International Journal of General Medicine

Dovepress
Taylor & Francis Group

Open Access Full Text Article

ORIGINAL RESEARCH

Aldehyde Dehydrogenase 2 rs671 Polymorphism is Associated with Susceptibility of Coronary Atherosclerosis in Patients with Hypertension

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Objective: Predisposing factors for coronary atherosclerosis in hypertensive individuals are unclear. Atherosclerosis is a chronic inflammatory disease caused by lipid deposition in the blood vessels, and aldehyde dehydrogenase 2 (ALDH2) is involved in this process. The aim of this study was to assess the relationship between *ALDH2* rs671 polymorphism, serum lipids, peripheral inflammation indices (pan-immune inflammation value (PIV), systemic immune inflammation index (SII), and system inflammation response index (SIRI)) and coronary atherosclerosis risk in hypertensive patients.

Methods: A total of 923 patients with hypertension (439 patients with coronary atherosclerosis, and 484 without) who were admitted to Meizhou People's Hospital between January 2019 and February 2024 were retrospectively analyzed. The relationship between *ALDH2* rs671 polymorphism, serum lipid levels, and peripheral inflammation indices and the risk of coronary atherosclerosis was analyzed.

Results: There were 532 (57.6%), 337 (36.5%), and 54 (5.9%) individuals with *ALDH2* rs671 G/G, G/A, and A/A genotype, respectively. The frequency of the *ALDH2* rs671 G/A genotype, and the levels of TC, LDL-C, PIV, SII, and SIRI in patients with coronary atherosclerosis were higher than those in controls. Logistic analysis showed that body mass index (BMI) ≥24.0 kg/m^2 (odds ratio (OR): 1.670, 95% confidence interval (CI): 1.185–2.352, p=0.003), history of smoking (OR: 2.024, 95% CI: 1.263–3.243, p=0.003), *ALDH2* rs671 G/A genotype (OR: 1.821, 95% CI: 1.280–2.589, p=0.001), high TC (OR: 1.592, 95% CI: 1.021–2.485, p=0.040), high SII (OR: 2.290, 95% CI: 1.386–3.784, p=0.001), and high SIRI (OR: 1.727, 95% CI: 1.126–2.650, p=0.012) were associated with coronary atherosclerosis in patients with hypertension.

Conclusion: Overweight (BMI ≥24.0 kg/m^2), history of smoking, *ALDH2* rs671 G/A genotype, high TC, SII, and SIRI levels were associated risk factors for coronary atherosclerosis in patients with hypertension.

Keywords: coronary atherosclerosis, hypertension, *ALDH2*, systemic immune inflammation index, system inflammation response index

Introduction

Since the 20th century, chronic non-communicable diseases have gradually become the main diseases in the spectrum of diseases, accounting for more than 60% of disability-adjusted life years (DALYs).[1,2] Cardiovascular disease (CVD) has become a key public health problem that endangers the life and health of global residents owing to its high prevalence and fatality rate, accounting for 24% of DALYs related to chronic non-communicable diseases.[3] Atherosclerotic cardiovascular disease (ASCVD) is a major cause of death and disease burden globally.[4] Coronary atherosclerosis refers to the lipid deposition of the intima and subintima of the coronary artery wall, resulting in intima migration and proliferation.[5,6] Coronary atherosclerosis is caused by many pathogenic factors, and its risk factors, including age, gender, high blood pressure, diabetes and unhealthy lifestyle.[7]

Received: 17 October 2024
Accepted: 2 February 2025
Published: 10 February 2025

Hypertension is a risk factor for stroke, coronary atherosclerosis, and other CVDs, and an increase of blood pressure is positively correlated with the occurrence of cardiovascular events.[8] More than 70% of patients with CVD cases are caused by some controllable factors, of which hypertension is the most important risk factor with a population attributable fraction (PAFs) of 22.3%. Individuals with hypertension have a higher likelihood of developing coronary atherosclerosis than those without.[9,10] Hypertension combined with coronary atherosclerosis is a common cardiovascular disease in the population, and the prevalence rate is increasing year by year.[11,12] It is well known that hypertension is a risk factor for coronary atherosclerosis, and some hypertensive patients are susceptible to coronary atherosclerosis, while some hypertensive patients are not susceptible to coronary atherosclerosis. Are other factors influencing the difference of coronary atherosclerosis risk in the same high-risk population? And what are the factors that affect it? The purpose of this study was to identify the differences between coronary atherosclerosis and non-coronary atherosclerosis patients in hypertensive people and the risk factors for coronary atherosclerosis in hypertensive people. Understanding the risk factors of coronary atherosclerosis in hypertensive individuals can effectively reduce the incidence of coronary atherosclerosis.

Dyslipidemia is a risk factor for atherosclerosis (AS) formation.[13] In the early stage of AS, endothelial cell function is impaired, vascular intimal permeability increases, circulating lipoprotein enters the intima, and low-density lipoprotein cholesterol (LDL) is oxidized to form oxidized low-density lipoprotein (ox-LDL). The scavenger receptor on the surface of macrophages engulfs and absorbs ox-LDL, resulting in the production of foam cells that are rich in cholesterol esters.[14] Whether dyslipidemia is a risk factor for coronary atherosclerosis remains unclear. On the other hand, inflammation is also involved in the development of atherosclerosis.[15] The inflammatory theory holds that monocyte aggregation is the initiating factor of AS, and that monocytes with pro-inflammatory activity preferentially gather in atherosclerotic plaques and adhere to endothelial cells stimulated by cytokines.[16] Mononuclear cells adhering to the vascular endothelium enter the inner membrane and differentiate into macrophages, and phagocytic a large amount of ox-LDL is transformed into foam cells. Macrophages induce the production of several inflammatory factors. Under the action of pro-inflammatory factors, more immune cells enter the plaque and accelerate plaque, development of plaque.[17] In recent years, some comprehensive inflammatory indices converted from peripheral blood cell counts have received some attention in the diagnosis and prognosis evaluation of some diseases, such as the pan-immune inflammation value (PIV), systemic immune inflammation index (SII), and system inflammation response index (SIRI). Several studies have suggested that PIV,[18,19] SII,[20,21] and SIRI[22] are associated with CVDs. However, the relationship between these inflammatory indices and coronary atherosclerosis in patients with hypertension has not been studied.

In addition, aldehyde dehydrogenase 2 (ALDH2) participates in the oxidation of toxic aldehyde from myocardial metabolism 4-hydroxy-2-nonenal (4-HNE).[23,24] Several studies have confirmed that ALDH2 plays a protective role in hypertension, coronary artery disease, and myocardial infarction by metabolizing toxic aldehydes, changing drinking habits, and mediating nitric oxide production.[25,26] The activity status of ALDH2 is influenced by the polymorphisms of its coding gene, *ALDH2* gene.[27] SNP rs671 (G>A) is an important polymorphism in *ALDH2* gene, and a variant of this polymorphism can lead to the decrease of ALDH2 enzyme activity.[28,29] *ALDH2* gene polymorphism was associated with coronary atherosclerosis,[30–32] but these studies are basically based on general population studies, and this relationship needs to be clarified in hypertensive people. We aimed to investigate the relationship between serum lipid levels, comprehensive inflammatory indices, and *ALDH2* rs671 polymorphism and susceptibility to coronary atherosclerosis in patients with hypertension.

Materials and Methods

Study Participants and Data Collection

A total of 923 patients with hypertension who were admitted to the Meizhou People's Hospital between January 2019 and February 2024 were retrospectively analyzed. The inclusion criteria were as follows: (1) patients who met the diagnostic criteria for hypertension (a mean SBP >140 mmHg and/or a mean DBP >90 mmHg);[33] (2) patients diagnosed with coronary atherosclerosis; (3) age ≥18 years old; and (4) complete clinical data. Criteria for the diagnosis of coronary atherosclerosis: coronary angiography (CAG) showed that at least one of the major epicardial vessels (including left main

branch, anterior descending branch, circumflex branch, and right coronary artery) had a diameter stenosis, or a clinical diagnosis of myocardial infarction.[34,35] The inclusion criteria for controls were as follows: (1) patients who met the diagnostic criteria for hypertension, (2) individuals with non-coronary atherosclerosis who had undergone *ALDH2* gene polymorphisms, and (3) individuals with complete information. These hypertensive patients were divided into a study group (those with coronary atherosclerosis) and a control group (those without coronary atherosclerosis).

Collection of Clinical Data

Information such as age, sex, body mass index (BMI), history of smoking, history of alcohol consumption, history of diabetes mellitus, and *ALDH2* rs671 genotype were collected. In this study, BMI was divided into three grades: <18.5 kg/m^2, 18.5–23.9 kg/m^2, and ≥24.0 kg/m^2 according to the standard of the Chinese population.[36,37] Routine blood test data were collected before the treatment. The patient's venous blood was collected and blood cell analysis was performed using a Sysmex XE-2100 hematology analyzer (Sysmex Corporation, Japan). Serum lipids (total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C)) levels were assessed using automatic biochemical analysis system (Olympus AU5400 system, Tokyo, Japan). *ALDH2* genotype was amplified by PCR - microarray method (BaiO Technology Co, Ltd., Shanghai, China).

Statistical Analysis

Continuous variables were compared using either Student's *t*-test or the Mann–Whitney *U*-test. Comparison of the genotype composition ratio and allele frequency between individuals with and without coronary atherosclerosis was analyzed using the χ^2 test. Logistic regression analysis was used to examine the relationship between *ALDH2* rs671 genotypes and alleles and coronary atherosclerosis in patients with hypertension. $p<0.05$ was considered to represent statistical significance. All statistical analyses were performed using SPSS statistical software version 26.0 (IBM Inc., USA).

Results

Characteristics of Subjects

In this study, 660 (71.5%) male individuals and 263 (28.5%) female individuals had hypertension. There were 452 (49.0%) subjects with BMI range 18.5–23.9 kg/m^2, and 420 (45.5%) with ≥24 kg/m^2. In total 229 (24.8%) patients had a history of smoking, 132 (14.3%) patients with alcohol consumption, and 339 (36.7%) patients had diabetes mellitus, respectively (Table 1).

There were significant differences in the distribution of BMI grades ($p=0.049$), and proportions of history of alcoholism ($p<0.001$) and diabetes mellitus ($p=0.007$) between the study and control groups. There was no significant difference in the distribution of gender, proportion of history of smoking between the two groups (all $p>0.05$). The patients with coronary atherosclerosis had higher TC (4.65±1.22 vs 4.38±1.68 mmol/L, $p=0.013$) and LDL-C (2.64±0.89 vs 2.41±1.19 mmol/L, $p=0.004$), and PIV (522.02 (240.15, 1102.71) vs 349.92 (143.46, 834.34), $p<0.001$), SII (972.00 (551.38, 1767.33) vs 659.93 (323.50, 1398.65), $p<0.001$), and SIRI (2.48 (1.17, 5.09) vs 1.94 (0.98, 3.82), $p=0.001$) levels than those of controls (Table 1).

Distribution Frequencies of ALDH2 rs671 Genotypes and Alleles in Patients with Coronary Atherosclerosis and Controls

There were 532 (57.6%), 337 (36.5%), and 54 (5.9%) individuals with *ALDH2* rs671 G/G, G/A, and A/A genotype, respectively. The *ALDH2* rs671 genotypes in the patients with coronary atherosclerosis ($\chi^2=0.595$, $p=0.441$), and controls ($\chi^2=0.070$, $p=0.791$) conformed to the Hardy-Weinberg equilibrium, respectively. The frequency of the *ALDH2* rs671 G/G genotype was lower (48.3% vs 66.1%, $p<0.001$), whereas that of the *ALDH2* rs671 G/A genotype was higher (43.5% vs 30.2%, $p<0.001$) in the patients with coronary atherosclerosis than in controls. The frequency of the *ALDH2* rs671 G allele was lower (70.0% vs 81.2%) and that of *ALDH2* rs671 A allele was higher (30.0% vs 18.8%) in the patients with coronary atherosclerosis than in controls ($p<0.001$) (Table 2).

Table 1 Clinical Characteristics of the Patients with Hypertension of This Study

Variables	Total (n=923)	Controls (n=484)	Patients Coronary Atherosclerosis (n=439)	*p* values
Gender				
Male, n(%)	660(71.5%)	349(72.1%)	311(70.8%)	0.715
Female, n(%)	263(28.5%)	135(27.9%)	128(29.2%)	
BMI (kg/m^2)				
<18.5	51(5.5%)	30(6.2%)	21(4.8%)	0.049
18.5–23.9	452(49.0%)	252(52.1%)	200(45.6%)	
≥24.0	420(45.5%)	202(41.7%)	218(49.7%)	
History of smoking, n(%)	229(24.8%)	127(26.2%)	102(23.2%)	0.321
History of alcoholism, n(%)	132(14.3%)	109(22.5%)	23(5.2%)	<0.001
Diabetes mellitus, n(%)	339(36.7%)	158(32.6%)	181(41.2%)	0.007
Serum lipid levels				
TC, mmol/L	4.52±1.46	4.38±1.68	4.65±1.22	0.013
TG, mmol/L	1.72±1.23	1.64±1.22	1.79±1.23	0.089
HDL-C, mmol/L	1.15±0.45	1.11±0.50	1.18±0.39	0.051
LDL-C, mmol/L	2.53±1.05	2.41±1.19	2.64±0.89	0.004
Inflammatory indices levels				
PIV, median (P25, P75)	437.76(192.57, 955.08)	349.92(143.46, 834.34)	522.02(240.15, 1102.71)	<0.001
SII, median (P25, P75)	822.50(406.69, 1588.50)	659.93(323.50, 1398.65)	972.00(551.38, 1767.33)	<0.001
SIRI, median (P25, P75)	2.13(1.11, 4.41)	1.94(0.98, 3.82)	2.48(1.17, 5.09)	0.001

Notes: Values for age expressed as mean±SD.
Abbreviations: BMI, body mass index; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; PIV, pan-immune-inflammation-value; SII, systemic immune-inflammatory index; SIRI, systemic inflammatory response index; P25, 25th percentile; P75, 75th percentile.

Table 2 Distribution Frequencies of *ALDH2* rs671 Genotype and Allele in Patients and Controls

Variables	Genotype/ allele	Total (n=923)	Controls (n=484)	Patients Coronary Atherosclerosis (n=439)	χ^2	*p* values
ALDH2 rs671 genotypes						
	G/G	532(57.6%)	320(66.1%)	212(48.3%)	29.953	<0.001
	G/A	337(36.5%)	146(30.2%)	191(43.5%)	17.680	<0.001
	A/A	54(5.9%)	18(3.7%)	36(8.2%)	8.393	0.005
ALDH2 rs671 alleles						
	G	1401(75.9%)	786(81.2%)	615(70.0%)	31.302	<0.001
	A	445(24.1%)	182(18.8%)	263(30.0%)		
	HWE (χ^2, *P*)	χ^2=0.004, *p*=0.948	χ^2=0.070, *p*=0.791	χ^2=0.595, *p*=0.441		

Abbreviation: ALDH2, aldehyde dehydrogenase 2; HWE, Hardy Weinberg Equilibrium.

Clinical Characteristics of Subjects Stratified by ALDH2 rs671 Genotypes

There were significant differences in gender distribution (p=0.012), the proportions of history of smoking (p=0.001), history of alcohol consumption (p<0.001), and HDL-C level (p<0.001) among patients with different *ALDH2* rs671 genotypes. There were no significant differences in other serum lipid indices and inflammatory indices levels among patients with different *ALDH2* rs671 genotypes (all p>0.05) (Table 3).

Logistic Regression Analysis of Risk Factors of Coronary Atherosclerosis in Patients with Hypertension

Univariate analysis showed that BMI ≥24.0 kg/m^2 (BMI ≥24.0 kg/m^2 vs BMI 18.5–23.9 kg/m^2, odds ratio (OR): 1.360, 95% confidence interval (CI): 1.042–1.775, p=0.024), diabetes mellitus (yes vs no, OR: 1.452, 95% CI: 1.109–1.901,

Table 3 Clinical Characteristics of Subjects Stratified by *ALDH2* rs671 Genotypes

Variables	G/G (n=532)	G/A (n=337)	A/A (n=54)	*p* values
Gender				
Male, n(%)	400(75.2%)	222(65.9%)	38(70.4%)	0.012
Female, n(%)	132(24.8%)	115(34.1%)	16(29.6%)	
BMI (kg/m^2)				
<18.5	30(5.6%)	21(6.2%)	0(0)	0.264
18.5–23.9	252(47.4%)	172(51.0%)	28(51.9%)	
≥24.0	250(47.0%)	144(42.7%)	26(48.1%)	
History of smoking, n(%)	155(29.1%)	67(19.9%)	7(13.0%)	0.001
History of alcoholism, n(%)	115(21.6%)	17(5.0%)	0(0)	<0.001
Diabetes mellitus, n(%)	200(37.6%)	118(35.0%)	21(38.9%)	0.662
Serum lipid levels				
TC, mmol/L	4.45±1.44	4.58±1.47	4.82±1.59	0.186
TG, mmol/L	1.81±1.34	1.60±1.01	1.62±1.28	0.058
HDL-C, mmol/L	1.09±0.46	1.20±0.42	1.30±0.41	<0.001
LDL-C, mmol/L	2.48±1.02	2.59±1.05	2.68±1.28	0.225
Inflammatory indices levels				
PIV, median (P25, P75)	413.76(183.87, 950.24)	467.94(204.06, 982.47)	458.55(206.78, 1065.67)	0.414
SII, median (P25, P75)	769.86(370.20, 1543.17)	847.51(435.31, 1653.65)	881.83(538.48, 1653.66)	0.250
SIRI, median (P25, P75)	2.10(1.13, 4.25)	2.24(1.01, 4.88)	2.43(1.14, 4.91)	0.770

Notes: Values for age expressed as mean±SD.

Abbreviations: BMI, body mass index; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein-cholesterol; LDL-C, low-density lipoprotein-cholesterol; PIV, pan-immune-inflammation-value; SII, systemic immune-inflammatory index; SIRI, systemic inflammatory response index; P25, 25th percentile; P75, 75th percentile.

p=0.0071), *ALDH2* rs671 G/A genotype (G/A vs G/G, OR: 1.975, 95% CI: 1.497–2.604, *p*<0.001) and A/A genotype (A/A vs G/G, OR: 3.019, 95% CI: 1.670–5.456, *p*<0.001), and high TC, LDL-C, PIV, SII, and SIRI were significantly associated with coronary atherosclerosis. Multivariate logistic regression analysis showed that BMI ≥24.0 kg/m^2 (BMI ≥24.0 kg/m^2 vs BMI 18.5–23.9 kg/m^2, OR: 1.670, 95% CI: 1.185–2.352, *p*=0.003), history of smoking (yes vs no, OR: 2.024, 95% CI: 1.263–3.243, *p*=0.003), *ALDH2* rs671 G/A genotype (G/A vs G/G, OR: 1.821, 95% CI: 1.280–2.589, *p*=0.001), high TC level (OR: 1.592, 95% CI: 1.021–2.485, *p*=0.040), high SII level (OR: 2.290, 95% CI: 1.386–3.784, *p*=0.001), and high SIRI level (OR: 1.727, 95% CI: 1.126–2.650, *p*=0.012) were associated with coronary atherosclerosis (Table 4).

Table 4 Logistic Regression Analysis of Risk Factors for Coronary Atherosclerosis in Patients with Hypertension

Variables	Univariate OR (95% CI)	*p* values	Multivariate OR (95% CI)	*p* values
Gender (Male/Female)	0.940(0.706–1.251)	0.671	0.967(0.659–1.418)	0.863
BMI (kg/m^2)				
18.5–23.9	1.000(reference)	–	1.000(reference)	–
<18.5	0.882(0.490–1.588)	0.675	0.877(0.434–1.773)	0.715
≥24.0	1.360(1.042–1.775)	0.024	1.670(1.185–2.352)	0.003
History of smoking (Yes/No)	0.854(0.632–1.154)	0.304	2.024(1.263–3.243)	0.003
History of alcoholism (Yes/No)	0.191(0.119–0.307)	<0.001	0.145(0.073–0.287)	<0.001
Diabetes mellitus (Yes/No)	1.452(1.109–1.901)	0.007	1.375(0.979–1.932)	0.066
ALDH2 rs671 genotypes				
G/G	1.000(reference)	–	1.000(reference)	–
G/A	1.975(1.497–2.604)	<0.001	1.821(1.280–2.589)	0.001
A/A	3.019(1.670–5.456)	<0.001	1.624(0.830–3.175)	0.157

(*Continued*)

Table 4 (Continued).

Variables	Univariate OR (95% CI)	*p* values	Multivariate OR (95% CI)	*p* values
TC	1.844(1.376–2.469)	<0.001	1.592(1.021–2.485)	0.040
LDL-C	1.942(1.438–2.624)	<0.001	1.351(0.867–2.107)	0.184
PIV	1.840(1.402–2.415)	<0.001	0.701(0.420–1.168)	0.173
SII	2.661(1.970–3.594)	<0.001	2.290(1.386–3.784)	0.001
SIRI	1.774(1.334–2.358)	<0.001	1.727(1.126–2.650)	0.012

Abbreviations: BMI, body mass index; ALDH2, aldehyde dehydrogenase 2; TC, total cholesterol; LDL-C, low-density lipoprotein-cholesterol; PIV, pan-immune-inflammation-value; SII, systemic immune-inflammatory index; SIRI, systemic inflammatory response index; OR, odds ratio; CI, confidence interval.

Discussion

As one of the most common chronic diseases, hypertension can cause vascular endothelial damage owing to a continuous increase in blood pressure, resulting in atherosclerosis, which leads to CVDs.[38] 4-HNE produced during ischemia and hypoxia leads to hypertension by damaging vascular endothelial cells and inducing oxidative stress.[39] Notably, ALDH2 inhibits the oxidative stress process by metabolizing 4-HNE, thereby inhibiting the occurrence of hypertension.[40] Studies have shown that 4HNE induced migration of coronary endothelial cells is regulated by ALDH2 enzyme response,[41] and ALDH2 is involved in the formation of foam cells by regulating CD36 expression via 4-HNE/peroxisome proliferator-activated receptor gamma (PPARγ) pathway.[42] The development of atherosclerosis is a multifactorial process. The risk factors for coronary atherosclerosis in hypertensive patients are unknown and it is important to identify the risk of coronary atherosclerosis in patients with hypertension.

The most important polymorphic site known to be related to the enzymatic activity of ALDH2 is the SNP rs671. Several studies have shown an association between rs671 polymorphism in *ALDH2* gene and coronary atherosclerosis.[30,32,43] A study by Zhao et al showed regional differences in the association between the *ALDH2* rs671 genotype and coronary atherosclerosis risk.[44] Morita et al found that smoking and *ALDH2* rs671 A allele jointly increase the risk of coronary atherosclerosis, suggesting that *ALDH2* polymorphism synergistically affect the course of coronary atherosclerosis with smoking.[45] And *ALDH2* rs671 polymorphism was associated with hypertension.[46,47] *ALDH2* rs671 G/A genotype increases the risk of coronary atherosclerosis, and the results of this study enrich the data on the relationship between ALDH2 and CVDs.

Atherosclerosis is a chronic inflammatory disease caused by lipid deposition in the blood vessels.[48,49] The relationships between serum lipids (TC, TG, HDL-C, LDL-C), peripheral inflammation indices (PIV, SII, SIRI) and coronary atherosclerosis were analyzed, and the results showed that high TC, SII, and SIRI levels were associated with coronary atherosclerosis. The SII score was correlated the severity of coronary atherosclerosis.[50] SII is an indicator to predict the mortality of patients with myocardial infarction.[51] Several studies found that SIRI had correlation with the severity of coronary artery stenosis (CAS),[52] and coronary atherosclerosis.[53] In addition, a study by Demirtola A et al showed a correlation between PIV and the severity of coronary atherosclerosis.[54] PIV can be used as an indicator to predict the prognosis of patients with myocardial infarction.[18] No correlation was observed between PIV and coronary atherosclerosis in this study. In addition, a genome-wide association study (GWAS) showed an association between *ALDH2* rs671 and lower HDL-C level,[55] and the relationship was also confirmed in two clinical studies.[56,57] However, no similar results were observed in this study. It may be due to differences in sample size among these studies.

This study found that individuals with a normal BMI range had a significantly reduced risk of coronary atherosclerosis.[58] Several studies have shown that overweight individuals have a significantly increased risk of CVDs.[59–61] Chen et al found that high BMI was a risk factor for coronary atherosclerosis in patients with type 2 diabetes mellitus.[62] Moreover, BMI is associated with the prognosis of coronary atherosclerosis.[63,64] However, other studies have found no association between the risk of major adverse cardiovascular events and BMI.[65,66] The inconsistencies in these studies may be because BMI varies according to gender, age, and race.[67] Therefore, the relationship between BMI and susceptibility to coronary atherosclerosis in hypertensive patients requires further investigation.

This study reported that *ALDH2* polymorphism was associated with coronary atherosclerosis susceptibility in patients with hypertension. It provides valuable information for the identification of individuals at risk for coronary atherosclerosis among patients with hypertension. However, this study has some limitations. First, the subjects in this study were all from a single medical institution, and the findings were only applicable to hypertensive patients in the local population. Second, owing to the small number of cases with *ALDH2* rs671 A/A genotype, this study did not find a relationship between *ALDH2* rs671 A/A genotype and coronary atherosclerosis, which needs to be further analyzed after expanding the sample size. Third, this study did not analyze the risk factors for the severity of coronary atherosclerosis in hypertensive patients.

Conclusion

In summary, being overweight (BMI ≥24.0 kg/m^2), history of smoking, *ALDH2* rs671 G/A genotype, and high TC, SII, and SIRI levels were independently associated with coronary atherosclerosis in patients with hypertension. This suggests that hypertensive patients who are overweight, have a history of smoking, carried *ALDH2* rs671 G/A genotype, and have high TC, SII, and SIRI levels should be aware of the risk of coronary atherosclerosis.

Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Approval

All participants were informed on the study procedures and goals and the informed consent from all the participants. The study was performed under the guidance of the Declaration of Helsinki and approved by the Ethics Committee of Medicine, Meizhou People's Hospital.

Acknowledgments

The authors thank their colleagues, who were not listed in the authorship of the Department of Blood Transfusion, Meizhou People's Hospital, for their helpful comments on the manuscript.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by the Scientific Research Cultivation Project of Meizhou People's Hospital (Grant No.: PY-C2021054).

Disclosure

The authors declare that they have no competing interests in this work.

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