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Association between body shape index and coronary heart disease in individuals over 20 years old with obese



Huabin He^{1,2}, Yang Chen¹, Yanhui Liao¹, Longlong Hu¹, Hao Qin¹ and Renqiang Yang^{1*}

Abstract

Background While body mass index (BMI) defines obesity as a well-established risk factor for cardiovascular disease, the paradoxical theory of BMI suggests that obesity may indeed have a favorable impact on the prognosis of cardiovascular disease. Therefore, this study aims to assess the correlation between body shape index (ABSI), which is a novel measure of obesity, and coronary heart disease (CHD) among obese individuals in the United States.

Methods The data from the National Health and Nutrition Examination Survey (NHANES) were evaluated by us for 5046 patients. We assessed the exposure variable ABSI, which includes waist circumference (WC), height, and BMI. The outcome variable was CHD.

Results The cross-sectional study included a total of 5046 obese adults aged over 20 years, with an average age (standard deviation: SD) of 49.86 (16.24) years and a male proportion of 44.57%. The odds ratio (OR) values for CHD in Model 1, Model 2, 3 were found to be 2.45 (95%CI: 2.12, 2.83), 1.53 (95%CI:1.30, 1.81) and 1.31 (95%CI:1.09, 1.56) per SD increase in ABSI, respectively. In the fully adjusted model, we designated participants in the T1 group as the reference group. Our findings indicate a significant increase in the prevalence of CHD (OR:1.82, 95%CI: 1.07–3.10) only within the T3 group. Although there is an increased prevalence of CHD (OR:1.32, 95%CI: 0.77–2.29) in the T2 group, no statistically significant difference was observed.

Conclusions The increase in ABSI is strongly associated with the rise in CHD prevalence among obese individuals in the United States.

Keywords Body shape index, Coronary heart disease, Obese patients, NHANES

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Introduction

According to the global report on the burden of ischemic heart disease (coronary heart disease) spanning from 1990 to 2019, it is estimated that in 2019, a staggering 9.14 million individuals succumbed to coronary heart disease (CHD) worldwide, while an alarming number of 197 million people were affected by this condition [1]. However, according to the 2022 statistics on heart disease and stroke in the United States, heart disease remains the leading cause of mortality among Americans. In 2018, CHD accounted for approximately 12.6% (360,900 deaths) of all fatalities in the United State [2]. This has imposed a significant social and economic burden on American society, with estimated direct and indirect costs associated with heart disease in the United States amounting to \$228.7 billion annually from 2017 to 2018 [2, 3]. Therefore, it is imperative to continue prioritizing the traditional risk factors as the fundamental basis for primary prevention of CHD. However, researchers are particularly concerned about the profound impact of obesity on the burden of CHD in healthy American adults [4].

Additionally, according to data from the World Health Organization (WHO), there has been a significant increase in the prevalence of overweight and obesity, ranking fifth among global mortality risk factors after hypertension, smoking, hyperglycemia, and physical inactivity. In high-income and middle-income countries, the prevalence of overweight and obesity among adults has exceeded 50%, making them the third leading cause of death following hypertension and tobacco use. In the United States, overweight and obesity are ranked as the second most preventable causes of mortality after smoking [5]. Since the early 1990s, body mass index (BMI) has been widely used to classify overweight and obesity as well as investigate associated risks. While it provides reliable insights into overweight conditions, it does not differentiate between adipose tissue and lean body mass [6-8]. Therefore, several studies have reported a paradoxical association between BMI and cardiovascular disease prognosis, suggesting that a higher BMI is associated with improved outcomes [9–11]. Hence, the ongoing debate concerning the reliability of using BMI as a measure for overweight and obesity, along with the necessity to develop more inclusive approaches to evaluate the influence of fat content on cardiovascular diseases in individuals with a BMI \geq 30 kg/m², requires immediate investigation into alternative indicators of obesity that can outperform BMI. ABSI is an all-encompassing index that precisely evaluates central obesity by integrating waist circumference, BMI, and height [12]. Additionally, several studies have demonstrated the independent predictive value of ABSI for mortality, regardless of BMI levels [13].

Therefore, we investigated the association between a novel obesity index called ABSI and CHD in individuals classified as obese with a BMI of \geq 30 kg/m².

Methods

Study design and population

The analysis utilized publicly available data from the combined 2013-2018 NHANES cycle. NHANES employed a complex, multi-stage probabilistic sampling design to obtain a nationally representative sample. The sampling details are as follows: A multi-stage sampling design is used. Firstly, the entire country is divided into primary sampling units (PSUs), typically counties or county-level independent cities. Then segments are randomly selected within each PSU. Finally, households and individuals are randomly selected within each chosen residential area. Stratified and unequal probability sampling methods are employed to ensure the representativeness of the sample in NHANES. The samples are stratified based on demographic characteristics such as age, gender, and race/ ethnicity to include different groups of people. Different individuals have varying probabilities of being sampled to ensure an adequate representation of minority and vulnerable groups. The objectives of NHANES are to provide objective health statistics and address public health issues that affect the general population. Detailed survey operating manuals, consent documents, and pamphlets for each period can be found on the NHANES website at https://www.cdc.gov/nchs/nhanes/index.htm. The study design of NHANES was approved by the Institutional Review Board of the National Center for Health Statistics (NCHS)(approval number: Continuation of Protocol #2011-17and Protocol #2018-01), and all participants provided informed consent.

Between 2013 and 2018, a total of 6,058 obese participants aged over 20 with complete ABSI and CHD data were identified. After excluding 1,012 patients with missing covariates, our final data analysis included 5,046 participants. Please refer to Fig. 1 for the detailed flowchart.

Defnition of the exposure and outcome variable

The exposure variable in this study is ABSI, which comprises waist circumference (WC), height, and BMI. ABSI is calculated as WC divided by the square of BMI multiplied by the square root of height [8]. The outcome variable, CHD, is assessed through a questionnaire, and the patients' CHD status is coded by trained clinicians according to the International Statistical Classifcation of Diseases and Related Health Problems, Tenth Revision (ICD-10) [14, 15] using codes I00-I09, I11, and I13 for initial identification and I20-51 for further confirmation. Relevant literature has further substantiated the questionnaire-based collection of CHD diseases in terms of its accuracy [16].



Fig. 1 Flow chart of research population

Data collection

Sociodemographic characteristics

The participants reported their age, sex, race (Hispanic, non-Hispanic White, non-Hispanic Black, other race), poverty income ratio, current smoking status (no or yes), disease history, and medication history. Current smokers were confirmed to smoke every day or several days [17]. Anthropometric indicators include height, weight, waist circumference (WC), and blood pressure (BP). Body height and weight were measured without shoes or thick clothing using a medical scale. WC (cm) was measured at the level of the umbilicus during normal end expiration while standing, or if the umbilical level was lower due to visceral fat accumulation, between the costal margin and iliac crest. BMI, calculated as weight divided by height squared in kg/m2, was used as an indicator. Blood pressure measurements were taken using a mercury sphygmomanometer following the standard protocol recommended by the American Heart Association at that time. Three readings were obtained consecutively from the same arm. Systolic blood pressure and diastolic blood pressure (DBP) were defined as the average of these three measurements. Hypertension was defined as SBP \geq 140 mm Hg and/or DBP \geq 90 mm Hg, or if an individual reported taking antihypertensive medication or being diagnosed with hypertension by a healthcare professional. Diabetes mellitus (DM) was defined as having a fasting blood glucose level of \geq 7 mm/L, being diagnosed with diabetes by a healthcare professional, or currently using medication to manage high blood glucose levels [18].

Laboratory measurements

Blood samples were analyzed under fasting conditions following an overnight fast of more than 8 h. All blood parameters, including blood lipids, renal function parameters, and glycated hemoglobin A1c (HbA1c%), were measured at a certified medical and chemical laboratory diagnostic department using standardized international laboratory methods. The glomerular filtration rate (eGFR) was estimated using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [19].

Statistical analysis

The threshold for statistical significance was set at a twotailed P-value of less than 0.05. The statistical packages R (http://www.r-project.org) and Empower (R) (www. empowerstats.com) were employed to conduct all statistical analyses.

The normality of evaluation variables in this study was assessed through both Quantile-Quantile plots (Q-Q plots) and Anderson-Darling tests. Specifically, two methods were employed to examine whether a normal distribution exists: comparing the histogram of sample data with that of a standard normal curve and comparing normalized quantiles from sample data with those from a standard normal distribution using a Q-Q diagram. In this diagram, the correlation between the sample data and normal data can indicate whether they conform to a normal distribution. For normally distributed data, scattered points on the Q-Q diagram should approximate a straight line, indicating a high positive correlation. Since this study had a sample size of ≥ 200 , we employed an Anderson-Darling test to determine if variables conformed to a normal distribution. When $p \ge 0.05$, it was considered that the data conformed to a normal distribution.Mean and standard deviation (SD) were used to describe the continuous variables, and one-way analysis of variance (ANOVA) was performed for comparison. Percentages were used to describe the categorical variables, and a chi-square test was conducted for comparison. Multivariate logistic regression models were used to calculate the odds ratio (OR) values of CHD and explore the relationship between ABSI and outcome variables. Three models were constructed in this study to exclude confounding factors. Model 1 represented the unadjusted data, model 2 was adjusted for age, sex, BMI, SBP, DBP, model 3 was adjusted for age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL, eGFR, DM, Hypertension, antihypertensive drugs, Lipoprotein-lowering drugs, hypoglycemic drugs. Covariate were identified by having the outcome variable as a traditional risk factor and by having the effect of the calculated covariate on the outcome variable estimate changed by at least 10% individually [20]. A generalized additive model and fitting curve (penalized spline method) was performed to detect the linear relationships between the ABSI and CHD. In addition, possible modifications of the association between ABSI and CHD were also assessed for the following variables: sex (male vs. female), age (<65 vs. \geq 65), race ((non-hispanic white vs. non-hispanic black vs. mexican american vs. other hispanic vs. other races), DM (no vs. yes) and hypertension (no vs. yes).

Results

Baseline characteristics of the population

The cross-sectional study included a total of 5046 obese adults who were over the age of 20, with an average age of 49.86 years (SD:16.24) and a male proportion of 44.57% (2249 individuals). The mean ABSI value was calculated to be 0.082 (SD:0.005). Among the participants, there were 2,714 individuals diagnosed with hypertension, 1,259 (24.95%) individuals diagnosed with diabetes, and 240 individuals diagnosed with coronary heart disease. Table 1 presents the demographic and clinical baseline characteristics of the third-class population based on ABSI. We observed that individuals with the highest ABSI level (ABSI \geq 0.084, *n*=1682) exhibited several distinctive characteristics: predominantly males, advanced age, primarily non-Hispanic whites, and a significant proportion were current smokers. Compared to participants in the ABSI T1 group (ABSI<0.080, n=1682), those in the T3 group (ABSI \geq 0.084, *n*=1682) exhibited higher BMI, SBP, glycosylated hemoglobin levels, and eGFR values, as well as lower DBP levels. Additionally, they had a lower poverty income ratio and showed higher TC and HDL values. Furthermore, the T3 group (ABSI \geq 0.084, n=1682) had a greater prevalence of comorbidities such as diabetes and hypertension compared to the T1 group (ABSI \geq 0.084, *n*=1682). Moreover, medication rates for antihypertensive drugs, hypoglycemic drugs, and lipidlowering drugs were higher among patients in the T3 group than those in the T1 group.

Association between ABSI and CHD

The fitting curve in Fig. 2 clearly demonstrates the doseresponse association between ABSI and CHD, revealing an L-shaped positive relationship between these two variables. To quantitatively assess the impact of an increase in ABSI on CHD prevalence, we constructed three logistic regression models to evaluate the association between ABSI and CHD (Table 2). Furthermore, in the regression analysis, we assessed the impact of ABSI both as a continuous variable and as a categorical variable on CHD. The odds ratio (OR) values for CHD in Model 1, Model 2, 3 were found to be 2.45 (95%CI: 2.12, 2.83), 1.53 (95%CI:1.30, 1.81) and 1.31 (95%CI:1.09, 1.56) per SD increase in ABSI, respectively. In the fully adjusted model, we designated participants in the T1 group as the reference group. Our findings indicate a significant increase in the prevalence of CHD (OR:1.82, 95%CI: 1.07–3.10) only within the T3 group. Although there is an increased prevalence of CHD (OR:1.32, 95%CI: 0.77-2.29) in the T2 group, there is no statistically significant difference observed in this increase. When considering ABSI as a trisection variable, we observed that participants in the T3 group exhibited a significantly higher prevalence of CHD compared to those in the T2 group,

Characteristics	ABSI			P value
	Tertiles1 (< 0.080)	Tertile 2 (0.080–0.084)	Tertile 3(≥0.084)	
N	1682	1682	1682	
Males, <i>N</i> (%)	569 (33.83%)	818 (48.63%)	862 (51.25%)	< 0.001
Age, year	42.74 ± 14.55	49.35±15.22	57.47 ± 15.46	< 0.001
BMI, kg/m ²	36.93 ± 6.58	36.07 ± 5.74	35.42 ± 4.97	< 0.001
Race				< 0.001
Non-Hispanic White, N(%)	511 (30.38%)	629 (37.40%)	820 (48.75%)	
Non-Hispanic Black, N(%)	536 (31.87%)	405 (24.08%)	312 (18.55%)	
Mexican American, N(%)	279 (16.59%)	320 (19.02%)	283 (16.83%)	
Other Hispanic, N(%)	190 (11.30%)	189 (11.24%)	163 (9.69%)	
Other races, N(%)	166 (9.87%)	139 (8.26%)	104 (6.18%)	
Current smoking, N(%)	282 (16.77%)	293 (17.42%)	298 (17.72%)	< 0.001
SBP, mmHg	124.23 ± 16.41	127.68±17.31	129.82±18.14	< 0.001
DBP, mmHg	72.74±11.50	73.12±11.53	71.23±13.02	< 0.001
Poverty income ratio	2.50 ± 1.58	2.48±1.61	2.29 ± 1.52	< 0.001
HbA1c,%	5.77±1.10	6.02 ± 1.22	6.33 ± 1.43	< 0.001
TC, mg/dL	190.92±39.54	192.11±43.49	187.75±45.12	0.009
HDL, mg/dL	50.29 ± 14.02	48.12±13.60	47.01 ± 12.76	< 0.001
eGFR, mL/min/1.73 m ²	99.33±22.27	94.22±23.07	86.30 ± 24.85	< 0.001
Comorbidities, N (%)				
DM	219 (13.02%)	404 (24.02%)	636 (37.81%)	< 0.001
Hypertension	714 (42.45%)	898 (53.39%)	1102 (65.52%)	< 0.001
CHD	21 (1.249%)	58 (3.448%)	161 (9.572%)	< 0.001
Medication use, N (%)				
Antihypertensive drugs	455 (27.05%)	660 (39.24%)	927 (55.11%)	< 0.001
Lipoprotein-lowering drugs	217 (12.90%)	384 (22.83%)	653 (38.82%)	< 0.001
hypoglycemic drugs	149 (8.86%)	290 (17.24%)	507 (30.14%)	< 0.001

Abbreviations: ABSI: A Body Shape Index; BMI body mass index, SBP systolic blood pressure, DBP diastolic blood pressure, HbA1c, glycosylated hemoglobin; TC, total cholesterol; HDL, high-density lipoprotein cholesterol; HbA1c, glycosylated hemoglobin; eGFR, estimated glomerular filtration rate; DM, Diabetes mellitus;

with an odds ratio (OR) for CHD much greater than that of the T2 group. This finding is consistent with our fitted curve and supports a significant positive association between elevated ABSI levels and increased prevalence of CHD.

Subgroup analyses

In the subgroup analysis(Fig. 3), we evaluated how ABSI, as a continuous variable, is associated with the prevalence of CHD to explore possible interactions and examine whether other variables within different subgroups affect this relationship. The P-value for interaction across all subgroups was determined to be greater than 0.05, suggesting that these specific subgroups do not modify the primary finding that an increase in ABSI is linked to a higher prevalence of CHD. These subgroups included sex (male vs. female), age (<65 vs. \geq 65), race ((non-hispanic white vs. non-hispanic black vs. mexican american vs. other hispanic vs. other races), DM (no vs. yes) and hypertension (no vs. yes). Each subgroup analysis adjusted for age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL, eGFR, DM, Hypertension, antihypertensive drugs, Lipoprotein-lowering drugs, hypoglycemic drugs. except for the stratifying variable.

Discussion

In this population-based cross-sectional study, we observed a significant positive L-shaped association between the increase in ABSI among obese individuals and the prevalence of CHD.

Previous studies have predominantly focused on the association between obesity and cardiovascular diseases as defined by BMI [21–23]. However, due to the limitations of BMI in accurately discerning fat, bone, and muscle mass [24], the concept of the obesity paradox emerged. Furthermore, Franciane and his colleagues exclusively conducted a study on the correlation between BMI and coronary artery disease (CAD). They meticulously assessed the BMI of 703 inpatients admitted to the cardiology department, while also evaluating the severity of their CAD. The conclusive findings revealed that obesity served as an independent risk factor for early-onset CAD and exhibited a strong association with comorbidities such as diabetes and hypertension [25]. Canoy et al. conducted a 9-year cohort follow-up study among



Fig. 2 Association between body shape index and coronary heart disease. A linear association between body shape index and coronary heart disease was found (*P* < 0.05). The solid line and dashed line represent the estimated values and their corresponding 95% confidence interval. Adjustment factors included age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL, eGFR, DM, Hypertension, antihypertensive drugs, Lipoprotein-lowering drugs, hypoglycemic drugs

ABSI	Events (%)	CHD, OR (95%CI)		
		Model1	Model 2	Model 3
Per 1SD increase	240(4.76%)	2.45 (2.12, 2.83)	1.53 (1.30, 1.81)	1.31 (1.09, 1.56)
Tertiles1 (< 0.080)	21 (1.249%)	1	1	1
Tertile 2 (0.080–0.084)	58 (3.448%)	2.82 (1.71, 4.68)	1.64 (0.97, 2.77)	1.32 (0.77, 2.29)
Tertile 3(≥0.084)	161 (9.572%)	8.37 (5.29, 13.26)	2.78 (1.69, 4.59)	1.82 (1.07, 3.10)
P for trend		< 0.0001	< 0.0001	0.011

Model 1 was adjusted for none

Model 2 was adjusted for age, sex, BMI, SBP, DBP

Model 3 was adjusted for age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL, eGFR, DM, Hypertension, antihypertensive drugs, Lipoprotein-lowering drugs, hypoglycemic drugs

1.2 million women aged 55–74 to investigate the association between BMI and first hospitalization for CHD and CHD mortality. The relative risk of CHD is 1.23(95% CI: 1.22–1.25) when BMI increases by 5 kg/m². The relationship between BMI and CHD mortality follows a J-shaped curve, which differs from its incidence pattern. In both the lowest and highest BMI categories, the relative risk of coronary heart disease mortality is greater than that of incidence [26]. Firstly, Franciane did not provide a quantitative assessment of the severity of BMI and the presence of coronary heart disease; instead, only correlation analysis was conducted. Moreover, this study exclusively focused on inpatients from the cardiology department who had undergone coronary angiography, without any relevant investigation into the association between BMI and CHD within the general population. Secondly, Canoy et al. exclusively investigated the impact of BMI on fatal and



Fig. 3 Stratified analyses by potential modifiers of the association between body shape index and coronary heart disease. **Each subgroup analysis adjusted for age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL, eGFR, DM, Hypertension, antihypertensive drugs, Lipoprotein-lowering drugs, hypoglycemic drugs. except for the stratifying variable

non-fatal CHD in women, revealing that the majority of coronary heart disease events occurred in non-obese women. Even a slight increase in BMI was associated with an elevated incidence of the disease, suggesting that BMI may not be applicable to individuals who are not obese. Lastly, due to inherent limitations of BMI itself, researchers have been continuously exploring alternative obesity indicators to overcome the obesity paradox.

Krakauer and his colleagues [13] have proposed a novel obesity index, ABSI, which is derived from weight and height measurements using NHANES data in the United States. Notably, ABSI demonstrates superior predictive capability for premature mortality compared to traditional measures such as or WC [13, 27]. Furthermore, numerous studies have substantiated the utility of ABSI as a prognostic indicator for cardiovascular disease and all-cause mortality [28, 29]. Therefore, this study represents the first attempt to assess the impact of ABSI on CHD in obese individuals residing in the United States. This research addresses existing gaps in knowledge regarding novel obesity indices and their association with cardiovascular diseases, while also broadening. This cross-sectional study shows that the prevalence of CHD increases with the increase of ABSI level, and there is a positive correlation between them, and this positive correlation is independent of other covariates. The ABSI is primarily utilized as a reliable index for assessing body fat distribution and measuring the accumulation of adipose tissue. An elevation in ABSI signifies excessive fat deposition, leading to the generation of cytotoxic substances and induction of oxidative stress, which can detrimentally impact the integrity of blood vessel walls. Consequently, this process promotes plaque formation and luminal obstruction, thereby contributing to the pathogenesis and progression of CHD.

The presence of the obesity paradox leads individuals to believe that BMI-defined obesity confers cardiovascular benefits, thereby discouraging weight loss efforts in this population. Therefore, this study focuses on patients with a BMI \geq 30 kg/m2 and aims to assess the association between obesity and CHD using the novel obesity index ABSI. The findings reveal an L-shaped positive correlation between ABSI levels and the incidence of coronary heart disease among obese patients. These aforementioned findings imply the imperative need for monitoring the ABSI index of obese patients in clinical practice in order to mitigate the incidence and progression of coronary heart disease among this patient population. Additionally, it is crucial to establish corresponding healthcare policies, promote adherence to a recommended BMI level as part of these policies, and provide necessary weight loss interventions and related education for individuals who are obese.

Our research has several limitations. Firstly, due to the cross-sectional nature of the data, it is not possible to establish a causal relationship between ABSI and CHD prevalence. To establish causality and potential mechanisms, large-scale cohort studies and basic experiments are required. Secondly, the observed relationship is limited to American adults, which may restrict the generalizability of our findings. Lastly, while we have adjusted for confounding factors through covariate adjustment, we cannot completely eliminate the influence of unmeasured or unknown factors on our results; for instance, we considered participants' family history of heart disease, tim-

ing of coronary heart disease onset in them, and whether

Conclusions

they underwent surgery.

The findings of this study demonstrate a high prevalence of CHD, which is significantly associated with an increase in ABSI. Importantly, the L-shaped positive association between ABSI and coronary heart disease remains robust even after adjusting for confounding factors such as age, sex, BMI, race, poverty income ratio, SBP, DBP, smoking status, HbA1c%, TC, HDL cholesterol levels, eGFR (estimated glomerular filtration rate), DM (diabetes mellitus), hypertension, antihypertensive drugs, lipoprotein-lowering drugs, and hypoglycemic drugs. The findings remain consistent across diverse ethnic groups in the United States. Currently, there is an urgent need to develop effective health policies that target the reduction of identified risk factors and mitigate the imminent impact of CHD on individuals with obesity in the United States.

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Author contributions

HBH participated in literature search, study design, data collection, data analysis, data interpretation, and wrote the manuscript. HBH, YC, YHL, LLH and HQ conceived of the study, and participated in its design, coordination, data collection and analysis. RQY participated in study design and provided the critical revision. All authors read and approved the final manuscript.

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None.

Data availability

Publicly available datasets were analyzed in this study. This data can be found here: https://www.cdc.gov/nchs/nhanes/index.htm.

Declarations

Conflict of interest

The authors declare that they have no conflict of interest.

Ethics approval and consent to participate

All procedures performed in studies involving human participants were following the ethical standards of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

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

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