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Original Research

Association between Non-High-Density Lipoprotein Cholesterol to High-Density Lipoprotein Cholesterol Ratio and Abdominal aortic calcification：A cross-sectional NHANES study

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Abstract

Background and Aim The ratio of non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol (NHHR) is an emerging lipid index that may surpass traditional single lipid indices in evaluating cardiovascular disease risk. This study aims to explore the relationship between NHHR and abdominal aortic calcification (AAC) in adult populations. **Methods** This study assessed the association between levels of the NHHR and AAC using data from the 2013-2014 National Health and Nutrition Examination Survey (NHANES). The NHHR was calculated as the ratio of non-HDL-C to HDL-C. AAC was evaluated using dual-energy X-ray absorptiometry data. The analysis employed multivariate logistic regression, sensitivity analysis, and smoothing curve fitting to examine the NHHR-AAC relationship. Additionally, subgroup analyses and interaction tests were conducted to determine the consistency of this association across different populations. **Results** The study included 3,030 participants, with 26.24% identified as having AAC. There was a significant positive relationship between NHHR and AAC: each unit increase in NHHR was associated with 15% higher odds of AAC prevalence [OR 1.15 (95% CI 1.08, 1.24)]. Participants in the highest NHHR quartile had 71% greater odds of AAC prevalence compared to those in the lowest quartile [OR 1.71 (95% CI 1.30, 2.26)]. This association was particularly strong among individuals with a BMI under 25. **Conclusions** Our research findings reveal a significant correlation between the NHHR and AAC among adult populations in the United States. Given its ease of use and effectiveness, monitoring NHHR could serve as a valuable early intervention strategy to prevent AAC

Keywords: non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol ratio, Lipid ratio, abdominal aortic calcification, NHANES

1. Introduction

The abdominal aortic calcification (AAC) is a manifestation of atherosclerosis in the abdominal aorta [1,2]. A study found that the prevalence of AAC across the United States is 28.8% [3]. Moreover, the incidence of AAC is notably higher in individuals of advanced age, as well as those with smoking habits and hypertension, compared to the general population. Although AAC is typically asymptomatic, a growing body of evidence suggests that it is associated with an increased risk of developing cardiovascular diseases [4,5]. A meta-analysis has shown that patients with AAC have a significantly higher relative risk of experiencing coronary artery events, cerebrovascular events, all cardiovascular events,

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and cardiovascular mortality compared to those in the control group [6]. Therefore, it is critically important to conduct in-depth research on AAC and explore its relationship with cardiovascular risk factors.

The ratio of non-high-density lipoprotein cholesterol to high-density lipoprotein cholesterol (NHHR) is a novel indicator that reflects lipid levels and atherosclerotic burden [7,8]. Recent research indicates that NHHR is linked with various cardiovascular diseases [9-11]. Specifically, in the Chinese population, NHHR shows an independent association with abdominal aortic aneurysm [12]. Further studies have established that NHHR is negatively correlated with the development of coronary collateral circulation in patients with chronic total occlusion of the coronary arteries [13,14]. Furthermore, NHHR may increase coronary artery disease risk and negatively impact non-ST-segment elevation myocardial infarction prognosis [15,16].

Lipid levels are among the most critical factors influencing atherosclerosis and are closely related to the occurrence of AAC. Although the NHHR is a reliable lipid indicator associated with atherosclerotic characteristics, its relationship with AAC has not been extensively studied. Therefore, this study aims to conduct a cross-sectional analysis using the National Health and Nutrition Examination Survey (NHANES) database to explore the relationship between NHHR and AAC.

2. Methods

2.1 Study population

The National Health and Nutrition Examination Survey (NHANES) database for 2013-2014 includes data from 10,176 participants, comprising demographic characteristics, socioeconomic information, dietary data, health-related questions, structured interviews, physical examinations, and laboratory test results. After excluding individuals with missing data on AAC or on high-density lipoprotein cholesterol and total cholesterol, the study included 3,030 participants. The participant inclusion flowchart is presented in Figure 1. Informed consent was obtained from all participants before any research procedures were conducted, and all research procedures were approved by the National Center for Health Statistics Institutional Review Board. More detailed information about the process is available at the Centers for Disease Control and Prevention (CDC) website

2.2 Assessment of NHHR

The calculation method for the NHHR is defined as NHHR = Non-HDL-c / HDL-c[17]. Since the NHANES database does not provide direct data for non-high-density lipoprotein cholesterol, Non-HDL-c is calculated by subtracting high-density lipoprotein cholesterol (HDL-c) from total cholesterol (TC): NHHR = (TC - HDL-c) / HDL-c.[18]

2.3 Abdominal aortic calcification evaluation

Abdominal aortic calcification scores are determin ed using dual-energy X-ray absorptiometry (DXA) of the lateral lumbar spine, encompassing lumbar vertebrae L1-L4, according to the Kauppila scoring system. This system grades the degree of calcifica tion on the anterior and posterior aortic walls at eac h vertebral level from L1 to L4, using a scale of 0-3. The total Kauppila score ranges from 0 to 24, wi th scores of 6 or higher indicating severe abdomina l aortic calcification. All DXA scans are performed and analyzed by trained technicians using the sam e scanner. Further details are available on the CDC website (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/DXXAAC_H.htm).

2.4 Covariate

Demographic information was collected via standardized questionnaires, covering gender, age, race, education level, marital status, and income-to-poverty ratio (PIR). Health-related data included blood pressure, fasting blood glucose, oral glucose tolerance test (OGTT), glycated hemoglobin (HbA1c), vitamin D, serum calcium, apolipoprotein B (ApoB), triglycerides, and low-density lipoprotein cholesterol (LDL-c). Body Mass Index (BMI), systolic and diastolic blood pressure were measured by medical professionals. Hypertension was defined based on either a sustained blood pressure of ≥140 mmHg systolic or ≥90 mmHg diastolic over three consecutive measurements, or a physician-diagnosed history. Diabetes was defined by a fasting blood glucose level of ≥7.0 mmol/L, an OGTT result of ≥11.1 mmol/L, an HbA1c of ≥6.5%, or a physician-confirmed diagnosis. Smoking status was categorized into daily, occasional, or non-smoker based on current usage, and drinking status was assessed based on consumption of alcohol at least 12 times in the past year. Total cholesterol, HDL, LDL, triglycerides, HbA1c, and fasting glucose levels were quantified using standard biochemical techniques. Missing data for all covariates were addressed through multiple imputation.

2.5 Statistic analysis

We used the R software version 4.3.1 and Free-Statistics version 2.0 for all statistical analyses. Differences in continuous variables were examined using Analysis of Variance (ANOVA), while chi-square tests were employed for categorical variables. To explore the relationship between the NHHR and AAC, logistic regression models were utilized. Model 1 was unadjusted; Model 2 adjusted for age, gender, and race; and Model 3 additionally adjusted for marital status, serum calcium, low-density lipoprotein cholesterol (LDL-c), Body Mass Index (BMI), and drinking status. NHHR was categorized into quintiles to examine the linear trend in the association with AAC using a trend test. Furthermore, we also performed curve fitting and subgroup analyses using fully adjusted models to determine the relationship between NHHR and AAC and to verify that this relationship was consistent across subgroups. Statistical significance was set at $p < 0.05$.

3. Results

3.1 Baseline characteristics of participants

A total of 3,030 participants were included in the study, with 26.24% presenting with AAC and 10.86% presenting with severe AAC. The average age of participants was 58.61 ± 12.00 years. Of these, 48.15% were male, and 44.39% were non-Hispanic whites. The NHHR was categorized into quartiles: Q1 (<1.91), Q2 (1.92-2.64), Q3 (2.65-3.61), Q4 (>3.62). Participants in the higher NHHR quartiles were predominantly male, non-Hispanic white, more educated (some college), married, and had a higher prevalence of hypertension and diabetes. Baseline characteristics are detailed in Table 1.

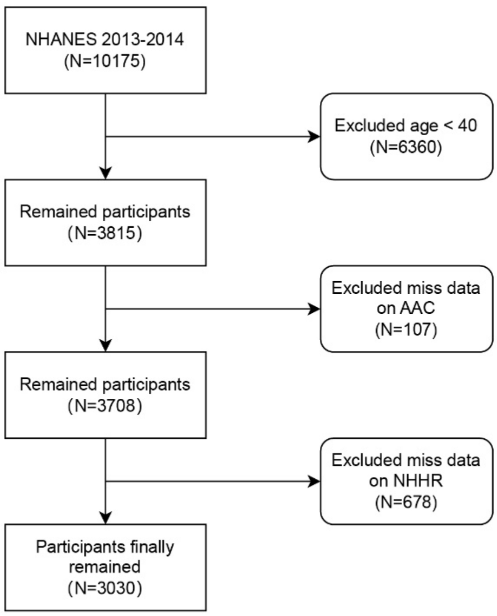


Figure 1 Flow chart of participants selection. NHANES, National Health and Nutrition Examination Survey.

Table 1 Characteristics of U.S. adults participating in the NHHR

Variables	Total (n = 3030)	NHHR				*P*
		Q1 (n = 758)	Q2 (n = 757)	Q3 (n = 756)	Q4 (n = 759)	
Age(years), Mean ± SD	58.61 ± 12.00	60.28 ± 12.79	59.83 ± 11.95	57.91 ± 11.51	56.42 ± 11.33	<0.001
Sex, n (%)						<0.001
Male	1459 (48.15)	287 (37.86)	311 (41.08)	393 (51.98)	468 (61.66)	
Female	1571 (51.85)	471 (62.14)	446 (58.92)	363 (48.02)	291 (38.34)	
Race, n (%)						<0.001
Mexican American	401 (13.23)	72 (9.50)	84 (11.10)	116 (15.34)	129 (17.00)	
Other Hispanic	286 (9.44)	46 (6.07)	76 (10.04)	73 (9.66)	91 (11.99)	
Non-Hispanic White	1345 (44.39)	364 (48.02)	327 (43.20)	324 (42.86)	330 (43.48)	
Non-Hispanic Black	582 (19.21)	187 (24.67)	158 (20.87)	130 (17.20)	107 (14.10)	
Other Race	416 (13.73)	89 (11.74)	112 (14.80)	113 (14.95)	102 (13.44)	
Education, n (%)						<0.001
Less Than 9th	284 (9.37)	47 (6.20)	68 (8.98)	73 (9.66)	96 (12.65)	
9–11th	409 (13.50)	100 (13.19)	91 (12.02)	106 (14.02)	112 (14.76)	
High School	685 (22.61)	160 (21.11)	154 (20.34)	188 (24.87)	183 (24.11)	
Some College	852 (28.12)	219 (28.89)	215 (28.40)	204 (26.98)	214 (28.19)	
College Graduate	800 (26.40)	232 (30.61)	229 (30.25)	185 (24.47)	154 (20.29)	
PIR, Mean ± SD	2.69 ± 1.64	2.88 ± 1.67	2.77 ± 1.65	2.61 ± 1.62	2.49 ± 1.61	<0.001
BMI, Mean ± SD	28.46 ± 5.56	26.43 ± 5.50	28.26 ± 5.53	29.19 ± 5.63	29.97 ± 4.90	<0.001
Ca(mmol/L), Mean ± SD	2.36 ± 0.09	2.36 ± 0.09	2.36 ± 0.10	2.36 ± 0.09	2.37 ± 0.09	0.425
TG (mg/dL), Mean ± SD	125.16 ± 135.02	94.45 ± 69.33	107.24 ± 59.44	128.74 ± 164.95	170.16 ± 184.71	<0.001
TC (mg/dL), Mean ± SD	194.91 ± 43.71	173.46 ± 36.17	186.06 ± 35.36	195.23 ± 34.53	224.83 ± 49.60	<0.001
HDL (mg/dL), Mean ± SD	54.08 ± 16.80	71.44 ± 17.57	57.14 ± 11.27	48.04 ± 8.67	39.71 ± 7.99	<0.001
LDL (mg/dL), Mean ± SD	115.46 ± 39.23	101.10 ± 36.93	112.20 ± 34.04	120.38 ± 36.38	128.13 ± 43.73	<0.001
HbA1c (%), Mean ± SD	5.92 ± 1.16	5.68 ± 0.88	5.89 ± 1.12	5.96 ± 1.17	6.15 ± 1.37	<0.001
OGTT (mmol/L), Mean ± SD	8.12 ± 4.36	7.43 ± 3.69	7.98 ± 4.10	8.25 ± 4.40	8.83 ± 5.02	<0.001
ApoB(mg/dL), Mean ± SD	94.03 ± 26.33	81.48 ± 25.35	90.07 ± 22.05	97.29 ± 22.54	107.27 ± 27.89	<0.001
VitD(nmol/L), Mean ± SD	65.32 ± 29.18	69.92 ± 32.60	67.12 ± 29.77	64.08 ± 28.21	60.17 ± 24.74	<.001
Marital, n (%)						0.013
No	1207 (39.83)	332 (43.80)	312 (41.22)	289 (38.23)	274 (36.10)	
Yes	1823 (60.17)	426 (56.20)	445 (58.78)	467 (61.77)	485 (63.90)	
Current smoke, n (%)						0.021
Everyday	950 (31.35)	237 (31.27)	215 (28.40)	225 (29.76)	273 (35.97)	
Sometimes	204 (6.73)	59 (7.78)	44 (5.81)	55 (7.28)	46 (6.06)	
No	1876 (61.91)	462 (60.95)	498 (65.79)	476 (62.96)	440 (57.97)	
Diabetes, n (%)						<0.001
No	2212 (73.00)	588 (77.57)	567 (74.90)	535 (70.77)	522 (68.77)	
Yes	818 (27.00)	170 (22.43)	190 (25.10)	221 (29.23)	237 (31.23)	

Hypertension, n (%)						0.617
No	1461 (48.22)	380 (50.13)	354 (46.76)	362 (47.88)	365 (48.09)	
Yes	1569 (51.78)	378 (49.87)	403 (53.24)	394 (52.12)	394 (51.91)	
Drinker, n (%)						0.413
No	865 (28.55)	211 (27.84)	203 (26.82)	231 (30.56)	220 (28.99)	
Yes	2165 (71.45)	547 (72.16)	554 (73.18)	525 (69.44)	539 (71.01)	
AAC, n (%)						0.672
No	2235 (73.76)	568 (74.93)	548 (72.39)	554 (73.28)	565 (74.44)	
Yes	795 (26.24)	190 (25.07)	209 (27.61)	202 (26.72)	194 (25.56)	
Severe AAC, n (%)						0.104
No	2701 (89.14)	661 (87.2)	670 (88.51)	687 (90.87)	683 (89.99)	
Yes	329 (10.86)	97 (12.8)	87 (11.49)	69 (9.13)	76 (10.01)	

F: ANOVA; χ^2: Chi-square test; SD: standard deviation

3.2 Association of NHHR and AAC

The association between the NHHR and AAC is shown in Table 2. In the fully adjusted continuous model, NHHR demonstrated a significant association with AAC, with an odds ratio (OR) of 1.15 (95% CI: 1.08-1.24, P<0.001), indicating a 15% increase in the prevalence of AAC. In Model 2, adjusted only for gender, age, and race, the association remained significant (OR: 1.10, 95% CI: 1.03-1.17, P=0.004), showing a 10% increase in AAC prevalence. In the quartile-based regression analysis, Model 1, unadjusted, showed no significant correlation. However, Model 2, adjusted for gender, age, and race, revealed significant associations for NHHR quartiles Q3 and Q4, with ORs ranging from 1.05 to 1.73 and 1.07 to 1.78, respectively (P<0.05). Model 3, which included adjustments for gender, age, race, marital status, BMI, serum calcium, LDL cholesterol, and drinking status, found that higher NHHR quartiles significantly increased AAC prevalence, with a 71% increase in the Q4 group (OR: 1.30-2.26, P<0.05). The smooth curve fitting analysis depicted in Figure 2 shows a nonlinear relationship between NHHR and AAC, with a P-value for nonlinearity of 0.067 and a significant positive association when NHHR exceeds 2.649.

Figure 2 The nonlinear associations between NHHR and AAC. Red line shows the relationship between NHHR and the odds ratio of AAC. Shaded Area (Pink) represents the confidence interval around the estimate, indicating the range of values that are likely to include the true value for the population. Blue bars likely represent a histogram of the distribution of NHHR values within the dataset used for the analysis.

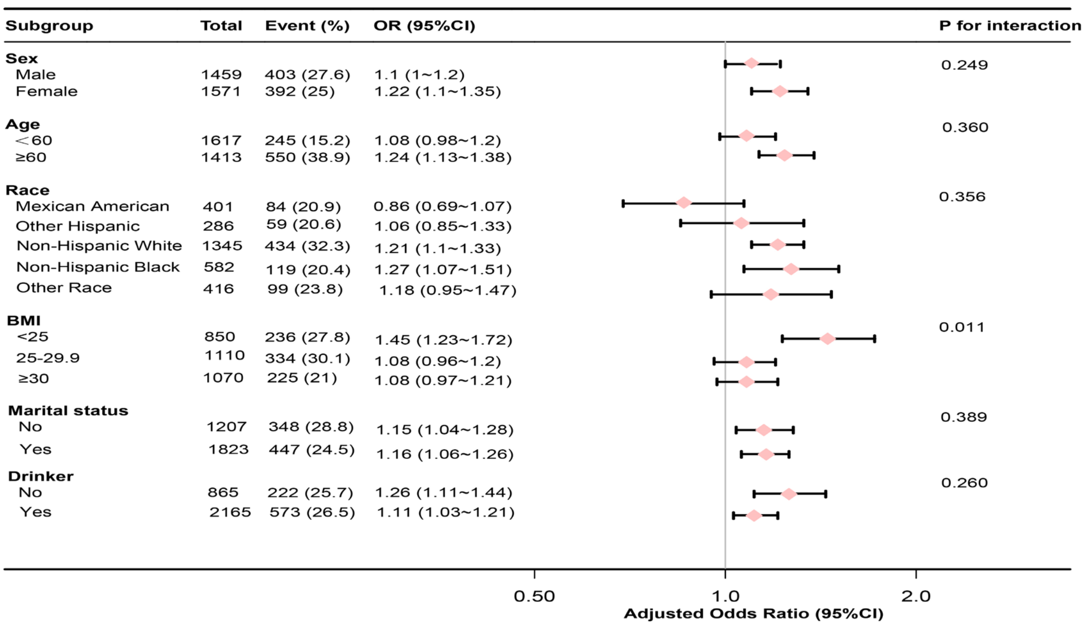
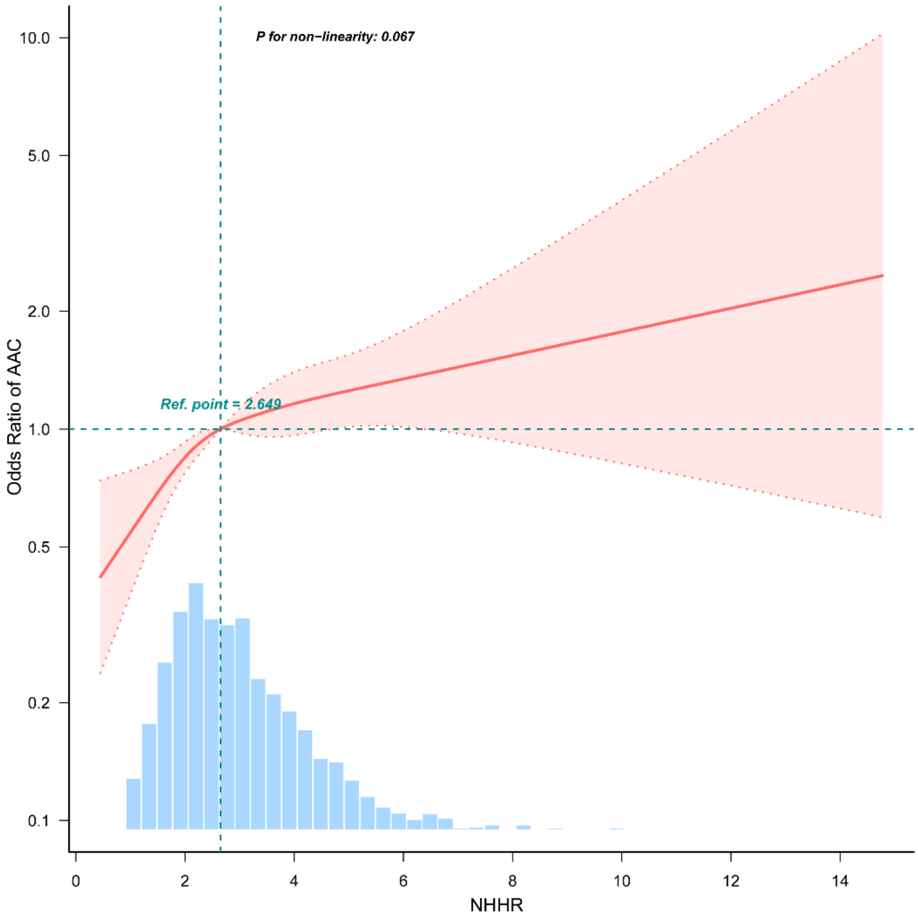


Table 2 Associations between NHHR and abdominal aortic calcification

Variables	Model 1		Model 2		Model 3	
	OR (95%CI)	*P*	OR (95%CI)	*P*	OR (95%CI)	*P*
Continuous NHHR	1.00 (0.94 ~ 1.06)	0.986	1.10 (1.03 ~ 1.17)	0.004	1.15 (1.08 ~ 1.24)	<0.001
Quantile						
1	1.00 (Reference)		1.00 (Reference)		1.00 (Reference)	
2	1.14 (0.91 ~ 1.43)	0.261	1.24 (0.97 ~ 1.59)	0.087	1.37 (1.06 ~ 1.76)	0.015
3	1.09 (0.87 ~ 1.37)	0.463	0.34 (1.05 ~ 1.73)	0.021	1.60 (1.23 ~ 2.08)	<0.001
4	1.03 (0.81 ~ 1.29)	0.825	1.38 (1.07 ~ 1.78)	0.014	1.71 (1.30 ~ 2.26)	<0.001
P for trend		0.946		0.019		<0.001

OR: Odds Ratio; CI: Confidence Interval; Model1: Crude; Model2: Adjust: Sex, Age, Race; Model3: Adjust: Sex, Age, Race, Marital status, BMI, Ca, LDL, Drinker

Figure 3 the result of subgroup analysis and interaction



3.3 Subgroup analysis

In the subgroup analysis of the fully adjusted model, stratified by factors including age, gender, race, marital status, drinking status, and BMI, we found that there was a difference between different sexes, races and BMI levels. Furthermore, the results, displayed in Figure 3, indicated a significant variation in the NHHR-AAC relationship across different BMI subgroups, with an interaction p-value of 0.011. Specifically, in individuals with a BMI less than 25, each unit increase in NHHR was associated with a 45% increased risk of AAC. Conversely, no significant association between NHHR and AAC was observed in individuals with a BMI ranging from 25 to 29.9 or those with a BMI of 30 or greater.

4. Discussion

It is the first study to investigate the relationship between NHHR and AAC to our knowledge. In our cross-sectional analysis of 3,030 participants, we observed a positive correlation between NHHR and AAC. Notably, higherprevalence of AAC with higher NHHR levels. Furthermore, we found that BMI influences the stability of this relationship, suggesting that individuals with lower BMI may need to monitor NHHR more closely.

An increasing body of evidence underscores the significant role of lipid metabolism in cardiovascular diseases [19-21]. For instance, a prospective cohort study demonstrated that elevated serum remnant cholesterol is linked to increased mortality in cardiovascular disease patients and reduced mortality in those with liver and stomach cancers [22]. Moreover, cholesterol efflux related to HDL is pivotal in the formation of atherosclerotic plaques [23]. Rikhi and colleagues [24] identified that high levels of small dense low-density lipoprotein cholesterol (sd-LDL-C) elevate the risk of coronary artery calcification. Similarly, AAC is strongly associated with various cardiovascular diseases [25] and can impact the clinical outcomes in acute aortic dissection by limiting the distal extent and affecting the vascular supply from the true lumen [26]. Additionally, the AAC score has been shown to predict all-cause and cardiovascular mortality in patients undergoing maintenance hemodialysis, with significantly higher mortality rates in patients with an AAC score greater than 4.5 [27]. The abdominal aortic calcification index (ACI), which quantifies the extent of calcification, has also been found effective in predicting mid-term cardiovascular events post-percutaneous coronary intervention in acute coronary syndrome patients [28].

One plausible explanation is that the NHHR may escalate the risk of cardiovascular diseases by influencing atherosclerosis in blood vessels[29]. Cholesterol plays a critical role in the genesis, proliferation, activation, and signaling of immune cells, which contributes to the onset and progression of atherosclerosis [30]. Furthermore, 25-hydroxycholesterol has been shown to promote the calcification of vascular smooth muscle cells through the activation of endoplasmic reticulum stress [31]. Additionally, in the context of disrupted cholesterol metabolism, reactive oxygen species (ROS) can hasten the osseous differentiation of vascular smooth muscle cells, leading to vascular calcification [32].

Nevertheless, the cross-sectional design of our study precludes the determination of causality between NHHR and AAC; thus, further prospective studies are warranted. Additionally, some data were collected through self-reporting, which may be prone to recall bias. Also, since the study population predominantly comprised non-Hispanic whites and non-Hispanic blacks, caution should be exercised in generalizing these results to other ethnic groups.

The purpose of this study was to evaluate whether the NHHR is associated with the occurrence of AAC. After adjusting for confounding variables, we observed a significant increase in AAC prevalence with rising NHHR levels. NHHR, being an accessible and economical measure from standard blood tests, could be key in the progression of AAC. Further research is required to elucidate the underlying mechanisms linking NHHR with AAC.

Data availability: The data were derived from the NHANES database, a publicly accessible and freel y available resource available at https://www.cdc.g ov/nchs/nhanes.

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