Original Research

Association of body roundness index with myocardial infarction in US adults: the Nhanes 2013-2018 cross-sectional study

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Abstract

Background Cardiovascular diseases (CVD) are intimately linked to obesity, and its mechanisms can cause metabolic abnormalities affecting multiple systems, which raises the risk of adverse effects on population health. It has been demonstrated that the Body Roundness Index (BRI), a quantitative assessment derived from human morphologic factors, is highly effective in determining the accumulation of visceral fat. There has yet insufficient data to suggest the possible biological relationship and clinical importance between BRI and myocardial infarction (MI), and previous research has not thoroughly examined the independent association between the two conditions. The current study, which was based on the National Health and Nutrition Examination Survey (NHANES) database, sought to provide a new theoretical foundation for cardiovascular disease risk stratification by revealing the dose-response relationship between BRI and MI and its potential modulation mechanism through a large-sample cross-sectional analysis. **Methods** This cross-sectional analysis utilized data from the NHANES database, including 13,243 U.S. adults aged 20 years and older. The main statistical approach employed was hierarchical logistic regression modeling. Firstly, to elucidate the relationship between BRI and MI, we constructed both univariate and multivariate models to screen for confounding factors. Furthermore, the Restricted Cubic Spline (RCS) model was implemented to validate the nonlinear relationship between BRI and MI events, and subgroup interaction effects tests were conducted to assess heterogeneity. Receiver Operating Characteristic (ROC) analysis was performed, and the area under the curve (AUC) was calculated and compared with the conventional obesity index, BMI, to evaluate the predictive performance of the BRI and determine its clinical diagnostic utility. **Results** Participants in the MI group had significantly higher BRI scores (6.5 vs 5.6) than those in the non-MI group, and the fully adjusted model in the multifactorial regression analyses additionally highlighted a significant negative association between BRI and MI (OR = 0.94, 95% CI: 0.91-0.98) and this relationship remained after transforming the BRI into a quartile-stratified analysis. Subgroup analyses also observed significant gender differences (p for interaction=0.04) and the strength of the association was significantly altered in patients with comorbid hypertension or diabetes mellitus. Furthermore, the analysis of ROC showed that BRI (AUC=0.621) was more accurate than BMI (AUC=0.544) in predicting the risk of developing MI, suggesting its potential clinical value as an independent predictor. **Conclusion** Our results demonstrate that BRI and myocardial infarction are significantly inversely correlated, and keeping an eye on BRI levels is crucial for lowering or preventing myocardial infarction risk. As a sensitivity factor to forecast the likelihood of myocardial infarction, BRI might be helpful.

Keywords: body roundness index, myocardial infarction, obesity, cross-sectional study, NHANES

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1. Introduction

One of the conditions with the greatest rates of morbidity and death in the world is myocardial infarction (MI) [1], whose pathophysiologic basis is the permanent necrosis of cardiomyocytes brought on by an abrupt interruption of coronary blood flow. The European Society of Cardiology (ESC) guidelines for 2023 state that acute coronary syndromes can be classified as either non-ST-segment elevation myocardial infarction (NSTEMI) or ST-segment elevation myocardial infarction (STEMI) [2]. Epidemiologic data indicate that approximately 70% of patients with STEMI have complete coronary occlusion. MI frequently begins with chest discomfort [3], which is defined by persistent posterior sternal soreness. Malignant arrhythmias or abrupt cardiac death are common complications that enhance the poor prognosis and greatly increase the degree of myocardial ischemia. Coronary atherosclerosis is one of the main pathogenic mechanisms of MI [4], and it is highly linked to a variety of risk factors, such as obesity, smoking, diabetes mellitus, hypertension, and disorders of lipid metabolism. These potential risks can accelerate the process of atherosclerosis by encouraging endothelial dysfunction and an inflammatory response. The high mortality rate and quick onset of myocardial infarction have made it an important concern for the public on a worldwide scale.

The prevalence and incidence of obesity have risen considerably worldwide in the past few decades [5-6], and obesity is a complicated multifactorial condition in which excessive fat buildup can have a detrimental effect on the body's health [7]. An imbalance in energy intake, genetic predisposition, and dysbiosis of the gut microbiota interact in a number of ways that contribute to obesity [5]. A popular metric for evaluating obesity is body mass index (BMI), and a person is considered obese if their BMI is ≥30 [6]. But it is becoming increasingly clear that BMI has limitations [8-11]: it is less sensitive to age, gender, and ethnicity; it ignores variation in body fat distribution; and it cannot differentiate between fat and muscle mass. Moreover, it has been demon-

strateed that an excessive distribution of fat, independent of BMI, is a precursor to coronary artery disease. In light of everything mentioned above, Thomas et al. [12] introduced the idea of the Body Roundness Index (BRI), which integrates waist circumference and height data to objectively evaluate the extent of abdominal fat accumulation. It emphasizes the biological significance of abdominal fat, which is frequently related to cardiovascular disease, diabetes mellitus, and hypertension, rather than adding weight parameters to the BRI formula [10,13]. In comparison to BMI, it offers a more comprehensive and precise representation of an individual's level of obesity and health. Higher BRI trajectories were linked to a higher risk of cardiovascular events, according to a new study based on longitudinal trajectory modeling [14]. This suggests that BRI may be utilized as a predictor of the onset of cardiovascular disease. A previous national cohort study [15] based on NHANES data revealed for the first time a U-shaped association between BRI and all-cause mortality, suggesting that BRI can better represent visceral fat distribution by integrating waist circumference parameters. In summary, we are able to clearly identify the connection between BRI and cardiovascular disease are interrelated [16]. Even though BRI's value in predicting the risk of cardiovascular disease is becoming more widely acknowledged, detailed research on its precise relationship to myocardial infarction events is still lacking. There is no evidence to clarify whether BRI can be used as an independent predictive marker for MI, and its potential mechanism has not been elucidated. Therefore, the present study tried to further investigate the strength and clinical significance of the association between BRI and the incidence of myocardial infarction using the NHANES data while offering a fresh viewpoint for improving the cardiovascular risk assessment system.

2. Methods

2.1 Description of the survey

The National Health and Nutrition Examination Survey (NHANES) is a national cross-sectional research program led by the Centers for Disease

Control and Prevention (CDC) that aims to systematically monitor the health and nutritional status of ambulatory residents in the United States. The survey utilizes a stratified multistage probability sampling methodology, with data updated every two years to ensure a nationally representative sample that covers a wide range of age, gender, race, and income group [17]. All data are anonymized and made public through the official website, allowing researchers to freely access and use them for epidemiological analysis, public health policy making and disease risk modeling. Its unique advantage is that it combines objective physical examination data with subjective behavioral reports, providing a high-quality evidence base for exploring the complex associations between environment, nutrition and disease.

2.2 Study Population

Data gathered by NHANES from the 2013–2018 cycle was used in this investigation. The study population was selected based on the following three criteria: (1) Adults aged 20 years and above; (2) A clear "yes" answer to the question "Have you been told by your doctor that you have heart disease? "In the cardiovascular disease module of the NHANES questionnaire; (3) complete waist circumference and height measurements must be provided.

2.3 Exposure Variables and Outcomes

The BRI was calculated by combining two crucial physical measurements, height (Body Height, BH) and waist circumference (Waist Circumference, WC). These measurements were obtained by professionally trained medical technicians at the Mobile Examination Center (MEC).The formula for the BRI is as follows: BRI = 364.2 - 365.5 × $\sqrt{[1 - (WC/(2\pi))^2/(0.5 \times BH)^2]}$ [18], where the units of WC and BH are centimeters (cm) .Based on previous literature, it is known that higher BRI values indicate higher visceral fat accumulation, and in this study, a quartile grouping strategy (Q1-Q4) was used to transform continuous BRI into an ordered categorical variable in order to analyze its relationship with myocardial infarction [19-20].In this study, the definition of coronary artery disease was derived from the

statement, "You were told by your doctor that you have coronary artery disease" [21]. Answering the question "Were you told by your doctor that you have heart disease?" Participants who answered "yes" were considered to have MI.

2.4 Covariates

In order to consider more comprehensively the possible influences on the relationship between BRI and MI, and with reference to previous literature, in the present study we screened a variety of covariates [19,20] such as demographic characteristics, lifestyle factors, health status, and laboratory test results from a multidimensional perspective. Demographic characteristics included age, sex (male or female), race (including Mexican American, non-Hispanic white, non-Hispanic black, and other races), poverty-to-income ratio (PIR), and educational attainment (ranging from less than high school to college and beyond). Physical examination data included body mass index (BMI), waist circumference (WC), and height (Height). Questionnaire data included smoking status (rated by having smoked at least 100 cigarettes in your lifetime), and physical activity (rated by whether your job involves moderate-intensity activities that result in small increases in breathing or heart rate). Health status variables included chronic kidney disease, hypertension, hypercholesterolemia, and diabetes, as determined by physician diagnostic records or self-report. Laboratory tests included fasting glucose (FPG), triglycerides (TG), total cholesterol (TC) and high-density lipoprotein cholesterol (HDL-C), blood uric acid (SUA), alanine aminotransferase (ALT), aspartate aminotransferase (AST), and white blood cell count. All data were obtained through standardized questionnaires, physical examinations, and laboratory tests collected by licensed registered healthcare professionals. Questionnaires were administered through face-to-face interviews to ensure that participants provided accurate responses relevant to the study. All measurement tools were tested for reliability and validity, and the specific measurement protocols and procedures are detailed on the official

NHANES website.

2.5 Statistical analysis

Descriptive analyses based on myocardial infarction status were performed after identifying participants who met the inclusion criteria. Continuous variables were expressed as means (standard deviation) and categorical variables as percentages. Logistic regression analysis was constructed and three models based on increasing and decreasing covariates were built to calculate the MI risk ratio (OR) and 95% confidence intervals (CI) corresponding to each 1 standard deviation (SD) increase in BRI. To verify the robustness of the results, the continuous BRI was discretized by quartiles (Q1-Q4), and the trend of association between different BRI intervals was analyzed by using the trend test (P for trend). Meanwhile, RCS analysis (number of nodes = 3) was utilized to explore the nonlinear association between BRI and MI. Further subgroup interaction effect analyses based on demographic and lifestyle factors were performed to identify potential effect modifiers between BRI and MI. Finally, to confirm that BRI was more accurate than BMI in predicting the incidence of MI, ROC was performed, with all data analyses using $p < 0.05$ as the threshold for a statistically significant difference. The data analysis was performed using R Statistical Platform (version 4.2) and Xiantao Academic Tools.

3. Results

3.1 Characterization of the study population

Initially, data from 36,379 individuals were retrieved from the NHANES database for this investigation. Following a multi-phase screening procedure (Figure 1), 13,243 qualified samples were identified, encompassing 12,691 (95.83%) in the non-MI group and 552 (4.17%) in the MI group. Baseline characterization (Table 1) revealed that subjects in the MI group were primarily older males (59.2%), non-Hispanic whites (51.8%), and frequently presented with comorbid conditions such as hypertension and diabetes mellitus compared to the non-MI group. Furthermore, subjects in the MI group exhibited elevated levels of waist circumference, fasting glucose, uric acid, and WBC counts, as well as reduced levels of TC and HDL (P < 0.001), and notably, moderate physical activity levels, ALT, AST, and triglyceride levels did not appear to significantly impact the risk of myocardial infarction.
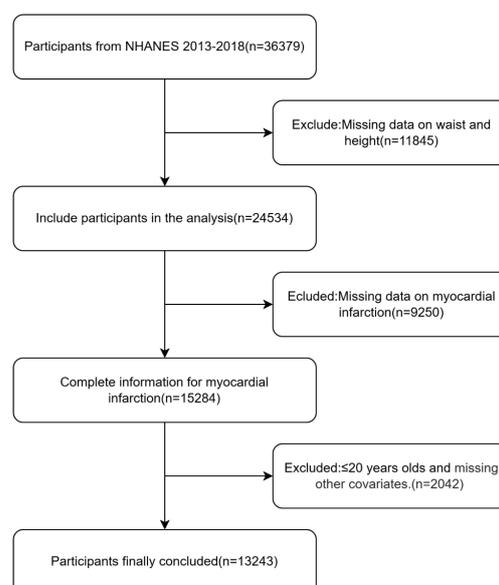

Figure 1 Flow chart of study participants.

Table 1 Baseline characteristics of participants

Variables	Total (n = 13243)	MI (n = 552)	non-MI (n = 12691)	P-value
Age (years)				<0.001
<65	10313 (77.9)	225 (40.8)	10088 (79.5)	
>65	2930 (22.1)	327 (59.2)	2603 (20.5)	
Gender (%)				<0.001
Male	6412 (48.4)	368 (66.7)	6044 (47.6)	
Female	6831 (51.6)	184 (33.3)	6647 (52.4)	
Race (%)				<0.001
Mexican American	1864 (14.1)	54 (9.8)	1810 (14.3)	

Non-Hispanic White	5095 (38.5)	286 (51.8)	4809 (37.9)	
Non-Hispanic Black	2795 (21.1)	106 (19.2)	2689 (21.2)	
Others	3489 (26.3)	106 (19.2)	3383 (26.7)	
Education level (%)				<0.001
Under high school	2661 (20.1)	156 (28.3)	2505 (19.7)	
High school	3020 (22.8)	147 (26.6)	2873 (22.6)	
Above high school	7562 (57.1)	249 (45.1)	7313 (57.6)	
PIR	2.5 ± 1.6	2.1 ± 1.5	2.5 ± 1.6	<0.001
Activity status (%)				0.077
Yes	5179 (39.1)	196 (35.5)	4983 (39.3)	
No	8064 (60.9)	356 (64.5)	7708 (60.7)	
Smoking (%)				<0.001
Yes	5675 (42.9)	361 (65.4)	5314 (41.9)	
No	7568 (57.1)	191 (34.6)	7377 (58.1)	
Hypertension (%)				<0.001
Yes	4711 (35.6)	402 (72.8)	4309 (34)	
No	8532 (64.4)	150 (27.2)	8382 (66)	
Hypercholesterolemia (%)				<0.001
Yes	4518 (34.1)	374 (67.8)	4144 (32.7)	
No	8725 (65.9)	178 (32.2)	8547 (67.3)	
Diabetes (%)				<0.001
Yes	1861 (14.1)	226 (40.9)	1635 (12.9)	
No	11382 (85.9)	326 (59.1)	11056 (87.1)	
CKD (%)				<0.001
Yes	434 (3.3)	68 (12.3)	366 (2.9)	
No	12809 (96.7)	484 (87.7)	12325 (97.1)	
FPG (mg/dL)	111.8 ± 36.8	121.6 ± 47.9	111.4 ± 36.2	<0.001
TC (mmol/L)	189.4 ± 41.7	172.6 ± 43.6	190.1 ± 41.5	<0.001
TG (mg/dL)	96.0 (64.0, 143.0)	99.0(66.0,143.0)	95.0 (64.0, 142.0)	0.355
HDL-C (mg/dL)	53.5 ± 16.5	49.2 ± 15.6	53.7 ± 16.5	<0.001
SUA (mg/dL)	5.4 ± 1.5	5.8 ± 1.5	5.4 ± 1.4	<0.001
WBC (×10^9/L)	7.4 ± 4.3	7.8 ± 5.3	7.3 ± 4.2	0.011
AST (U/L)	21.0 (18.0, 26.0)	21.0 (18.0, 26.2)	21.0 (18.0, 26.0)	0.243
ALT (U/L)	19.0 (15.0, 27.0)	20.0 (15.0, 27.0)	19.0 (15.0, 27.0)	0.577
Height (cm)	166.8 ± 10.1	167.9 ± 9.4	166.8 ± 10.1	0.015
BMI (kg/cm2)	29.3 ± 7.0	30.3 ± 7.4	29.3 ± 7.0	<0.001
WC (cm)	99.9 ± 16.8	107.1 ± 16.5	99.6 ± 16.8	< 0.001
BRI	5.6 ± 2.4	6.5 ± 2.4	5.6 ± 2.4	<0.001

Mean (SD) for continuous variables, % for categorical variables. PIR, Poverty income ratio; CKD: Chronic kidney disease: Fasting plasma glucose;

TC: Total cholesterol; TG: Triglyceride; HDL-C: High-density lipoprotein cholesterol; SUA: Serum uric acid; WBC: White blood cell count; AST:

Aspartate Aminotransferase; ALT: Alanine Aminotransferase; BMI: Body mass index; WC: Waist Circumference; BRI: Body roundness index.

3.2 Relationship between BRI and prevalence of myocardial infarction

Logistic regression analysis was employed to evaluate the predictive impact of BRI on MI, with findings presented in Table 2. The results indicated a negative correlation between BRI and MI prevalence in Model 1. Following sequential covariate adjustments, BRI maintained significant predictive capacity and a robust association with MI risk, demonstrating stable results. Further adjustment for all variables revealed a 6% reduction in MI prevalence for each unit increase in BRI (OR = 0.94, 95% CI: 0.91-0.98). Categorization of BRI demonstrated a substantial decrease in MI prevalence with increasing BRI quartiles compared to the lowest quartile (test of trend < 0.001). This trend persisted after adjusting for all covariates, indicating a strong negative association between BRI and MI. RCS regression analysis, as depicted in Figure 2, further revealed a nonlinear association between BRI and MI risk (p<0.05 for nonlinearity test). The risk of MI increased sharply with decreasing BRI levels, particularly below 5.2 (Table 3), with the curve flattening at higher BRI levels, suggesting a threshold effect of BRI on MI prediction. Low BRI levels may serve as an early warning indicator for cardiovascular events.

Table 2 BRI: Body roundness index

Exposure	Model 1 OR (95%CI), P-value	Model 2 OR (95%CI), P-value	Model 3 OR (95%CI), P-value
BRI (continuous)	0.88 (0.85 ~ 0.90) <0.001	0.86 (0.83 ~ 0.89) <0.001	0.94 (0.91 ~ 0.98), 0.007
BRI (quartile)			
Q1	[Reference]	[Reference]	[Reference]
Q2	0.50 (0.36 ~ 0.68), <0.001	0.63 (0.46 ~ 0.87), 0.005	0.78 (0.55 ~ 1.09), 0.140
Q3	0.39 (0.29 ~ 0.53), <0.001	0.56 (0.41 ~ 0.76), <0.001	0.87 (0.62 ~ 1.22), 0.411
Q4	0.27 (0.20 ~ 0.36), <0.001	0.32 (0.23 ~ 0.43), <0.001	0.63 (0.45 ~ 0.88), 0.007
P for trend	<.001	<.001	0.005

OR: Odds Ratio; CI: Confidence Interval. Model 1: No covariates adjusted; Model 2: Adjusted for Age, Sex, and Race; Model 3: Adjusted for Age, Gender, Race, Educational level, PIR, Activity status, Smoking, Hypertension, Hypercholesterolemia, Diabetes, CKD, Diabetes, FPG, TC, TG, HDLC, SUA, WBC, AST, ALT.
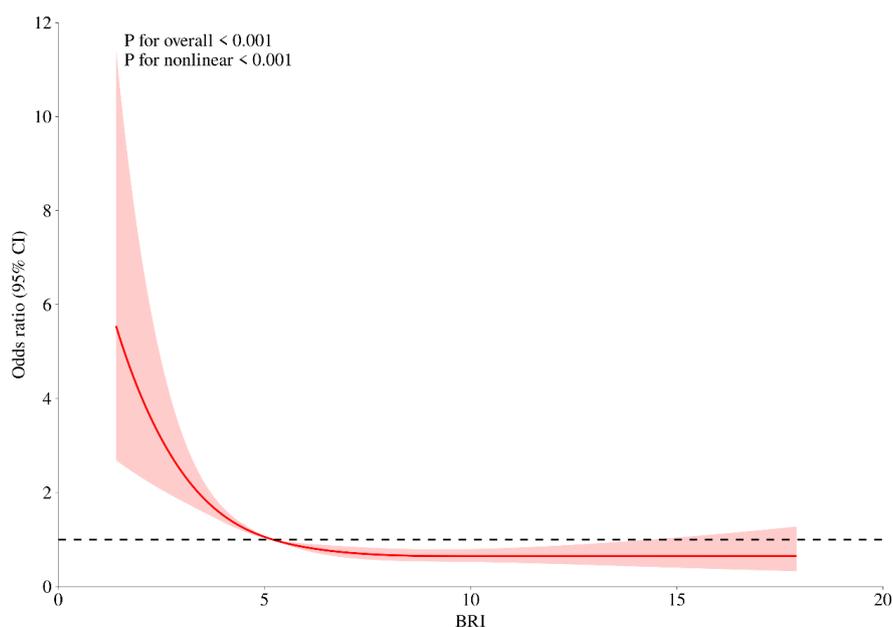

Figure 2 Restricted cubic spline for the association between the Body roundness index (BRI) and myocardial infarction.

Table 3 Analysis of the BRI saturation effect and myocardial infarction.

	BRI （%)	OR (95%CI) p-value
Myocardial Infarction	standard linear model	0.88 (0.85 - 0.90) <.001
	BRI < 5.10	0.61 (0.50 - 0.74) <.001
	BRI ≥5.10	0.95 (0.90 - 0.99) 0.015
	Log-likelihood ratio test	<.001

3.3 Subgroup analysis

Subgroup analyses and interaction tests were conducted to evaluate the association between BRI and MI across various subgroups, including gender, age, race, education, smoking status, physical activity, diabetes mellitus, hypertension, hypercholesterolemia, and chronic kidney disease (Figure 3). The results revealed that the impact of BRI on myocardial infarction varied across different populations. Specifically, the effect differed within the gender and age groups (<65 years: OR = 0.86, 95% CI: 0.82–0.89; ≥65 years: OR = 0.94, 95% CI: 0.89–0.99), as well as in those with hypercholesterolemia (yes: OR = 0.92, 95% CI: 0.88–0.97; no: OR = 0.86, 95% CI: 0.82–0.90) and diabetes mellitus (yes: OR = 0.97, 95% CI: 0.92–1.02; no: OR = 0.90, 95% CI: 0.87–0.94). These results suggest a significant interaction between sex, age, hypercholesterolemia, diabetes mellitus, and the association of BRI with myocardial infarction (interaction P < 0.05). Conversely, no significant interaction was observed between moderate activity, smoking, hypertension, chronic kidney disease, education, and the effect of BRI (interaction P > 0.05).
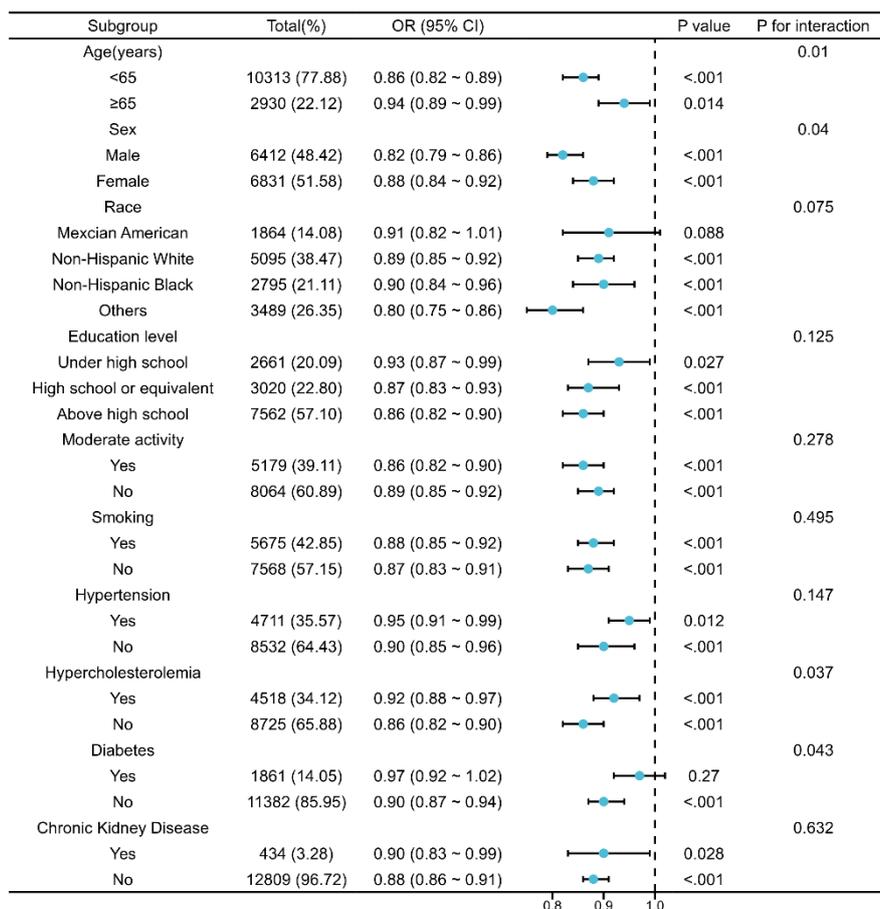
Subgroup	Total(%)	OR (95% CI)		P value	P for interaction
Age(years)					0.01
<65	10313 (77.88)	0.86 (0.82 ~ 0.89)		<.001	
≥65	2930 (22.12)	0.94 (0.89 ~ 0.99)		0.014	
Sex					0.04
Male	6412 (48.42)	0.82 (0.79 ~ 0.86)		<.001	
Female	6831 (51.58)	0.88 (0.84 ~ 0.92)		<.001	
Race					0.075
Mexcian American	1864 (14.08)	0.91 (0.82 ~ 1.01)		0.088	
Non-Hispanic White	5095 (38.47)	0.89 (0.85 ~ 0.92)		<.001	
Non-Hispanic Black	2795 (21.11)	0.90 (0.84 ~ 0.96)		<.001	
Others	3489 (26.35)	0.80 (0.75 ~ 0.86)		<.001	
Education level					0.125
Under high school	2661 (20.09)	0.93 (0.87 ~ 0.99)		0.027	
High school or equivalent	3020 (22.80)	0.87 (0.83 ~ 0.93)		<.001	
Above high school	7562 (57.10)	0.86 (0.82 ~ 0.90)		<.001	
Moderate activity					0.278
Yes	5179 (39.11)	0.86 (0.82 ~ 0.90)		<.001	
No	8064 (60.89)	0.89 (0.85 ~ 0.92)		<.001	
Smoking					0.495
Yes	5675 (42.85)	0.88 (0.85 ~ 0.92)		<.001	
No	7568 (57.15)	0.87 (0.83 ~ 0.91)		<.001	
Hypertension					0.147
Yes	4711 (35.57)	0.95 (0.91 ~ 0.99)		0.012	
No	8532 (64.43)	0.90 (0.85 ~ 0.96)		<.001	
Hypercholesterolemia					0.037
Yes	4518 (34.12)	0.92 (0.88 ~ 0.97)		<.001	
No	8725 (65.88)	0.86 (0.82 ~ 0.90)		<.001	
Diabetes					0.043
Yes	1861 (14.05)	0.97 (0.92 ~ 1.02)		0.27	
No	11382 (85.95)	0.90 (0.87 ~ 0.94)		<.001	
Chronic Kidney Disease					0.632
Yes	434 (3.28)	0.90 (0.83 ~ 0.99)		0.028	
No	12809 (96.72)	0.88 (0.86 ~ 0.91)		<.001	

Figure 3 Subgroup analysis of the association between BRI and myocardial infarction.

3.4 BRI as a predictor of myocardial infarction

Figure 4 shows the area under the curve (AUC) of diagnostic power in the prediction of myocardial infarction. These results indicate that both BMI and

BRI exhibit statistically significant diagnostic capabilities for identifying myocardial infarction (AUC > 0.5). Additionally, BRI outperformed BMI (AUC = 0.621).
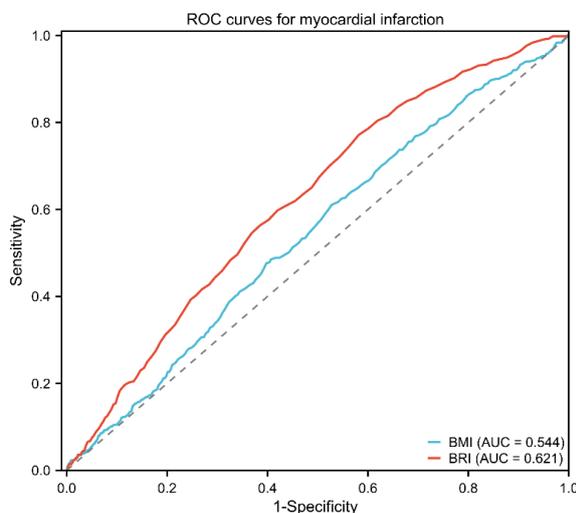

Figure 4 ROC curves of anthropometric indicators to predict high MI risk.

4. Discussion

This cross-sectional study used data from the NHANES 2013–2018 to rigorously examine the correlation between BRI and the incidence of MI in 13,243 U.S. adults. The findings demonstrated a strong and negative correlation between BRI and MI risk, which held true regardless of other cardiovascular risk factors and conventional measures of obesity, such as waist circumference and BMI. In particular, with every unit increase in BRI, the risk of MI fell by 6% (OR = 0.94, 95% CI: 0.91-0.98). This connection was more powerful among men, elderly people (>65), and non-Hispanic whites. In order to examine the relationship's relevance at various levels, subgroup analyses were carried out. All of these results imply that a lower BRI corresponds to a higher risk of MI and could be a more reliable cardiovascular risk assessment tool. Finally, ROC confirmed that BRI was a better predictor of myocardial infarction than BMI.

As an upcoming body fat distribution index, BRI may accurately measure the level of visceral adipose tissue (VAT) accumulation since its formula incorporates data on waist circumference and height. In the present study, we found that the predictive validity of BRI for MI was superior to that of BMI, which may be attributed to its superiority in identifying VAT-specific pathologic effects [22]. Although many previous studies have used BMI as an indicator of obesity to predict the risk of multiple cardiovascular diseases, its inability to differentiate between the heterogeneity of visceral and subcutaneous fat is noteworthy [23].Because VAT has been shown to significantly increase the risk of CVD through multiple pathological mechanisms centered on metabolic activation of visceral adipose tissue, especially abdominal fat, and its abnormal secretory function. Firstly, VAT exacerbated the generation of pro-inflammatory cytokines like IL-6 and TNF-α while suppressing the production of lipocalin, causing endothelial dysfunction and systemic inflammation. Secondly, the portal circulation's concentration of free fatty acids (NEFA) was higher, aggravating hepatic insulin resistance and ectopic lipid deposition, which accelerated the formation of atherosclerotic plaque. Finally, lipotoxic substances from visceral fat diffuse to the myocardium and vascular wall, leading to myocardial lipid peroxidation, decreased vascular elasticity, and collagen deposition [24-25].In addition, VAT increases the expression of plasminogen activator inhibitor-1 (PAI-1), which inhibits fibrinolysis and promotes pathologic thrombosis, further amplifying the cardiovascular risk [26]. Compared with BMI, BRI captures the pathologic effects of VAT more precisely because it directly reflects abdominal fat distribution. An observational study and Mendelian randomization analysis [27] using the NHANES database verified a significant positive and causal association between hypertension and VAT. Meanwhile, other cross-sectional analysis with 31,535 US adults [21] showed that the Weight-adjusted Waist Index (WWI) was positively associated with the risk of MI (OR=1.15, 95% CI:1.09-1.22), and its predictive efficacy was better than that of BMI. All of the above studies have supported the importance of rationally quantifying visceral adiposity in predicting CVD risk, and the present study utilized the BRI to further validate this theory, suggesting that the

quantification of visceral adiposity distribution is central to risk assessment.

It is known that obese patients are often associated with multiple underlying diseases [5,13,28], and timely identification of intervenable risk factors associated with myocardial infarction can be effective in improving myocardial infarction survival [23]. There is a significant pathophysiologic synergy between central obesity and Metabolic Syndrome (MetS), which increases the risk of death in patients with MI.A meta-analysis by Zahra et al. [29] found that metabolic syndrome increased the risk of MI in overweight patients by 1.68 times. Obesity has a significant impact on the prognosis of the metabolic syndrome [29], and studies have reported that [30] adopting an "obesity-centered" definition greatly increases the prevalence of MetS in patients with acute myocardial infarction. Similarly, metabolically healthy obese individuals are at higher risk for MI [31]. In a case-control study of 27,000 participants (including 15,152 patients with a first myocardial infarction) from 52 countries, Yusuf and colleagues [32] showed that the risk of MI increased 37% for every 1 standard deviation increase in Waist-to-Hip Ratio (WHR) (SD=0.085), and the association was consistent across race. Similarly in a cross-sectional study involving 31,535 US adults [21], WWI was found to be positively associated with myocardial infarction, and its predictive value for myocardial infarction was superior to that of waist circumference and body mass index, explaining the superior predictive value of internal adiposity for the prediction of disease, and the BRI was used as a more refined indicator of abdominal obesity in the present study to further quantify the risk contribution, which side-steps the BRI's ability to provide an internal obesity excellent degree of certainty about the degree of visceral obesity. Precisely, the findings of the present study emphasize the importance of body fat distribution (rather than weight dependence) in cardiovascular risk assessment.

5. Limitations

A significant correlation between BRI and myocardial infarction has not been found among other factors, despite the fact that this study found that older adults over 65, particularly those with hypercholesterolemia and diabetes mellitus, had MI and that those with lower BRI were at risk for adverse cardiovascular events. This could be because of confounding variables that were not measured and the sample's limited representativeness (non-Hispanic Blacks made up only 21.1% of the total) There are no noteworthy correlations between BRI and myocardial infarction, among other variables. Second, the NHANES data relied on patients' self-reported history of MI, which could be prone to recall bias; In particular, for underlying conditions like diabetes mellitus and hypertension, bias might have been introduced by the presence of other undiagnosed conditions. To reduce bias from self-reporting, future research ought to encompass objective data including imaging or biomarkers (like troponin). Finally, the current BRI formula does not take into account the skeletal muscle mass index and dietary patterns, which may affect the accuracy of the results. Future studies should explore how these potential confounders intervene in the relationship between the two for a more comprehensive risk assessment. In addition to this, studies linking BRI to the prevalence of myocardial infarction are still limited, and a large number of clinical studies are needed to confirm its validity and generalizability.

6. Conclusion

According to the study's findings, the relationship between BRI and MI was not statistically significant in non-Hispanic black and Mexican-origin groups, but it was in non-Hispanic whites (OR=0.88, 95% CI:0.83-0.93). This discrepancy may result from racial differences in fat distribution: Hispanic metabolic risk may be more genetically driven, while African-Americans have a higher subcutaneous fat storage capacity because of genetic evolutionary adaptations. However, this implies that cross-ethnic cohort studies are necessary to further evaluate the clinical application of BRI in the future.

In this study, contrary to the conventional belief that

obesity increases cardiovascular risk, our multivariate model revealed that higher BRI were independently associated with lower MI risk after adjusting for multiple confounding factors (OR = 0.94, 95% CI: 0.91–0.98). This finding resonates with the "obesity paradox" observed in specific populations, such as those with chronic heart failure or stable coronary artery disease. Our study is the first to link this phenomenon to BRI, an indicator that focuses more on central obesity and body shape. This suggests that when assessing cardiovascular risk, the body's "shape" or fat distribution may contain more complex and sometimes even protective physiological information than simple weight or BMI. This prompts us to re-examine the relationship between body composition and cardiovascular health [33].

The outcomes give methods for addressing obesity an evidence-based foundation. Lowering BRI with pharmacologically targeted therapies [34] or lifestyle interventions may be an effective way to prevent MI as BRI represents a modifiable visceral distribution profile. The application of GLP-1 receptor agonists to help diabetic patients lose weight was found to significantly reduce their risk of adverse cardiovascular events in the major GRADE clinical trial [35]. It makes sense to assume that BRI metrics can be expanded upon and put into models for assessing cardiovascular risk (such as ASCVD risk scores).

Reference

[1] Zheng D, Cao L. Association between myocardial infarction and triglyceride-glucose index: A study based on NHANES database. *Glob Heart* (2024) 19:23. doi: 10.5334/gh.1303

[2] Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, Claeys MJ, Dan G-A, Dweck MR, Galbraith M, et al. 2023 ESC guidelines for the management of acute coronary syndromes. *Eur Heart J* (2023) 44:3720–3826. doi: 10.1093/eurheartj/ehad191

[3] Arora G, Bittner V. Chest pain characteristics and gender in the early diagnosis of acute myocardial infarction. *Curr Cardiol Rep* (2015) 17:5. doi: 10.1007/s11886-014-0557-5

[4] Vazzana N, Santilli F, Sestili S, Cuccurullo C, Davi G. Determinants of increased cardiovascular disease in obesity and metabolic syndrome. *Curr Med Chem* (2011) 18:5267–5280. doi: 10.2174/092986711798184299

[5] Lin X, Li H. Obesity: Epidemiology, pathophysiology, and therapeutics. *Front Endocrinol (Lausanne)* (2021) 12:706978. doi: 10.3389/fendo.2021.706978

[6] Blüher M. Obesity: Global epidemiology and pathogenesis. *Nat Rev Endocrinol* (2019) 15:288–298. doi: 10.1038/s41574-019-0176-8

[7] Simoons ML, Bonneux L. Obesity, cardiology, and beyond. *J Am Coll Cardiol* (2008) 52:986–987. doi: 10.1016/j.jacc.2008.05.052

[8] Sánchez E, Sánchez M, Betriu À, Rius F, Torres G, Purroy F, Pamplona R, Ortega M, López-Cano C, Hernández M, et al. Are obesity indices useful for detecting subclinical atheromatosis in a middle-aged population? *Obesity Facts* (2020) 13:29. doi: 10.1159/000502696

[9] Chang Y, Guo X, Li T, Li S, Guo J, Sun Y. A body shape index and body roundness index: Two new body indices to identify left ventricular hypertrophy among rural populations in northeast China. *Heart Lung Circ* (2016) 25:358–364. doi: 10.1016/j.hlc.2015.08.009

[10] Schweitzer K. Could the body roundness index

one day replace the BMI? *JAMA* (2024) doi: 10.1001/jama.2024.20115

[11] Curtis JP, Selter JG, Wang Y, Rathore SS, Jovin IS, Jadbabaie F, Kosiborod M, Portnay EL, Sokol SI, Bader F, et al. The obesity paradox: Body mass index and outcomes in patients with heart failure. *Arch Intern Med* (2005) 165:55–61. doi: 10.1001/archinte.165.1.55

[12] Thomas DM, Bredlau C, Bosy-Westphal A, Mueller M, Shen W, Gallagher D, Maeda Y, McDougall A, Peterson CM, Ravussin E, et al. Relationships between body roundness with body fat and visceral adipose tissue emerging from a new geometrical model: Body roundness with body fat & visceral adipose tissue. *Obesity* (2013) 21:2264–2271. doi: 10.1002/oby.20408

[13] Wang J, Wu M, Wu S, Tian Y. Relationship between body roundness index and the risk of heart failure in Chinese adults: The kailuan cohort study. *ESC Heart Fail* (2022) 9:1328–1337. doi: 10.1002/ehf2.13820

[14] Yang M, Liu J, Shen Q, Chen H, Liu Y, Wang N, Yang Z, Zhu X, Zhang S, Li X, et al. Body roundness index trajectories and the incidence of cardiovascular disease: Evidence from the China health and retirement longitudinal study. *J Am Heart Assoc* (2024) 13: e034768. Doi: 10.1161/JAHA.124.034768

[15] Zhang X, Ma N, Lin Q, Chen K, Zheng F, Wu J, Dong X, Niu W. Body roundness index and all-cause mortality among US adults. *JAMA Netw Open* (2024) 7: e2415051. Doi: 10.1001/jamanetworkopen.2024.15051

[16] Wu M, Yu X, Xu L, Wu S, Tian Y. Associations of longitudinal trajectories in body roundness index with mortality and cardiovascular outcomes: A cohort study. *Am J Clin Nutr* (2022) 115:671–678. doi: 10.1093/ajcn/nqab412

[17] Lai X, Chen T. Association between the triglyceride-glucose index and serum soluble klotho in middle-aged and older adults from NHANES 2007-2016. *Sci Rep* (2024) 14:18408. doi: 10.1038/s41598-024-69226-5

[18] Qiu L, Xiao Z, Fan B, Li L, Sun G. Association of body roundness index with diabetes and prediabetes in US adults from NHANES 2007-2018: A cross-sectional study. *Lipids Health Dis* (2024) 23:252. doi: 10.1186/s12944-024-02238-2

[19] Zhang X, Liang J, Luo H, Zhang H, Xiang J, Guo L, Zhu X. The association between body roundness index and osteoporosis in American adults: Analysis from NHANES dataset. *Front Nutr* (2024) 11:1461540. doi: 10.3389/fnut.2024.1461540

[20] Ding Z, Zhuang Z, Tang R, Qu X, Huang Z, Sun M, Yuan F. Negative association between body roundness index and bone mineral density: Insights from NHANES. *Front Nutr* (2024) 11:1448938. doi: 10.3389/fnut.2024.1448938

[21] Yu J-H, Yin M-G. Association between weight-adjusted waist circumference index and myocardial infarction: Results from the national health and nutrition examination survey, 1999-2014. *Front Cardiovasc Med* (2024) 11:1451987. doi: 10.3389/fcvm.2024.1451987

[22] Liao J, Qiu M, Li J, Li Y, Han Y. Association of visceral adipose tissue with hypertension: Results from the NHANES 2011-2018 and mendelian randomization analyses. *J Clin Hypertens (Greenwich)* (2025) 27: e14953. Doi: 10.1111/jch.14953

[23] Zhang F, Ning Z, Wang C. Body roundness index and cognitive function in older adults: A nationwide perspective. *Front Aging Neurosci* (2024) 16:1466464. doi: 10.3389/fnagi.2024.1466464

[24] Wan H, Wang Y, Xiang Q, Fang S, Chen Y, Chen C, Zhang W, Zhang H, Xia F, Wang N, et al. Associations between abdominal obesity indices and diabetic complications: Chinese visceral adiposity index and neck circumference. *Cardiovasc Diabetol* (2020) 19:118. doi: 10.1186/s12933-020-01095-4

[25] Ibrahim MM. Subcutaneous and visceral

adipose tissue: Structural and functional differences. *Obes Rev* (2010) 11:11–18. doi: 10.1111/j.1467-789X.2009. 00623.x

[26] Van Gaal LF, Mertens IL, De Block CE. Mechanisms linking obesity with cardiovascular disease. *Nature* (2006) 444:875–880. doi: 10.1038/nature05487

[27] Li S, Liu Z, Joseph P, Hu B, Yin L, Tse LA, Rangarajan S, Wang C, Wang Y, Islam S, et al. Modifiable risk factors associated with cardiovascular disease and mortality in China: A PURE substudy. *Eur Heart J* (2022) 43:2852–2863. doi: 10.1093/eurheartj/ehac268

[28] Piché M-E, Tchernof A, Després J-P. Obesity phenotypes, diabetes, and cardiovascular diseases. *Circ Res* (2020) 126:1477–1500. doi: 10.1161/CIRCRESAHA.120.316101

[29] Sedaghat Z, Khodakarim S, Nejadghaderi SA, Sabour S. Association between metabolic syndrome and myocardial infarction among patients with excess body weight: A systematic review and meta-analysis. *BMC Public Health* (2024) 24:444. doi: 10.1186/s12889-024-17707-7

[30] Prasad SB, Fahrtash F, Malaiapan Y, Meredith IT, Cameron J. Obesity and the metabolic syndrome in patients with acute myocardial infarction. *Int J Cardiol* (2010) 144:450–451. doi: 10.1016/j.ijcard.2009.03.089

[31] Hansen L, Netterstrøm MK, Johansen NB, Rønn PF, Vistisen D, Husemoen LLN, Jørgensen ME, Rod NH, Færch K. Metabolically healthy obesity and ischemic heart disease: A 10-year follow-up of the Inter99 study. *J Clin Endocrinol Metab* (2017) 102:1934–1942. doi: 10.1210/jc.2016-3346

[32] Yusuf S, Hawken S, Ounpuu S, Bautista L, Franzosi MG, Commerford P, Lang CC, Rumboldt Z, Onen CL, Lisheng L, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 countries: A case-control study. *Lancet* (2005) 366:1640–1649. doi: 10.1016/S0140-6736(05)67663-5

[33] Xu J, Zhang L, Wu Q, Zhou Y, Jin Z, Li Z, Zhu Y. Body roundness index is a superior indicator to associate with the cardio-metabolic risk: Evidence from a cross-sectional study with 17,000 eastern-China adults. *BMC Cardiovasc Disord* (2021) 21:97. doi: 10.1186/s12872-021-01905-x

[34] Hansen MK, Olesen KKW, Gyldenkerne C, Thrane PG, Stødkilde-Jørgensen N, Mortensen MB, Maeng M. Eligibility for and preventive potential of semaglutide in overweight and obese patients with myocardial infarction. *J Am Coll Cardiol* (2024) 83:956–958. doi: 10.1016/j.jacc.2023.12.029

[35] Green JB, Everett BM, Ghosh A, Younes N, Krause-Steinrauf H, Barzilay J, Desouza C, Inzucchi SE, Pokharel Y, Schade D, et al. Cardiovascular Outcomes in GRADE (Glycemia Reduction Approaches in Type 2 Diabetes: A Comparative Effectiveness Study). *Circulation* (2024) 149:993–1003. doi: 10.1161/circulationaha.123.066604