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Original Article

Association between a body shape index and myocardial infarction: a cross-sectional analysis of NHANES data with mediation modeling

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Abstract: Background: Myocardial infarction (MI) represents a significant global health burden. A Body Shape Index (ABSI) may indicate MI risk related to central adiposity, but large-scale evidence remains limited. Objective: To examine the association between ABSI and MI, its mediating pathways, and subgroup variations using nationally representative data. Methods: The significant empirical analysis examined 44,281 adults from NHANES 1999-2018, and the key Survey-weighted logistic regression could plausibly demonstrate that ABSI-MI odds ratios (ORs) reveal important patterns. Restricted cubic splines tested linearity. Mediation analysis evaluated effects of hypertension, diabetes, and hyperlipidemia. Predictive performance was evaluated using the area under the curve (AUC). Survey-weighted logistic regression assessed ABSI-MI odds ratios (ORs). Restricted cubic splines assessed linearity. Mediation analysis evaluated effects of hypertension, diabetes, and hyperlipidemia. Predictive performance was compared using AUC. Results: The highest ABSI quartile was associated with 73% higher odds of MI (OR = 1.73, 95% CI 1.26-2.39) after full adjustment. A linear dose-response relationship was observed (P for nonlinearity = 0.655). Hypertension, diabetes, and hyperlipidemia mediated 80.1% of the association, with diabetes alone explaining 44.3%. Stronger associations were found in adults <65 years, women, smokers, and drinkers. In light of these findings, ABSI may suggest similar discrimination (AUC = 0.848) compared with BMI (AUC = 0.850), their combination achieved an AUC = 0.853. Conclusions: ABSI may demonstrate that it predicts MI independently and linearly in US adults. The relationship is largely mediated by metabolic conditions and is strongest among younger individuals and those with adverse lifestyle profiles. Adding ABSI to BMI may modestly improve MI risk stratification.

Keywords: A body shape index, myocardial infarction, nonlinear relationship, risk stratification, NHANES

Introduction

Cardiovascular diseases (CVDs) remain the chief culprit in global burden of disease, exerting disproportionate pressure on health systems [1]. Moreover, the most serious type in coronary artery disease is myocardial infarction (MI), accounts for a significant proportion of this burden, with incidence rising among younger cohorts and prognosis remaining guarded [2, 3]. Furthermore, the important evidence could demonstrate that although timely percutaneous coronary intervention (PCI) improves acute survival, it appears to show that it fails to salvage irreversibly damaged myocardium or reverse underlying atherosclerosis. Consequently, the risks of restenosis and recurrent ischemic events may persist, conferring enduring impairment in health-related quality of life [4, 5]. However, establishing pragmatic, early-warning biomarkers that can be deployed at scale might indicate importance for primary prevention.

Central adiposity is not overall obesity, but the key to cardiovascular disease (CVD) [6, 7]. In 2015, about 4 million deaths are associated with higher body mass index were due to CVD [8]. BMI cannot distinguish muscle from fat or

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identify fat distribution [9-11]. Waist circumference (WC) and waist-to-hip ratio (WHR) solve this problem to some extent, and with WC being more feasible for mortality assessment [12-14]. Even so, WC is a one-dimensional index, cannot distinguish visceral from subcutaneous fat, and provides relatively limited information.

To quantify visceral adiposity while accounting for body size, Krakauer et al. [15] devised A Body Shape Index (ABSI):

ABSI = WC/(BMI^(2/3) × height^(1/2)).

ABSI combines WC, height, and weight to reflect abnormal body geometry patterns of visceral fat under different body sizes. In cohort studies, ABSI is more strongly linked to all-cause mortality than WC and BMI [15, 16]. Computed tomography (CT) is the gold standard for assessing visceral fat, but cost and radiation problems limit its wide - scale use [17]. Conversely, ABSI based on conventional anthropometry has the characteristics of simplicity, economy, effectiveness, and good reproducibility.

More and more evidence links ABSI (visceral fat, an indicator of central obesity) to CVD. Large - scale cohort studies show that higher ABSI is related to higher cardiovascular mortality from CVD: in a Chinese cohort, with every 0.01 unit elevation in ABSI, the mortality rate from cardiovascular disease increases by 30%, the Rotterdam study found that in men, with each one-standard-deviation increment in ABSI, the risk increased by 18%. Meta-analysis shows that for each standard deviation increase in ABSI, all - cause mortality increases by 55% and cardiovascular disease risk increases by 21%; regarding mortality prediction, ABSI outperforms BMI and waist circumference. ABSI alone can predict stroke, and when used in combination with the triglyceride - glucose index (TyG index), it can enhance the assessment of the risk of atherosclerotic cardiovascular disease [18-21]. Evidence linking ABSI to cardiovascular endpoints remains fragmented. Small studies suggest that ABSI can identify high-risk individuals in the normal BMI population [22]; however, there is a lack of large-scale studies on MI. Whether ABSI has a linear dose-response relationship with MI, a threshold effect, or can add predictive value beyond traditional indices such as BMI needs further confirmation.

Using NHANES data from 1999-2018, we explored the cross - sectional association of ABSI - MI. We hypothesized that ABSI is independently associated with MI prevalence, operates through metabolic mediators, and displayes effect modification across demographic and lifestyle subgroups. Rigorous statistical approaches - including restricted cubic splines, multivariable-adjusted logistic regression, causal mediation analysis, and subgroup stratification - were employed to test these hypotheses. Our findings may inform the integration of simple anthropometric metrics into early MI risk stratification and targeted prevention strategies.

Methods

Data source and study population

Data from the NHANES was used. NHANES is a complex multi-stage probability sample of the US civilian non-institutionalized population. Standard interviews and MEC assessments are used to collect demographic, socioeconomic, dietary, and health-related information NCHS REB approved all protocols, and participants all gave written consent. The detailed protocol of anthropometry can be viewed at https://www.cdc.gov/nchs/nhanes.

Outcomes and covariates

Myocardial infarction (MI) was defined by “did a doctor or health professional ever say you had a myocardial infarction (MI)?”. A positive answer is the definition of myocardial infarction (MI). ABSI was computed for each participant from measured waist circumference, weight, and height. First, calculate the mean (ABSImean) and standard deviation ($ABSI_{SD}$) of a specific gender. Then, convert the original ABSI value to a z-score using the formula: $ABSI_z$ = (ABSI - $ABSI_{mean}$)/$ABSI_{SD}$.

Covariate selection was informed by a directed acyclic graph (DAG) synthesized with prior evidence. Age, gender, race/ethnicity, and smoking status were retained in all models a priori. Education level, poverty-to-income ratio (PIR), and BMI were included as potential confounders. Hypertension, diabetes and dyslipidemia were treated as potential mediators and were therefore excluded from primary models to avoid overadjustment. There are three situations for smoking: never smoked, ever smoked,

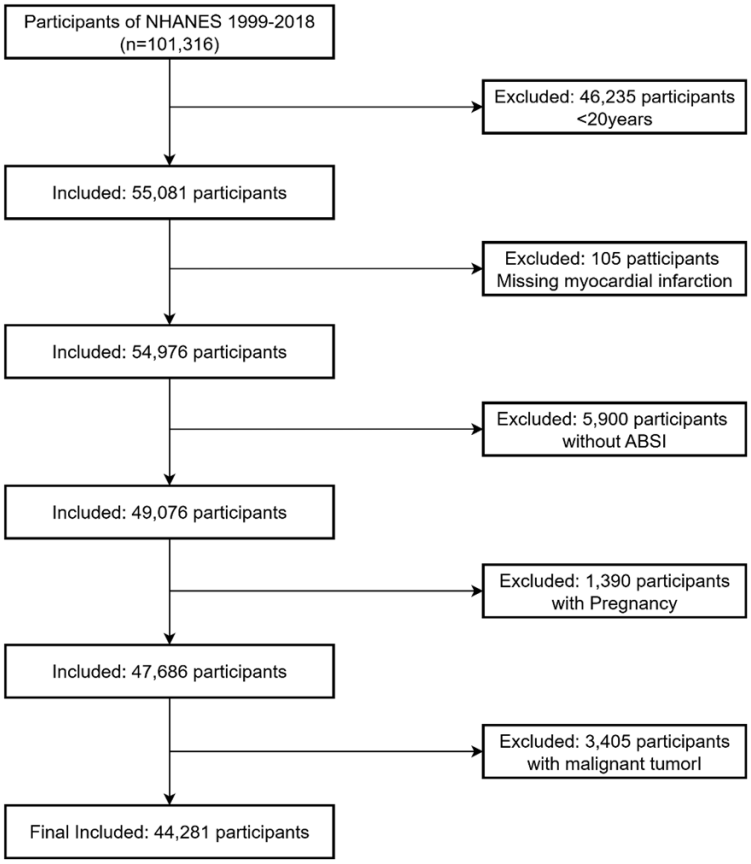

Figure 1. Flowchart of study population selection.

and currently smoking; there are three situations for drinking: never drink, drink a little, and drink a medium/large amount.

Measurement and quality control

Trained health technicians carry out anthropometric measurements in the MEC according to the standard procedures of NHANES in the United States. Measure height with a stadiometer and weigh body weight with an electronic scale, all measurements were recorded to the nearest 0.1 cm and 0.1 kg. Use a steel tape measure at the iliac crest to measure the waist circumference (WC) at the end of normal expiration, accurate to 0.1 cm. Two measurements were obtained for each variable; a third was taken if the initial two exceeded acceptable limits, with the final value averaged accordingly.

Fasting venous blood samples (≥8 hours) were collected for laboratory assessments. Detecting total cholesterol, high - density lipoprotein cholesterol (HDL-C) and triglycerides by enzymatic method (Roche Cobas 6000 or equivalent). Low-density lipoprotein cholesterol (LDL - C) was calculated using the Friedewald formula for participants with fasting duration ≥8.5 hours and triglycerides ≤400 mg/dL. The result is output in English as follows: FPG is detected by the hexokinase method, and GHb is detected by high-performance liquid chromatography (HPLC). All procedures followed CDC protocols, with certified laboratories maintaining intra-batch coefficients of variation below 5% and regular external quality control calibration.

Disease definitions

(1) Hypertension: self-reported diagnosis, antihypertensive use, systolic blood pressure exceeds 140 mmHg, or diastolic blood pressure exceeds 90 mmHg.

(2) Hyperlipidemia: self-reported high cholesterol, lipid-lowering medication use, or elevated total cholesterol or LDL cholesterol.

(3) Diabetes: self-reported diagnosis, glycated hemoglobin ≥6.5%, fasting blood glucose ≥7.0 mmol/L or taking hypoglycemic drugs.

Using NHANES data from 1999-2018. We exclude the initial 101316 participants: 46,235 people under 20 years old, 105 people with missing myocardial infarction data, 5,900 people without anthropometric data needed for ABSI calculation, 1,390 pregnant women, and 3,405 people with malignant tumors. The remaining 44,281 adults aged 20 years and above were used for analysis (**Figure 1**). Missing values were multiply imputed: continuous variables (e.g., LDL-C, age) using predictive mean matching (PMM), binary variables (e.g., gender, coronary heart disease, hypertension) by logistic regression (logreg), and categorical variables with ≥3 levels (e.g., education, race/ethnicity, marital status) by polytomous logistic regression (polyreg).

Statistical analysis

Continuous variables with a normal distribution are presented as mean ± standard deviation (SD), and non-normally distributed continuous variables as median (interquartile range, IQR). Categorical variables are presented as unweighted frequencies (n) and weighted percentages (%). Odds ratios (ORs) and 95% confidence intervals (CIs) are reported to two decimal places. *P* values are reported to three decimal places; values <0.001 are expressed as <0.001. For mediation analysis, coefficients (β) and bootstrap CIs are reported to four decimal places due to their small magnitude. Percentages of mediation are presented to one decimal place. Results from the five imputed datasets were combined using Rubin's rules, and pooled estimates are presented accordingly. All analyses accounted for the NHANES complex survey design using appropriate sampling weights.

Baseline characteristics were compared using weighted χ^2 tests for categorical variables and weighted t-tests or Kruskal-Wallis tests for continuous variables. Dose-response shape was explored with restricted cubic splines (four equidistant knots); departure from linearity was tested by the Wald method. Divide ABSI into quartiles according to gender, and take the lowest quartile as the reference. Four sequentially adjusted, survey-weighted logistic regression models were fitted: Model 1 was not adjusted; Model 2 incorporated demographic - related factors, such as gender, age, race or ethnicity, educational attainment, marital status, and poverty - income ratio; Model 3 further adjusted for life - related factors and major comorbidities, such as smoking, alcohol consumption, diabetes, and hypertension. Model 4 also adjusts for lipid, blood glucose status, insulin, and serum uric acid in addition to hypercholesterolemia, systolic and diastolic blood pressure. Causal mediation was quantified using weighted bootstrap (3,000 draws) to estimate the indirect effects of hypertension, diabetes, and hyperlipidemia. Subgroup analysis is carried out by stratifying according to age (less than 65 years old and greater than or equal to 65 years old), gender, smoking, drinking and metabolic disease status; stratum-specific effects were estimated by survey-weighted logistic regression. Discrimination was assessed by survey-weighted ROC curves and AUCs (95% CIs). All analyses used R version 4.1.0 (packages: survey, rms, mediation, pROC) and accounted for the NHANES complex sampling design; missing values were multiply imputed (m = 20).

Results

Baseline characteristics

Among 44,281 participants (22,184 men [49.7%] and 22,097 women [50.3%]), 1,715 (3.87%) self-reported prior MI. ABSI differed markedly between those with and without prior MI (weighted F = 699.7, P<0.001). Disparities extended to demographics, laboratory markers, and comorbidities (all P<0.05) (**Table 1**).

Association between ABSI and myocardial infarction

Using multivariate logistic regression to analyze the association between ABSI - MI risk. The results showed that after adjusting for gender, race/ethnicity, education level (educ), smoking, alcohol consumption, diabetes, hypercholesterolemia (hyperchol), hypertension, age, and poverty - to - income ratio (model 4), ABSI was an independent risk factor for myocardial infarction (MI). Specifically, using the first quartile (Q1) of ABSI as the reference, the risk of myocardial infarction showed an increasing trend with higher ABSI levels: no significant difference in risk was observed for Q2 (OR = 1.29, 95% CI: 0.93-1.79, P = 0.123); The risk increased by 36% in the Q3 (OR = 1.36, 95% CI: 1.01 to 1.84, P = 0.044); the risk increased by 73% in the Q4 (OR = 1.73, 95% confidence interval: 1.26 to 2.39), *P* value less than 0.001. Trend testing also supports that there is an obvious dose - response relationship between ABSI level and myocardial infarction risk (the odds ratio for each quartile increase is 1.19, the 95% confidence interval is from 1.07 to 1.32, and P is 0.001). Detailed results are presented in **Table 2**.

Mediation analysis

Weighted bootstrap mediation (3,000 draws) showed that hypertension, diabetes, and hyperlipidemia collectively mediated 80.1% of the ABSI-MI association. The total effect coefficient was 0.0186 (95% CI 0.0152-0.0220). After adjusting for the three mediators, leaving a direct effect of 0.0037 (95% CI 0.0013-

Table 1. Baseline characteristics and between-group differences

Characteristic	Overall (N = 44281)	With MI (N = 1715)	Without MI (N = 42566)	Test Statistic	*P*-value
ABSI, Mean (SE)	0.081 (0.001)	0.085 (0.001)	0.081 (0.001)	699.730	<0.001
ABSI_z, Mean (SE)	-0.015 (0.009)	0.267 (0.036)	-0.024 (0.009)	69.023	<0.001
BMI, Mean (SE)	28.7 (0.1)	30.1 (0.2)	28.6 (0.1)	53.142	<0.001
BMI_z, Mean (SE)	-0.03 (0.01)	0.21 (0.03)	-0.04 (0.01)	61.871	<0.001
Age(years), Mean (SE)	45.8 (0.2)	63.4 (0.3)	45.3 (0.2)	2,560.885	<0.001
Poverty Income Ratio, Mean (SE)	3.01 (0.03)	2.60 (0.06)	3.02 (0.03)	50.628	<0.001
TCHO, Mean (SE)	5.07 (0.01)	4.73 (0.04)	5.08 (0.01)	64.441	<0.001
HDL-C, Mean (SE)	1.37 (0.01)	1.24 (0.01)	1.37 (0.01)	74.682	<0.001
LDL-C, Mean (SE)	3.00 (0.01)	2.62 (0.05)	3.01 (0.01)	70.744	<0.001
UA, Mean (SE)	321.75 (0.65)	357.29 (2.94)	320.66 (0.64)	156.569	<0.001
TG, Median (Q1, Q3)	1.19 (0.82, 1.76)	1.40 (1.01, 2.16)	1.17 (0.81, 1.75)	-7.248	<0.001
FBS, Median (Q1, Q3)	5.46 (5.10, 5.94)	5.94 (5.38, 6.99)	5.44 (5.07, 5.94)	-11.628	<0.001
HbA1c, Median (Q1, Q3)	5.40 (5.10, 5.70)	5.70 (5.40, 6.40)	5.40 (5.10, 5.70)	-27.142	<0.001
HOMA_IR, Median (Q1, Q3)	55.50 (35.58, 90.42)	68.10 (45.00, 113.70)	55.08 (35.40, 89.64)	-6.468	<0.001
UACR, Median (Q1, Q3)	6.27 (4.17, 11.23)	9.34 (5.44, 24.91)	6.21 (4.14, 11.03)	-14.715	<0.001
Age Group, n (%)				1,281.570	<0.001
<65 years	34,748 (85.27)	719 (50.72)	34,029 (86.32)		
≥65 years	9,533 (14.73)	996 (49.28)	8,537 (13.68)		
Gender, n (%)				142.080	<0.001
Female	22,097 (50.28)	554 (34.05)	21,543 (50.78)		
Male	22,184 (49.72)	1,161 (65.95)	21,023 (49.22)		
Race/Ethnicity, n (%)				60.619	<0.001
Mexican American	8,024 (8.47)	206 (4.00)	7,818 (8.61)		
Other Hispanic	3,767 (5.77)	115 (3.72)	3,652 (5.83)		
Non-Hispanic White	18,848 (67.40)	972 (76.04)	17,876 (67.13)		
Non-Hispanic Black	9,419 (11.34)	308 (9.56)	9,111 (11.40)		
Other Race	4,223 (7.02)	114 (6.67)	4,109 (7.03)		
Education Level, n (%)				192.647	<0.001
<9th	5,331 (5.79)	332 (11.32)	4,999 (5.62)		
9-11th	6,616 (11.28)	307 (16.83)	6,309 (11.12)		
HS/GED	10,252 (24.06)	433 (28.33)	9,819 (23.93)		
Some College	12,530 (30.96)	412 (27.64)	12,118 (31.06)		
College	9,505 (27.90)	228 (15.88)	9,277 (28.27)		
Marital Status, n (%)				482.865	<0.001
Married	23,050 (56.20)	925 (58.32)	22,125 (56.14)		
Widowed	3,290 (4.90)	317 (15.87)	2,973 (4.56)		
Divorced	4,511 (9.90)	236 (12.76)	4,275 (9.82)		
Separated	1,497 (2.54)	54 (2.66)	1,443 (2.54)		
Never married	8,124 (18.63)	99 (5.50)	8,025 (19.03)		
Living with partner	3,391 (7.83)	64 (4.90)	3,327 (7.91)		
Alcohol Consumption, n (%)				55.762	<0.001
Never drinker	10,130 (20.63)	392 (23.11)	9,738 (20.55)		
Moderate drinker	10,574 (26.78)	534 (34.09)	10,040 (26.55)		
Heavy drinker	19,495 (52.60)	632 (42.79)	18,863 (52.90)		
Smoking Status, n (%)				338.215	<0.001
Never smoker	24,242 (54.30)	573 (31.54)	23,669 (54.99)		
Former smoker	10,408 (23.71)	729 (42.44)	9,679 (23.14)		
Current smoker	9,597 (21.99)	413 (26.03)	9,184 (21.87)		
Hypertension, n (%)				776.458	<0.001
without hypertension	25,963 (64.14)	406 (27.76)	25,557 (65.25)		
with hypertension	18,288 (35.86)	1,309 (72.24)	16,979 (34.75)		
Diabetes, n (%)				863.438	<0.001
Without diabetes	37,390 (88.87)	1,028 (63.70)	36,362 (89.63)		
With diabetes	6,891 (11.13)	687 (36.30)	6,204 (10.37)		

Hypercholesterolemia, n (%)				660.896	<0.001
Without hypercholesterolemia	26,755 (62.29)	521 (28.48)	26,234 (63.32)		
With hypercholesterolemia	17,028 (37.71)	1,186 (71.52)	15,842 (36.68)		
CKD				689.299	<0.001
Without CKD	36,474 (86.39)	922 (61.87)	35,552 (87.13)		
With CKD	7,807 (13.61)	793 (38.13)	7,014 (12.87)		
AP				6,576.198	<0.001
Without AP	43,017 (97.88)	1,170 (65.80)	41,847 (98.84)		
With AP	1,138 (2.12)	506 (34.20)	632 (1.16)		
CHD				11,369.025	<0.001
Without CHD	42,478 (96.96)	832 (46.32)	41,646 (98.46)		
With CHD	1,638 (3.04)	823 (53.68)	815 (1.54)		
BP_usedrug				1,222.732	<0.001
Without BP_drug	31,109 (78.68)	596 (39.89)	30,513 (79.87)		
With BP_drug	10,811 (21.32)	1,046 (60.11)	9,765 (20.13)		
LDL_usedrug				1,744.622	<0.001
Without LDL_drug	28,791 (83.07)	705 (43.48)	28,086 (84.47)		
With LDL_drug	6,975 (16.93)	892 (56.52)	6,083 (15.53)		
Diabetes_usedrug				734.841	<0.001
Without Diabetes_drug	39,691 (92.75)	1,206 (73.59)	38,485 (93.33)		
With Diabetes_drug	4,559 (7.25)	507 (26.41)	4,052 (6.67)		
ABSI Quartile (Weighted), n (%)				892.458	<0.001
1	9,081 (21.79)	79 (4.54)	9,002 (22.32)		
2	9,758 (23.81)	166 (10.80)	9,592 (24.20)		
3	11,435 (26.18)	367 (21.04)	11,068 (26.33)		
4	14,007 (28.22)	1,103 (63.62)	12,904 (27.14)		
BMI Quartile (Weighted), n (%)				100.947	<0.001
1	4,508 (10.78)	120 (6.37)	4,388 (10.91)		
2	11,734 (27.57)	370 (19.56)	11,364 (27.81)		
3	13,919 (30.55)	573 (33.04)	13,346 (30.47)		
4	14,120 (31.11)	652 (41.03)	13,468 (30.81)		

Mean (SE); Median (Q1, Q3); n (unweighted) (%); F-statistic for normal continuous variables; Kruskal-Wallis χ^2 for non-normal continuous variables; Pearson χ^2 for categorical variables; *P*-values from survey-weighted tests accounting for complex survey design; Normally distributed continuous variables are shown as Mean (SE); non-normal variables as Median (Q1, Q3). Medians, quartiles, and weighted percentages are all reported with two decimal places. Test statistics are presented with two decimal places. *P*-values are to three decimals (<0.001 as <0.001).

Table 2. Sequential multivariable-adjusted logistic modeling

Variable	Model 1		Model 2		Model 3		Model 4	
	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P	OR (95% CI)	P
ABSI (per SD increase)	1.36 (1.26-1.46)	<0.001	1.24 (1.15-1.33)	<0.001	1.2 (1.11-1.29)	<0.001	1.12 (1.04-1.21)	0.005
ABSI Q2 vs Q1	2.19 (1.57-3.06)	<0.001	1.46 (1.05-2.01)	0.023	1.44 (1.04-1.99)	0.028	1.29 (0.93-1.79)	0.123
ABSI Q3 vs Q1	3.93 (2.9-5.33)	<0.001	1.72 (1.27-2.33)	<0.001	1.66 (1.23-2.24)	0.001	1.36 (1.01-1.84)	0.044
ABSI Q4 vs Q1	11.52 (8.59-15.45)	<0.001	2.55 (1.84-3.51)	<0.001	2.34 (1.7-3.22)	<0.001	1.73 (1.26-2.39)	<0.001
Trend Test	2.36 (2.15-2.58)	<0.001	1.36 (1.22-1.51)	<0.001	1.31 (1.18-1.46)	<0.001	1.19 (1.07-1.32)	0.001

OR: Odds Ratio; CI: Confidence Interval; Model 1: Crude; Model 2: adjusted for gender, race, age; Model 3: adjusted for gender, race, education, smoking, alcohol, age, PIR; Model 4: adjusted for gender, race, education, smoking, alcohol, diabetes, hypercholesterolemia, hypertension, age, PIR. Note: OR and 95% CI reported with two decimal places; *P* values to three decimals.

0.0061). The indirect effects via hypertension, diabetes, and hyperlipidemia were 0.0025 (95% CI 0.0015-0.0036), 0.0082 (95% CI 0.0062-0.0105), and 0.0042 (95% CI 0.0031-0.0053), respectively; all 95% confidence intervals excluded 0, and *P* values were statistically significant at the 0.05 level. Diabetes contributed the largest share (44.3%), followed by hyperlipidemia (22.3%) and hypertension (13.4%); detailed estimates are provided in **Tables 3** and **4**.

Subgroup analysis

Overall, elevated ABSI was significantly associated with increased risk of myocardial infarc-

Table 3. Mediation effect

Path/Effect	Coefficient	SE	95% BootCI LL	95% BootCI UL	*P*-value
Total Effect (c)					
ABSI_z -> MI	0.0186	0.0017	0.0152	0.0220	<0.001
Direct Effect (c')					
ABSI_z -> MI (cont. all mediators)	0.0037	0.0012	0.0013	0.0061	0.0047
Path Coefficients (Parallel Model)					
ABSI_z -> Hypertension (a path)	0.1565	0.0154	0.1261	0.1871	<0.001
Hypertension -> MI (b path)	0.0160	0.0029	0.0104	0.0218	<0.001
ABSI_z -> Diabetes (a path)	0.3540	0.0205	0.3143	0.3934	<0.001
Diabetes -> MI (b path)	0.0233	0.0027	0.0180	0.0286	<0.001
ABSI_z -> Hyperlipidemia (a path)	0.1739	0.0146	0.1449	0.2026	<0.001
Hyperlipidemia -> MI (b path)	0.0239	0.0025	0.0192	0.0289	<0.001
Indirect Effects (a*b)					
ABSI_z -> Hypertension -> MI	0.0025	0.0005	0.0015	0.0036	<0.001
ABSI_z -> Diabetes -> MI	0.0082	0.0011	0.0062	0.0105	<0.001
ABSI_z -> Hyperlipidemia -> MI	0.0042	0.0006	0.0031	0.0053	<0.001

Note: All coefficients (β) are standardized and reported with four decimal places; SE and 95% bootstrap CIs also have four decimal places. Bootstrap based on 3,000 draws. MI, myocardial infarction; ABSI_z, standardized A Body Shape Index.

Table 4. Proportion mediated

Mediator	Indirect Effect	% of Total Effect	% of Indirect Effect
Hypertension	0.0025	13.4%	16.8%
Diabetes	0.0082	44.3%	55.3%
Hyperlipidemia	0.0042	22.3%	27.9%
Total indirect	0.0149	80.1%	100.0%
Direct effect	0.0037	19.9%	-

Note: Percentages are rounded to one decimal place. Indirect effects are the same as in **Table 3**, shown with four decimal places.

tion. However, the strength of this association varied across subgroups defined by age, smoking status, and alcohol consumption, with a statistically significant interaction observed only for the age subgroup. Conversely, in subgroups based on the presence of hypertension, diabetes, or dyslipidemia, this positive association remained consistently stable within each category. There are subgroup-specific odds ratios (ORs) and their 95% confidence intervals (CIs) in **Figure 2**.

Nonlinear relationship between ABSI and myocardial infarction

Analysis using restricted cubic splines (RCS) combined with the bootstrap method was conducted to assess the relationship between ABSI and myocardial infarction. The distribution of ABSI z-scores in the study population is presented in the lower panel as a frequency histogram, which also indicates the positions of the four RCS knots used to fit the curve in the upper panel. The upper panel demonstrates that as the ABSI z-score increases, the relative odds ratio (OR) for myocardial infarction shows an upward trend. The *P*-value of the nonlinear test is 0.656, and the *P*-value of the Wald test for restricted cubic splines (RCS) is less than 0.001. The association is illustrated in **Figure 3**.

AUC analysis

The comparison of ROC curves based on NHANES weighted data revealed differences in predictive performance for myocardial infarction (MI) among the different models. Survey-weighted ROC analysis was performed on each of the 5 imputed datasets; AUCs were pooled by Rubin's rules. Model 1 (covariates: age, gender, race/ethnicity, education, marital status, poverty-to-income ratio, smoking, alcohol consumption) yielded an AUC of 0.845 (95% CI 0.835-0.855). Adding BMI (Model 2) increased the AUC to 0.850 (95% CI 0.841-0.860), whereas adding ABSI alone (Model 3) yielded 0.848 (95% CI 0.838-0.858). The full model incorporating both ABSI and BMI (Model 4) achieved the highest AUC of 0.853 (95% CI 0.843-0.862). When the 95% confidence interval does

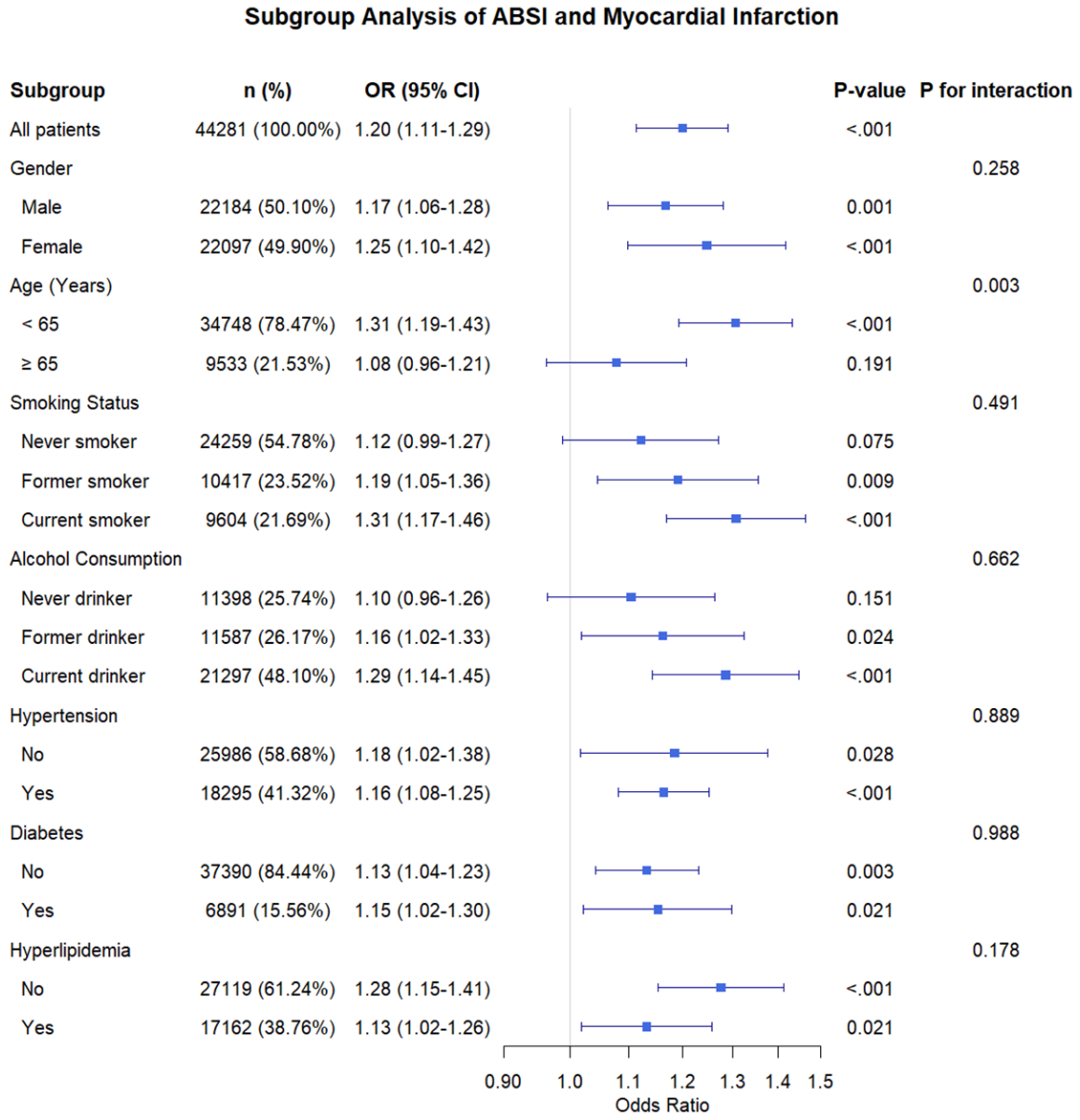

Figure 2. Subgroup analysis.

not contain zero, it corresponds to a *P* value below 0.05, demonstrating statistical significance. See **Figure 4** for details.

Discussion

Using data from 44,281 adults in the National Health and Nutrition Examination Survey (NHANES) from 1999 to 2018, this study examined the association between ABSI and myocardial infarction (MI) and related mechanisms. We found an independent linear association between ABSI and MI risk. Hypertension, diabetes, and hyperlipidemia mediate 80.1% of the association, among which diabetes accounts for 44.3%. Stronger associations were observed among adults aged <65 years in women, smokers, drinkers, and individuals without hyperlipidemia. The area under the curve (AUC) of ABSI for predicting myocardial infarction is 0.848, which is roughly similar to 0.850 of BMI; after combining the two, the area under the curve achieved 0.853.

This study showed that the group with the highest quartile of ABSI has a 73% higher risk of

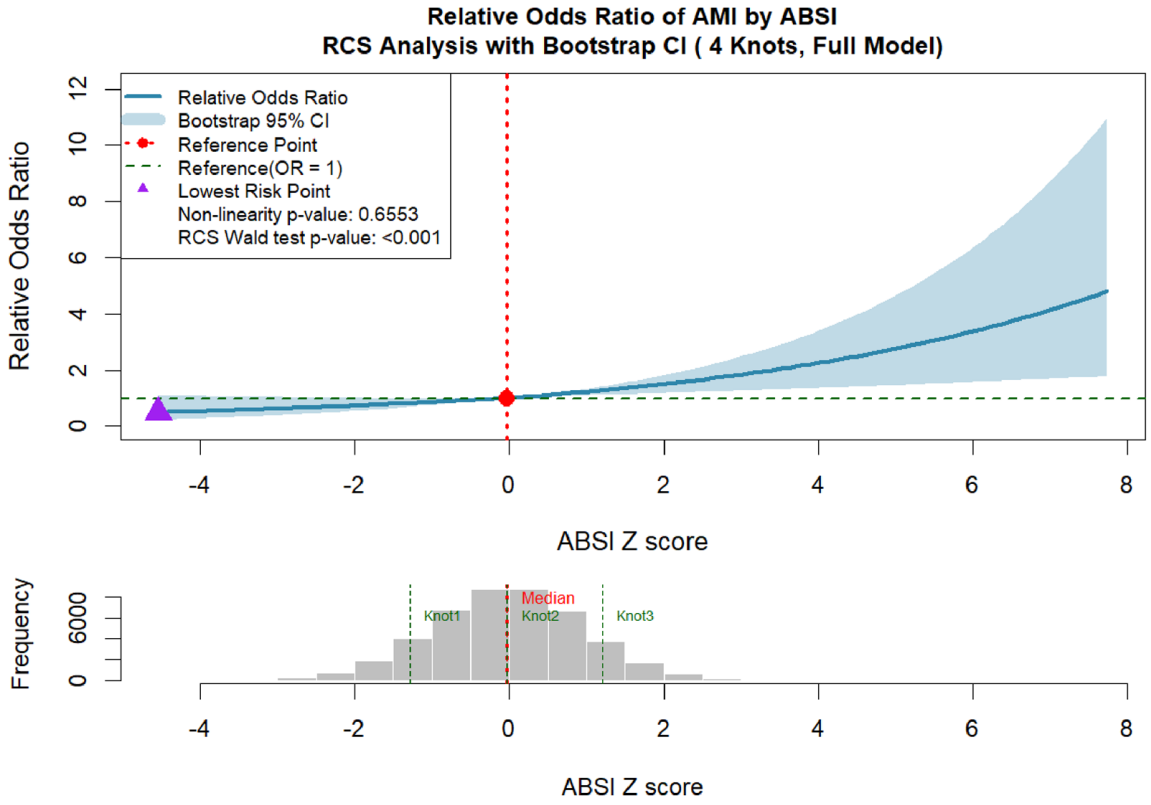

Figure 3. RCS curve.

myocardial infarction than the lowest quartile (OR = 1.73, 95% confidence interval 1.26 to 2.39), which is consistent with previous research results. Krakauer et al. were the first to confirm that ABSI is stronger than BMI and waist circumference in predicting all-cause mortality [15]. Christakoudi et al. confirmed in a European cohort study that ABSI is better for death risk stratification [16]. Kajikawa et al. found that ABSI can independently predict cardiovascular disease events in US adults [23], Zhou et al. pointed out that in normal-weight Chinese adults, ABSI is positively linked to CVD mortality [21]. This study extends these findings to myocardial infarction and confirms it in a nationally representative sample in the US.

There is controversy about the dose-response relationship between ABSI and cardiovascular outcomes. Some studies suggest there may be nonlinear or threshold effects [24], but most lack the ability to detect such patterns. Using data from 44,281 adults and with the help of restricted cubic spline analysis, we found a linear association between ABSI and MI risk (P for nonlinearity = 0.656), which is the same as in recent large cohort studies [18]. This has clinical significance: MI risk gradually increases with the increase of ABSI, and there is an increasing risk even with a moderate increase. These results support using ABSI as a continuous variable for risk stratification. However, there may be nonlinear patterns in specific subgroups (such as diabetic patients) or at extreme values, and verification is needed again [25].

This study for the first time quantifies the risk - associated path of ABSI - MI. The mediation analysis showed that hypertension, diabetes, and hyperlipidemia collectively mediated 80.1% of the total effect, among which diabetes accounts for 44.3%, hyperlipidemia accounts for 22.3%, and hypertension accounts for 13.4%. The research results show that insulin resistance and blood glucose disorders with high visceral fat are key factors for cardiovascular disease risk related to ABSI. This risk appears to be mediated primarily through metabolic abnormalities, not direct cardiac or vascular effects. The practical significance is that

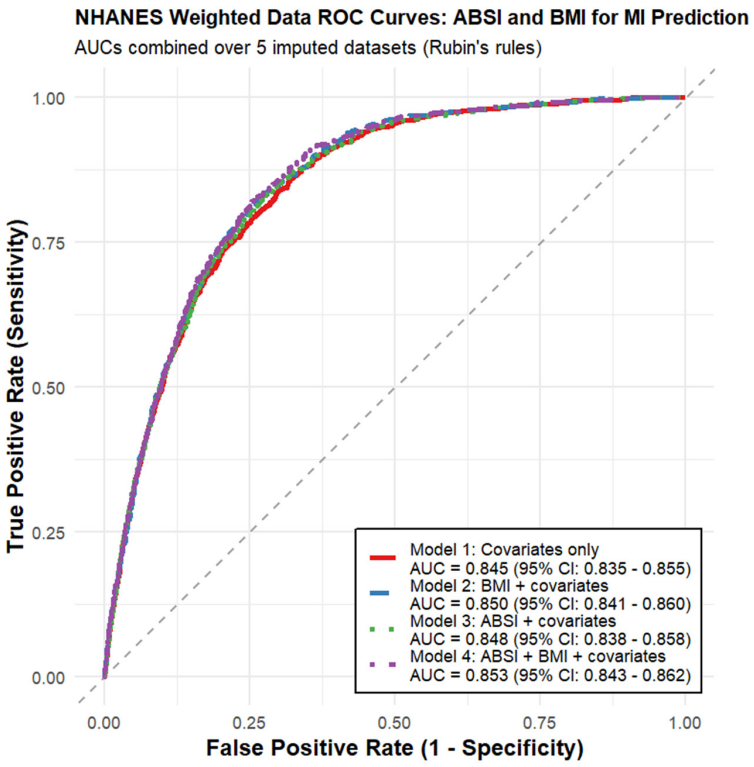

Figure 4. Receiver operating characteristic curve.

for people with high ABSI, interventions should focus on metabolic risk management, especially blood glucose control and diabetes prevention, not just focusing on body shape. The remaining direct effect (19.9%) suggests that inflammation, dysregulation of adipokines or endothelial dysfunction may also be related influencing factors. However, medical models assume that various factors are independent of each other, which may underestimate the synergistic effects among metabolic conditions such as diabetes and hypertension. Future research needs to use more complex models to explore these interactions.

In the subgroup analysis, there was significant heterogeneity in the association between ABSI and myocardial infarction (MI). The association was relatively stronger (OR = 1.31) among adults under 65 years old and relatively weaker (OR = 1.08) among people aged 65 and above. This may reflect disease saturation and competing risks: metabolic diseases mediating the ABSI effect in the elderly often already exist, thus masking the independent effect of ABSI [26-28]. The research results show that ABSI may be an early risk marker for young people, while the elderly need to first pay attention to existing comorbidities.

There is not much difference in gender interaction, but women with a history of pregnancy showed slightly higher risk than men. After menopause, redistribution of visceral fat, which poses a greater threat to women's cardiovascular system [29, 30]. Smoking and alcohol consumption amplified the risk of ABSI-related myocardial infarction, indicating an association between central obesity and an unhealthy lifestyle. Therefore, lifestyle intervention may enhance the cardiovascular benefits of reducing ABSI, especially for people who smoke and drink [31].

Among non-hyperlipidemic individuals, the association between ABSI and MI was stronger (OR = 1.28), than among hyperlipidemic individuals (OR = 1.13). There may be two situations: lipid-lowering treatment in diagnosed hyperlipidemia may offset the risk associated with ABSI; ABSI may identify high-risk people with visceral obesity but normal blood lipids, that is, the metabolically obese normal weight phenotype. This indicates that ABSI has an additional role in detecting subclinical high-risk populations missed by BMI and standard blood lipid measurements [32-34].

In this study, ABSI predicted MI with an AUC of 0.848, comparable to BMI (AUC = 0.850), with modest improvement to 0.853 when combined. Although the overall gain was small, subgroup analyses suggest ABSI adds value by identifying high-risk individuals missed by BMI in specific populations. Ji et al.'s systematic review similarly found that ABSI outperforms traditional indices in detecting metabolic abnormalities among normal-weight individuals [33]. We propose using ABSI as a complement to BMI - particularly in younger adults, those with normal BMI but unhealthy lifestyles, and individuals with suspected visceral adiposity despite appearing metabolically normal.

This study has advantages. First, the NHANES data of 44281 participants from 1999 to 2018 make the results more general. Second, strict methods such as survey-weighted analysis and multiple imputation solve the bias situations of complex sampling and missing data. Third, this is the first study to quantify mediating pathways of the ABSI-MI risk. Hypertension, diabetes, and hyperlipidemia explain 80.1% of the effect, with diabetes accounting for 44.3%, providing evidence for the path of obesity-cardiovascular disease. The fourth subgroup analysis showed that age, gender, and lifestyle can change the effect. The association is stronger in younger adults, smokers or drinkers, and people without metabolic diseases. ABSI also has additional value in people with normal BMI but excessive visceral fat.

There are limitations in this study. First, the cross-sectional study design restricts causal inference and cannot exclude reverse causality; prospective studies need to be used to confirm the direction of association. Second, waist circumference cannot distinguish visceral from subcutaneous fat; there is an association between ABSI and the visceral fat measurement obtained by CT, but direct imaging is the gold standard. Third, lifestyle factors are self-reported, which is prone to recall bias, and data on drug dosage and compliance were unavailable. Fourth, there are no measurements of inflammatory markers and adipokines, which might have led to overestimation of the direct effect. Fifth, mediation analysis assumes path independence, and may not capture the synergistic effects among metabolic diseases. Sixth, NHANES only includes non-institutionalized residents in the United States, and caution should be exercised when extending to other populations or clinical settings.

In summary, ABSI is an independent, linear risk indicator for MI that operates primarily through metabolic syndrome; the magnitude of this association varies by age, gender and lifestyle factors. ABSI may serve as a useful adjunct to BMI in clinical risk stratification. In younger adults and those with unhealthy lifestyles, ABSI could act as a sensitive early marker of metabolic dysregulation and future cardiovascular events. In older or comorbid individuals, aggressive management of established metabolic disease remains paramount. Prospective cohort studies are warranted to validate these findings and determine whether ABSI-guided prevention strategies improve population-level cardiovascular outcomes.

Conclusion

Using NHANES data, we determined that there is an association between ABSI and myocardial infarction (MI), and this association is mediated by hypertension, diabetes, and hyperlipidemia. This association is most pronounced in people under 65 years old, smokers, drinkers, and those without metabolic diseases. The predictive performance of ABSI is not worse than that of BMI, captures the risk of central obesity not captured by BMI, and can also identify individuals with “normal metabolism” but excess visceral fat. In clinical practice, the interpretation of ABSI needs to be combined with age, gender, and lifestyle; younger high-risk individuals should receive intensified interventions, and older patients with comorbidities should prioritize disease management.

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All NHANES participants provided written informed consent.

Disclosure of conflict of interest

None.

Abbreviations

ABSI, A Body Shape Index; MI, Myocardial Infarction; AUC, Area Under the Curve; BMI, Body Mass Index; CHD, Coronary Heart Disease; CI, Confidence Interval; CVD, Cardiovascular Diseases; DBP, Diastolic Blood Pressure; FBS, Fasting Blood Sugar; GLP-1RA, Glucagon-like peptide-1 receptor agonist; HbA1c, Hemoglobin A1c; HDL-C, High-Density Lipoprotein Cholesterol; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance; LDL-C, Low-Density Lipoprotein Cholesterol; NA, Not Applicable/Not Available; NHANES, National Health and Nutrition Examination Survey; OR, Odds Ratio; PCI, Percutaneous Coronary Intervention; RCS, Restricted Cubic Spline; ROC, Receiver Operating Characteristic; SBP, Systolic Blood Pressure; SE, Standard Error; SGLT2i,

Sodium-glucose cotransporter-2 inhibitor; TG, Triglycerides; UA, Uric Acid; WC, Waist Circumference; WHR, Waist-to-Hip Ratio.

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