.05$, Supplementary Fig. 2).

Subgroup analysis

To thoroughly identify the reliability of this positive association and identify any subpopulation differences, we conducted subgroup analysis and interaction tests. Subgroup analysis was stratified by age, sex, race, education level, and moderate or vigorous activity, as well as marital, diabetes, hypertension, and smoking status. Across all subgroups, individuals exhibiting elevated BRI demonstrated a continuously heightened risk of osteoarthritis (all OR>1 and all $P<0.05$, Supplementary Fig. 3). In age-stratified analysis, the OR between BRI and OA risk was 1.17 (95% CI: 1.12–1.23; $P<0.0001$) for individuals aged 50 years or less, whereas the OR was 1.17 (95% CI: 1.13–1.21; $P<0.0001$) for those aged older than 50 years. Additionally, in sex-stratified analysis, the OR between BRI and OA risk was 1.18 (95% CI: 1.13–1.23; $P<0.0001$) for male and 1.17 (95% CI: 1.13–1.21; $P<0.0001$) for female. However, none of the subgroups showed a statistically significant interaction (all $P>0.05$).

Selection of variables

In the training set, the factors from baseline characteristics, other than BRI, were subjected to LASSO regression to identify potential predictors of OA (Fig. 2). Nine variables (age, sex, education level, diabetes, hypertension, smoking status, blood urea nitrogen, phosphorus and total protein) were selected based on the lambda.1se from 10-fold cross-validation. The nine variables were incorporated into MVRA for further analysis (Table 3).

Nomogram development for risk prediction

After identifying the variables of interest, we constructed a prediction model, which was visualized using a nomogram. The constructed model for predicting the risk of OA included the following predictors: BRI, age, sex, education level, diabetes, hypertension, smoking status, blood urea nitrogen, phosphorus and total protein (Fig. 3). The score for each predictor was computed and subsequently aggregated to obtain a total score. A vertical line was extended downwards from the total score to reflect the corresponding OA risk, with a higher score reflecting heightened risk.

Prediction model evaluation and validation

An area under the ROC curve of 0.830 (95% CI: 0.818–0.842) showcased that the prediction model possessed favorable discriminatory power in the training set (Fig. 4A). The calibration curve, generated using 1,000 bootstraps, exhibited a strong alignment between actual and predicted probabilities, which suggested that the prediction model was accurate in the training set (Fig. 4B). As shown by the DCA, if the risk threshold was less than 0.58, the predictive model provided a net benefit in the training set (Fig. 4C). Furthermore, the prediction model also exhibited favorable discriminatory power, high accuracy, and potential clinical benefits in the validation set (Supplementary Fig. 4).

Discussion

In this cross-sectional study involving 12,946 individuals from the 1999–2018 NHANES, BRI was positively correlated to OA risk, which remained stable in different population settings. To predict the OA risk, a nomogram model was constructed using the following factors: BRI, age, sex, education level, diabetes, hypertension, smoking status, blood urea nitrogen, phosphorus and total protein. The model exhibited favorable discriminatory power, high accuracy, and potential clinical benefits in training and validation sets. These findings underscore the model's value for evaluating OA risk.

Like osteoporosis, OA is an escalating public health concern [23, 24]. Obesity constitutes a crucial risk factor for OA onset and progression [25–27]. Indeed, the association between BMI, a conventional measure of obesity, and OA has been extensively reported. Nevertheless, BMI is not capable of precisely reflecting fat distribution or differentiating between adipose and lean tissue [28]. The fact that people with comparable levels of adiposity (as measured by BMI) exhibit considerable variations of

Table 1 Weighted demographic baseline characteristics of the participants

Characteristics	Body roundness index				*P*-value
	Q1	Q2	Q3	Q4	
Age (years)	37.99 (37.27 ,38.71)	45.01 (44.29 ,45.74)	48.78 (48.11 ,49.46)	48.96 (48.16 ,49.76)	< 0.0001
Sex (%)					< 0.0001
Male	50.17 (48.03 ,52.30)	56.88 (54.42 ,59.30)	56.34 (54.07 ,58.57)	41.95 (39.93 ,44.00)	
Female	49.83 (47.70 ,51.97)	43.12 (40.70 ,45.58)	43.66 (41.43 ,45.93)	58.05 (56.00 ,60.07)	
Race (%)					< 0.0001
Hispanic	9.37 (8.06 ,10.88)	13.22 (11.48 ,15.18)	15.88 (13.84 ,18.15)	14.24 (12.23 ,16.51)	
Non-Hispanic white	72.69 (70.39 ,74.88)	72.61 (70.18 ,74.92)	70.32 (67.66 ,72.85)	70.21 (67.26 ,73.00)	
Non-Hispanic black	10.59 (9.30 ,12.05)	7.91 (6.83 ,9.13)	8.79 (7.63 ,10.10)	11.70 (10.09 ,13.54)	
Other	7.34 (6.36 ,8.46)	6.26 (5.29 ,7.40)	5.02 (3.96 ,6.33)	3.85 (2.97 ,4.98)	
Education level (%)					< 0.0001
< high-school	11.57 (10.00 ,13.35)	12.29 (10.95 ,13.78)	15.62 (14.00 ,17.38)	16.52 (14.87 ,18.31)	
High-school	19.44 (17.32 ,21.75)	22.66 (20.29 ,25.23)	25.39 (23.44 ,27.46)	25.28 (23.18 ,27.50)	
> high-school	68.99 (65.81 ,71.99)	65.04 (62.23 ,67.76)	58.99 (56.33 ,61.59)	58.20 (55.83 ,60.53)	
Marital status (%)					< 0.0001
Live with others	59.43 (56.96 ,61.85)	67.77 (65.39 ,70.07)	70.90 (68.52 ,73.17)	64.34 (61.88 ,66.74)	
Live alone	40.57 (38.15 ,43.04)	32.23 (29.93 ,34.61)	29.10 (26.83 ,31.48)	35.66 (33.26 ,38.12)	
Diabetes status (%)					< 0.0001
Yes	1.41 (0.99 ,2.01)	3.97 (3.22 ,4.88)	7.77 (6.74 ,8.95)	14.48 (13.02 ,16.08)	
No/Borderline	98.59 (97.99 ,99.01)	96.03 (95.12 ,96.78)	92.23 (91.05 ,93.26)	85.52 (83.92 ,86.98)	
Hypertension status (%)					< 0.0001
Yes	11.05 (9.77 ,12.47)	23.39 (21.47 ,25.43)	32.52 (30.40 ,34.73)	46.21 (43.79 ,48.64)	
No	88.95 (87.53 ,90.23)	76.61 (74.57 ,78.53)	67.48 (65.27 ,69.60)	53.79 (51.36 ,56.21)	
Smoking status (%)					0.0639
Yes	46.26 (43.59 ,48.95)	48.69 (46.44 ,50.94)	49.39 (47.01 ,51.77)	50.16 (47.82 ,52.50)	
No	53.74 (51.05 ,56.41)	51.31 (49.06 ,53.56)	50.61 (48.23 ,52.99)	49.84 (47.50 ,52.18)	
Moderate or vigorous activity (%)					< 0.0001
Yes	72.36 (70.02 ,74.59)	64.92 (62.47 ,67.29)	57.01 (54.55 ,59.44)	49.34 (46.84 ,51.83)	
No/Unable to do activity	27.64 (25.41 ,29.98)	35.08 (32.71 ,37.53)	42.99 (40.56 ,45.45)	50.66 (48.17 ,53.16)	
Alcohol status (%)					< 0.0001
Yes	71.20 (68.86 ,73.44)	68.16 (65.88 ,70.37)	60.54 (57.90 ,63.13)	48.84 (46.08 ,51.62)	
No	28.80 (26.56 ,31.14)	31.84 (29.63 ,34.12)	39.46 (36.87 ,42.10)	51.16 (48.38 ,53.92)	
Osteoarthritis presence					< 0.0001
Yes	5.21 (4.38 ,6.18)	10.65 (9.31 ,12.17)	14.46 (12.91 ,16.17)	20.91 (18.83 ,23.16)	
No	94.79 (93.82 ,95.62)	89.35 (87.83 ,90.69)	85.54 (83.83 ,87.09)	79.09 (76.84 ,81.17)	
Family poverty–income ratio	3.21 (3.11 ,3.31)	3.29 (3.20, 3.38)	3.13 (3.04 ,3.21)	2.89 (2.80, 2.99)	< 0.0001
Albumin (g/dL)	4.39 (4.37, 4.41)	4.31 (4.29, 4.32)	4.24 (4.22, 4.25)	4.09 (4.07, 4.11)	< 0.0001
Alanine transaminase (U/L)	21.70 (20.94 ,22.46)	26.47 (24.75 ,28.20)	27.65 (26.81 ,28.48)	28.51 (27.67 ,29.36)	< 0.0001
Aspartate aminotransferase (U/L)	24.27 (23.48 ,25.05)	25.24 (24.62 ,25.86)	25.50 (24.74 ,26.27)	25.26 (24.61 ,25.92)	0.0756
Alkaline phosphatase (U/L)	61.59 (60.61 ,62.57)	66.42 (65.45 ,67.39)	70.25 (69.08 ,71.42)	74.45 (72.95 ,75.95)	< 0.0001
Blood urea nitrogen (mg/dL)	12.44 (12.22 ,12.66)	13.33 (13.11, 13.56)	13.56 (13.35, 13.77)	13.58 (13.31 ,13.85)	< 0.0001
Total calcium (mg/dL)	9.44 (9.42, 9.46)	9.39 (9.38, 9.41)	9.36 (9.34, 9.38)	9.31 (9.28, 9.33)	< 0.0001
Phosphorus (mg/dL)	3.71 (3.68, 3.73)	3.61 (3.59, 3.63)	3.58 (3.56, 3.60)	3.60 (3.57, 3.62)	< 0.0001
Total protein (g/dL)	7.21 (7.18, 7.23)	7.15 (7.12, 7.17)	7.14 (7.11, 7.17)	7.07 (7.04, 7.09)	< 0.0001
Uric acid (mg/dL)	4.94 (4.88 ,4.99)	5.37 (5.32 ,5.43)	5.67 (5.60 ,5.74)	5.93 (5.86, 6.00)	< 0.0001
Triglyceride (mg/dL)	90.22 (87.95 ,92.48)	119.86 (116.80 ,122.92)	132.88 (129.03 ,136.74)	141.28 (137.45 ,145.11)	< 0.0001
Low-density lipoprotein cholesterol (mg/dL)	106.55 (105.01 ,108.09)	119.64 (118.14 ,121.15)	122.04 (120.49 ,123.60)	116.95 (115.29 ,118.62)	< 0.0001
High-density lipoprotein cholesterol (mg/dL)	60.64 (59.82 ,61.46)	54.39 (53.55 ,55.23)	51.41 (50.72 ,52.09)	48.86 (48.21 ,49.51)	< 0.0001
Total cholesterol (mg/dL)	185.22 (183.55 ,186.89)	198.01 (196.29 ,199.73)	200.03 (198.24 ,201.82)	194.08 (192.06 ,196.10)	< 0.0001
Energy take (kcal/day)	2293.38 (2254.53 ,2332.23)	2196.64 (2157.91 ,2235.38)	2185.39 (2146.42 ,2224.35)	2061.39 (2027.06 ,2095.71)	< 0.0001
Protein take(gm/day)	87.07 (85.40 ,88.73)	85.69 (83.93 ,87.44)	84.95 (83.28 ,86.62)	81.10 (79.76 ,82.44)	< 0.0001

Table 2 Association between BRI and OA risk

	Odd ratio (95% confidence interval), *P*-value		
	Model 1	**Model 2**	**Model3**
Continuous BRI	1.23 (1.20, 1.27) < 0.0001	1.18 (1.14, 1.23) < 0.0001	1.18 (1.13, 1.23) < 0.0001
Categories BRI			
Quartile 1	Reference	Reference	Reference
Quartile 2	2.17 (1.75, 2.69) < 0.0001	1.57 (1.24, 2.00) 0.0003	1.58 (1.22, 2.03) 0.0006
Quartile 3	3.08 (2.48, 3.82) < 0.0001	1.84 (1.43, 2.37) < 0.0001	1.83 (1.40, 2.40) < 0.0001
Quartile 4	4.81 (3.86, 5.99) < 0.0001	2.86 (2.24, 3.65) < 0.0001	2.70 (1.99, 3.66) < 0.0001
P-for-trend	< 0.0001	< 0.0001	< 0.0001

BRI, body roundness index

metabolic disturbance provides strong support for these phenomena [29, 30]. In addition, as the field of obesity research progressed, the obesity paradox was characterized [31–33]. The current evidence suggests that obesity may be protective in older people or people with certain specific diseases and is linked to a reduction in mortality, i.e., the obesity paradox [34]. The reasons for the obesity paradox are still unclear; however, the bulk of research observing the obesity paradox has used BMI as the sole measure of obesity, which may not provide sufficient accuracy [35]. Improving comprehension of alternations in body composition and fat distribution should enable the accurate prediction of the connection between obesity, disease, and death in older individuals [35–37]. It is important to note that WC and waist-to-hip ratio (WHR) are also commonly employed as markers of obesity. Still, WC or WHR do not allow for accurate differentiation between subcutaneous and visceral fat [38]. Lipid-accumulation-product levels could function as an important biomarker for validating obesity. A cross-sectional study involving 7,492 individuals identified that at levels below 120.00 cm × mmol/L, lipid-accumulation-product is a potential indicator of OA [30]. Recently, the BRI has been regarded as a reliable and innovative measure for assessing obesity, providing a more realistic reflection of body

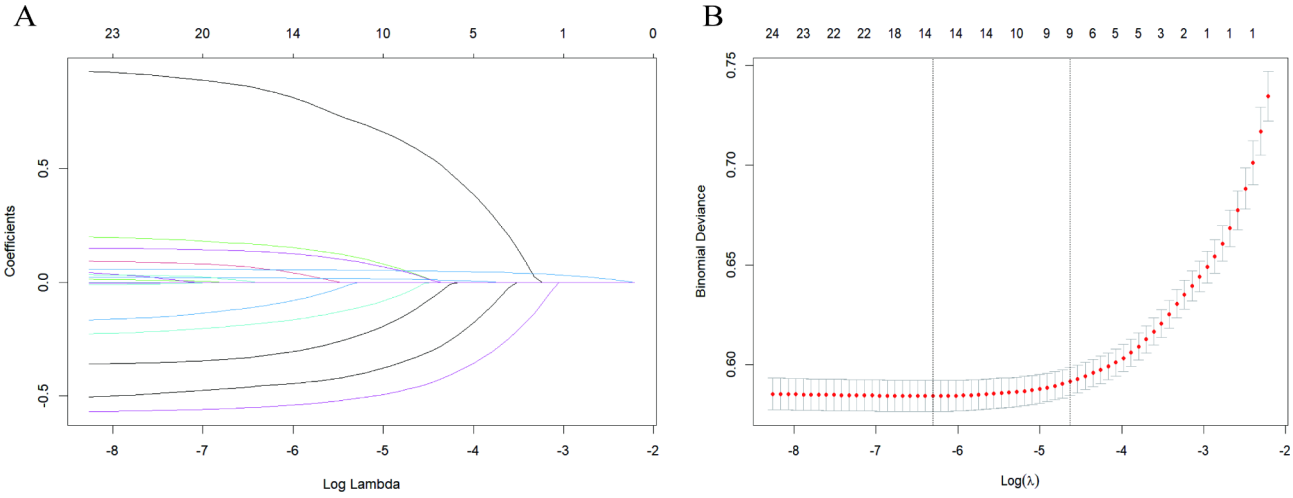

Fig. 2 Lasso regression for variables of interest: (**A**) regression coefficient profile diagram. (**B**) cross-validation curve

Table 3 MVRA for the variables of interest

	Estimate	Std. Error	z value	Odd ratio	95%CI-low	95%CI-upp	*P*-value
Intercept	-2.4254	0.6397	-3.7918	0.0884	0.0252	0.3098	0.0002
Age	0.0571	0.0026	21.9492	1.0588	1.0534	1.0642	< 0.0001
Sex (female)	0.8419	0.0783	10.7493	2.3208	1.9905	2.7059	< 0.0001
Education level (high-school)	0.1898	0.1091	1.7389	1.2090	0.9762	1.4973	0.0820
Education level (> high-school)	0.3855	0.0934	4.1261	1.4703	1.2243	1.7658	< 0.0001
Diabetes status (No/Borderline)	-0.2204	0.1025	-2.1499	0.8022	0.6562	0.9807	0.0316
Hypertension status (no)	-0.5759	0.0761	-7.5693	0.5622	0.4843	0.6526	< 0.0001
Smoking status (no)	-0.3804	0.0739	-5.1458	0.6836	0.5914	0.7902	< 0.0001
Blood urea nitrogen	0.0203	0.0060	3.3805	1.0205	1.0086	1.0326	0.0007
Phosphorus	0.1566	0.0686	2.2816	1.1695	1.0223	1.3379	0.0225
Total protein	-0.4958	0.0762	-6.5049	0.6091	0.5246	0.7072	< 0.0001

CI, confidence interval

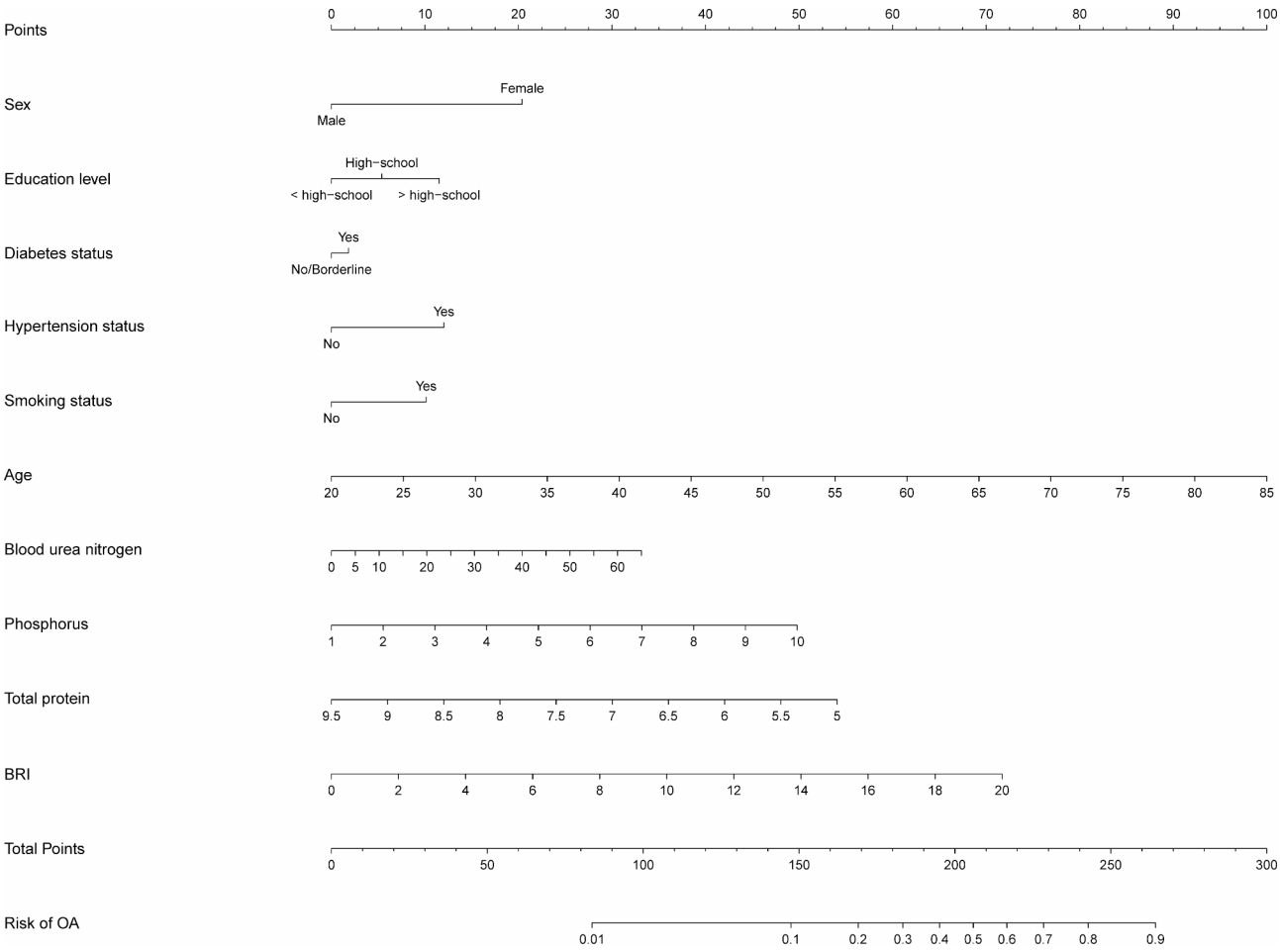

Fig. 3 Nomogram for predicting OA risk

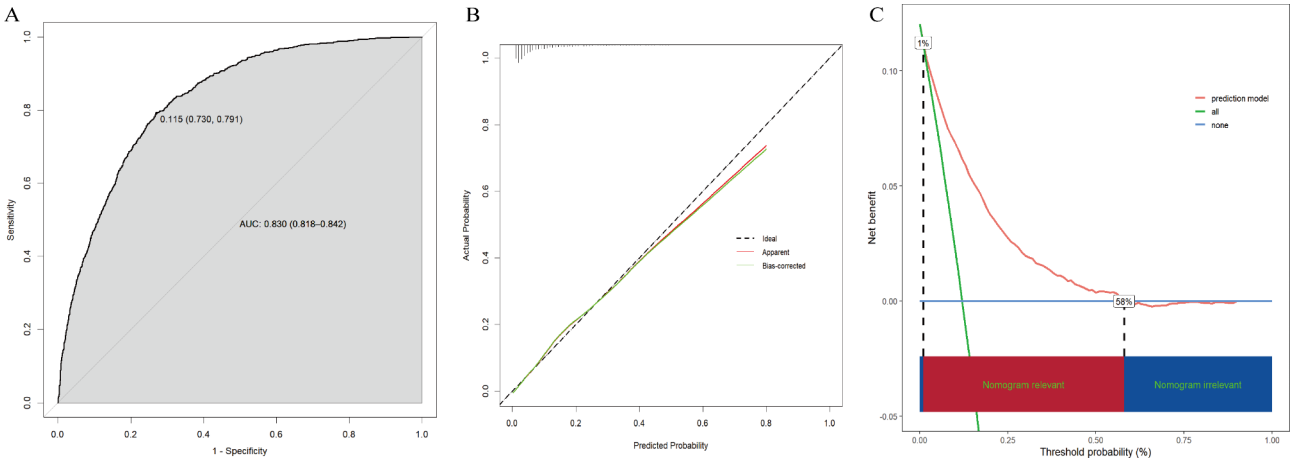

Fig. 4 Prediction model evaluation: (**A**) ROC curve. (**B**) calibration curve. (**C**) DCA

fat and visceral adipose tissue proportions through relatively easy computation [19].

The link between BRI and various diseases has been thoroughly investigated [39–46]. For example, Gao et al. reported that BRI exhibited a positive connection to colorectal cancer risk, and it was effective in risk prediction [39]. Additionally, Zhang et al. observed that BRI displayed a positive link with overactive bladder and that BRI exhibited a superior discriminatory capacity for overactive bladder compared to other anthropometric

parameters [40]. A prospective cohort study involving 13,209 participants reported that BRI trajectory is positively related to the occurrence of cardiovascular disease [41]. In the present study, BRI exhibited a positive connection with OA risk. As factors like age and sex influence the development of OA, we included them in the prediction model to improve the efficient assessment and management of high-risk OA groups.

The exact mechanism by which obesity can result in OA remains uncertain; however, mechanical factors might be partly liable for OA development. Indeed, obesity raises the mechanical loads on weight-bearing (WB) joints (including the knee and hip joints) [47, 48]. Moderate mechanical loading is beneficial for preserving articular cartilage homeostasis [49, 50]. Cartilage homeostasis is disrupted, and normal joint morphology is deformed, when abnormal excessive mechanical loading happens, thereby participating in OA development and advancement [51]. Several inflammatory pathways and related factors, such as IL-1β, TNF-α, and NF-κB, may be activated by the overloading of joints, resulting in irreversible degradation of the matrix and contributing to apoptosis [52–54]. Nevertheless, obesity is strongly related to OA in non-WB joints, including the hand [55, 56]. Considering only mechanical factors appears to be inadequate to account for the connection between obesity and OA occurrence in non-WB joints [50, 57]. Consequently, it has been speculated that adipose tissues are critical in this context. Adipose tissue-produced adipokines (leptin and adiponectin, among others) and inflammatory cytokines (IL-6 and TNF-α, among others) participate in cartilage degeneration, synovitis, and bone erosion, eventually resulting in OA [58, 59]. In addition, the degenerative process of OA is substantially influenced by obesity-related insulin resistance [60]. One possible explanation for how insulin resistance contributes to OA is the hyperglycemic state that occurs because of insulin resistance [54]. Hyperglycemia has been suggested to elevate inflammation, destroy subchondral bone, and cause chondrocyte dysfunction [61]. Moreover, by promoting the advanced glycation end-product formation and their buildup in articular cartilage, hyperglycemia creates an unhealthy environment that may facilitate the development of OA [62, 63]. Low-grade systemic inflammation, resulting from obesity-related gut dysbiosis, is being increasingly acknowledged as a contributing factor to OA [25, 64–66].

In the present study, following the adjustment for all covariables, OA risk increased by 18% with every unit rise in BRI. This finding provides insight into understanding the relationship between BRI and OA risk. Considering that BRI is a readily quantifiable and valuable indicator of obesity, it is advisable for the public to routinely measure BRI. For individuals with high BRI, effective obesity control, especially abdominal fat, may positively influence the prevention of OA. Moreover, sensitivity analysis showed that BRI was preferred over BMI in predicting OA (area under the curve 0.652 vs. 0.594, $P<0.05$). Doctors are advised to remain vigilant regarding individuals with a normal BMI but high BRI, as they may also experience high risk for OA. We developed an OA risk prediction model with favorable discriminatory power, high accuracy, and potential clinical benefits. Consequently, we encourage nurses to investigate BRI and other easily accessible factors in newly admitted patients. Then, according to the prediction model, OA risk in newly admitted patients can be assessed. Care for high-risk OA groups should be enhanced, and imaging should be done to further examine joint condition, if needed.

Our study possesses several advantages: For the first time, this study demonstrated a positive connection between BRI and OA risk. Furthermore, we classified the BRI into quartiles and conducted trend tests to confirm the reliability and precision of the data analyses. Finally, by using the BRI and other easily accessible factors, we developed a simple, rapid, and cost-effective predictive model to evaluate and manage high-risk OA groups. Nonetheless, realizing the limitations of our study is crucial. First, determination of whether participants had OA was based on self-reported interview questions, which could lead to recall bias and social desirability bias as well as affect the validity of our findings. Previous studies have also frequently used self-reported OA diagnoses because of the high concordance (85%) reported between self-reported and clinically confirmed OA cases [67–69]. However, future research should confirm our findings by precisely recognizing OA using clinical symptoms, physical examination, and imaging data. Second, because of constraints in the NHANES dataset, we disregarded a few factors that may influence OA, like occupational status, detailed medication use, and genetic factors. These factors might influence the relationship between BRI and OA risk. Moreover, these factors could hold predictive ability, but they were not included in our model. Our hospital plans to comprehensively investigate occupational status, detailed medication use, genetic factors, and other potential OA-related factors in our own database. Third, the cross-sectional design limited the definite determination of a causal connection between BRI and OA risk. Further studies should investigate the causal connection between BRI and OA risk through longitudinal or prospective study designs.

Conclusion

In this study, BRI was positively related to OA risk. Our predictive model demonstrated that combining BRI with other easily accessible factors could be helpful in assessing and managing high-risk OA groups. Considering that

BRI is a readily quantifiable and valuable indicator of obesity, it is advisable for the public to routinely measure BRI. For individuals with high BRI, effective obesity control, especially abdominal fat, may positively influence the prevention of OA. Further studies should investigate the causal connection between BRI and OA risk through longitudinal or prospective study designs. Additionally, our hospital plans to comprehensively investigate further OA-related factors in our own database, which could deepen the public's understanding of the relationship between BRI and OA risk, as well as further refine predictive models.

Supplementary Information

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Supplementary Material 1

Supplementary Material 2

Supplementary Material 3

Supplementary Material 4

Supplementary Material 5

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Author contributions
XDW and CX conceived and designed the study. XDW, ZJG, and CX collected the data and contributed to the design of the study. XDW, ZJG, MW, and CX prepared and revised the manuscript. All the authors have read and approved the final content of the manuscript.

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Data availability
The NHANES data for this study are publicly available and can be found at https://www.cdc.gov/nchs/nhanes/.

Declarations

Ethics approval and consent to participate
The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of the NCHS. Informed consent was obtained from all participants involved in the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

Abbreviations
osteoarthritis, OA; body mass index, BMI; body roundness index, BRI; waist circumference, WC; National Health and Nutrition Examination Survey, NHANES; confidence interval, CI; multivariable regression analysis, MVRA; odd ratio, OR; least absolute shrinkage and selection operator, LASSO; receiver operator characteristic, ROC; decision curve analysis, DCA; waist-to-hip ratio, WHR; weight-bearing, WB.

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