MEDICAL SCIENCE MONITOR

CLINICAL RESEARCH

e-ISSN 1643-3750
© Med Sci Monit, 2022; 28: e936425
DOI: 10.12659/MSM.936425

Received: 2022.02.17
Accepted: 2022.03.16
Available online: 2022.03.29
Published: 2022.05.14

Case-Control Study to Investigate the Association Between Serum Apolipoprotein B/A1 Ratio and Atrial Fibrillation by Sex in 920 Patients from China

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Statistical Analysis C
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Financial support: This study was supported by study on the expression of pacing gene-regulated by autonomic nerves in atrial fibrillation syndrome with phlegm fire and heart disturbance based on nonlinear theory, the National Natural Science Foundation of China (81603609)
Conflict of interest: None declared

Background: The serum apolipoprotein B/A1 ratio (APOB/APOA1) has been shown to predict cardiovascular events, whereas the effect of the APOB/APOA1 ratio on atrial fibrillation (AF) is less known. We investigated the association between the APOB/APOA1 ratio and AF by sex in 920 patients from China.

Material/Methods: We reviewed clinical data on 1840 hospitalized patients, including 920 patients with AF (male/female: 460/460, age: 68.62±10.36 years) and 920 age- and sex-matched patients without AF with sinus rhythm in China between January 2019 and September 2021. Pearson correlation analysis was performed to investigate the correlation between APOB/APOA1 ratio and AF-related metabolic factors. Logistic regression analysis was used to determine the odds ratios (ORs) and 95% confidence intervals (CIs).

Results: Low serum APOB/APOA1 ratios in male and female patients were significantly associated with AF after adjusting for confounding factors (OR 0.159, 95% CI 0.058-0.432, $P<0.05$). Serum APOB/APOA1 ratio was positively correlated with triglyceride (TG) (r=0.146, $P<0.05$) and total cholesterol (TC) (r=0.227, $P<0.05$) and was negatively correlated with albumin (ALB) (r=-0.128, $P<0.05$) and prealbumin (PAB) (r=-0.107, $P<0.05$). There was no significant difference of APOB/APOA1 ratio in different subtypes, complications, and statin use in patients with AF ($P>0.05$).

Conclusions: A low serum APOB/APOA1 ratio in male and female patients from China was significantly related to AF. This finding implies that a low serum APOB/APOA1 ratio may be associated with the causes of AF. Further studies are needed to determine causalities.

Keywords: Apolipoprotein A-I • Apolipoprotein B (3304-3317) • Atrial Fibrillation • Gender Identity • Inflammation

Full-text PDF: https://www.medscimonit.com/abstract/index/idArt/936425

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Background

Atrial fibrillation (AF) is the most common and challenging clinically significant sustained cardiac arrhythmia, affecting almost 33 million people worldwide [1-3]. As a growing health threat, AF can contribute to an increased risk of heart failure, stroke, cognitive impairment, systemic embolism, and even death [4-8], with associated hospitalization rates, mortality, and a considerable healthcare burden [9]. Currently, antiarrhythmic drugs and catheter ablation are clinically recommended to control heart rate and rhythm in patients [10]. Although antiarrhythmic drugs and catheter ablation are recommended as effective treatments, the success rate of a single surgery has been reported to be only 60% to 70%, and the potential complications of the surgery cannot be ignored [11-14]. Risk factor management as an upstream noninvasive treatment has a potential beneficial effect on AF [15]. Exploring available blood biomarkers related to AF may contribute to understanding its underlying pathological mechanism and implementing corresponding prevention strategies.

Guidelines have been recommended for treating cardiovascular disease through the management of blood lipid profiles [16]. The nontraditional lipid biomarkers apolipoprotein A1 (APOA1) and apolipoprotein B (APOB) are the key protein moieties correlated with HDL cholesterol (HDL-C) and LDL cholesterol (LDL-C), respectively; they play a significant role in lipid metabolism and are both considered to be associated with the risk of cardiovascular disease [17,18]. Generally, APOA1 is atheroprotective and APOB is anti-atheroprotective [19,20]. In addition, several studies have reported that the ApoB/ApoA1 ratio is a convenient and accurate specific marker for cardiovascular risk events [21], having an even better predictive advantage than lipid adjuvant in coronary heart disease and ischemic events [22-24]. Some researchers have recently confirmed that APOA1 and APOB are closely related to inflammation and oxidative stress, which are important reaction chains for AF initiation and complexity [25-27]. Moreover, the effects of dyslipidemia on AF remain controversial.

In this study, we aimed to explore the association between the serum APOB/APOA1 ratio and AF by sex in 920 patients from China.

Material and Methods

Study Design

This study followed the principles of the Helsinki Declaration and passed the review of the Medical Research Ethics Committee of the Affiliated Hospital of Shandong University of Traditional Chinese Medicine. No informed consent was required because the data were anonymized. This study used a matched case-control design of 1840 hospitalized patients' (male/female: 917/923, age: 68.35±10.92 years) electronic records from the Affiliated Hospital of Shandong University of Traditional Chinese Medicine. Inclusion criteria were as follows: Cases comprised patients aged 29 to 85 years diagnosed with AF between January 2019 and September 2021. Each patient with complete clinical data had age- and sex-matched controls. This study eventually involved 920 patients with AF and 920 controls. Meanwhile, we excluded patients with a history of cardiac surgery, heart failure, valvular disease, or malignancy, as well as patients with current liver or kidney dysfunction, hyperthyroidism, and infection and those taking diuretics. Patients with AF were diagnosed in a professional manner by their physicians, excluding self-described patients. We systematically screened clinical variables for all participants from the electronic medical record system, including baseline clinical data, laboratory data, subtypes of AF, and complications of AF. In addition, the data of patients with AF were stratified by sex and serum APOB/APOA1 ratio.

Screened Variables

The baseline data of patients were selected and included sex, age, types and complications of AF, use of statins, CCBs, β-blockers, and ACEI/ARB, as well as laboratory indicators, including serum APOB levels (0.75-1.55g/L), serum APOA1 levels (1.10-1.19 g/L), serum APOB/APOA1 ratio, blood lipid profiles (TC [3.0-5.7 mmol/L], TG [0.4-1.7 mmol/L], LDL-C [<3.63 mmol/L], HDL-C [>1.04 mmol/L]), alanine aminotransferase (ALT; 7-40 U/L), aspartate aminotransferase (AST; 13-35 U/L), lipoprotein (a) (Lp[a]; 0-30 mg/dL), serum uric acid (SUA; 2.6-6.0 mg/dL), serum creatinine (SCr; 41-81 μmol/L), serum albumin (ALB; 40-55 U/L), and prealbumin (PAB; 18-35 g/L). All laboratory measurements were taken in strict accordance with hospital standards. The turbidimetric inhibition immunoassay was used to determine APOB and APOA1. Serum APOB/APOA1 ratios were divided into 3 tertiles by sex (male: ≤0.61, 0.61-0.83, ≥0.83; female: ≤0.56, 0.56-0.77, ≥0.77).

Statistical Analysis

Data analysis was conducted using SPSS software (version 26.0; IBM Corp, Armonk, NY, USA) or GraphPad Prism software (version 9.0.0). Continuous data were presented as mean±standard deviation and compared using the *t* test and analysis of variance. Categorical data were expressed as n(%) and compared by the chi-square test. Pearson correlation analysis was presented as a scatter plot to evaluate correlations. Multivariate regression analysis was performed using odds ratios (ORs) and 95% confidence intervals (95% CI) to adjust for covariates. A 2-tailed $P<0.05$ was considered statistically significant.

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Table 1. Clinical characteristics.

Variable	AF group (n=920)	Control group (n=920)	P value
Age, years	68.62±10.36	68.08±11.46	0.289
Male, n (%)	460 (50.00)	457 (49.67)	0.889
Hypertension, n (%)	618 (67.17)	308 (33.48)	<0.05*
CHD, n (%)	812 (88.26)	227 (24.67)	<0.05*
Diabetes, n (%)	274 (29.787)	153 (16.63)	<0.05*
APOA1, g/L	1.13±0.26	1.22±0.25	<0.05*
APOB, g/L	0.79±0.38	0.99±0.24	<0.05*
APOB/APOA1 ratio	0.71±0.41	0.84±0.25	<0.05*
Men	0.76±0.51	0.88±0.25	<0.05*
Women	0.71±0.29	0.80±0.25	<0.05*
Lp (a), mg/L	23.67±27.08	22.54±24.49	0.348
TC, mmol/L	4.19±1.09	5.03±1.10	<0.05*
TG, mmol/L	1.24±0.88	1.38±1.25	<0.05*
LDL-C, mmol/L	1.08±0.30	1.20±0.31	<0.05*
HDL-C, mmol/L	2.50±0.89	2.97±0.86	<0.05*
AST, U/L	25.42±46.31	20.81±10.93	<0.05*
ALT, U/L	22.38±27.74	20.40±13.86	0.053
ALB, g/L	38.02±4.64	40.10±4.13	<0.05*
PAB, g/L	19.55±5.92	22.29±5.50	<0.05*
SUA, mg/dL	5.89±1.76	5.12±1.37	<0.05*
SCr, μmol/L	78.55±52.13	65.04±27.00	<0.05*
Statins, n (%)	605 (65.76)	211 (22.93)	<0.05*
CCBs, n (%)	331 (35.98)	161 (17.50)	<0.05*
β-blockers, n (%)	721 (78.37)	153 (16.63)	<0.05*
ACEI/ARB, n (%)	513 (55.16)	135 (14.67)	<0.05*

Data are presented as mean±SD or n(%). AF – atrial fibrillation; CHD – coronary heart disease; APOA1 – apolipoprotein A1; APOB – apolipoprotein B; Lp (a) – lipoprotein (a); TC – total cholesterol; TG – triglyceride; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; AST – aspartate aminotransferase; ALT – alanine aminotransferase; ALB – albumin; PAB – prealbumin; SCr – serum creatinine; SUA – serum uric acid. * Statistically significant value (P<0.05).

Results

Clinical Characteristics

As shown in **Table 1**, 920 patients with AF (male/female: 460/460, 68.62±10.36 years) and 920 age- and sex-matched patients without AF with sinus rhythm (male/female: 450/463, 68.08±11.46 years) were included. Patients with AF were more likely than controls to have hypertension, coronary heart disease, and diabetes (P<0.05) and were more likely to use statins, CCBs, β-blockers, and ACEI/ARB (P<0.05). Patients with AF also had significantly lower serum levels of APOAl, APOB, APOB/APOA1 ratio, TC, TG, LDL-C, HDL-C, ALB, and PAB (P<0.05) than controls and had significantly higher serum levels of AST, SUA, and SCr (P<0.05) than controls.

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Table 2. Correlation between serum APOB/APOA1 ratio and atrial fibrillation.

	Total		Men		Women	
	OR 95% CI	P value	OR 95% CI	P value	OR 95% CI	P value
Model 1	0.276 (0.193-0.395)	<0.05*	0.233 (0.140-0.389)	<0.05*	0.313 (0.188-0.521)	<0.05*
Model 2	0.312 (0.192-0.508)	<0.05*	0.291 (0.138-0.614)	<0.05*	0.279 (0.143-0.545)	<0.05*
Model 3	0.055 (0.024-0.126)	<0.05*	0.165 (0.052-0.524)	<0.05*	0.040 (0.011-0.146)	<0.05*
Model 4	0.159 (0.058-0.432)	<0.05*	0.602 (0.363-0.998)	<0.05*	0.033 (0.008-0.139)	<0.05*

Model 1: crude, no adjustment. **Model 2:** adjusted for hypertension, CHD, diabetes, β-blockers, CCB, ACEI/ARB, and statins. **Model 3:** adjusted for TG, TC, LDL-C, HDL-C, AST, SUA, Scr, PAB, and ALB. **Model 4:** adjusted for all these factors. CHD – coronary heart disease; APOA1 – apolipoprotein A1; APOB – apolipoprotein B; Lp (a) – lipoprotein (a); TC – total cholesterol; TG – triglyceride; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; AST – aspartate aminotransferase; ALT – alanine aminotransferase; ALB – albumin; PAB – prealbumin; SCr – serum creatinine; SUA – serum uric acid. * Statistically significant value (P<0.05).

Correlation Between Serum APOB/APOA1 Ratio and AF

As shown in **Table 2**, after adjusting for hypertension, CHD, diabetes, β-blockers, CCB, ACEI/ARB, and statins, the serum APOB/APOA1 ratio was related to AF (OR 0.312, 95% CI 0.192-0.508, P<0.05). After adjusting for TG, TC, LDL-C, HDL-C, AST, SUA, Scr, PAB, and ALB, the serum APOB/APOA1 ratio was still associated with AF (OR 0.055, 95% CI 0.024-0.126, P<0.05). Further, after adjusting for all confounding factors, the serum APOB/APOA1 ratio remained independently negatively correlated with AF (OR 0.159, 95% CI 0.058-0.432, P<0.05). Moreover, the serum APOB/APOA1 ratio was negatively correlated with AF in men and women (P<0.05).

Differences of Serum APOB/APOA1 Ratio in Male and Female Patients with AF

As shown in **Table 3**, there was no significant difference in serum APOB/APOA1 ratio in men and women with AF, regardless of AF subtypes, complications, and whether or not statins were used (P>0.05).

Differences of Serum APOB/APOA1 Ratio in Patients with AF by Different Subtypes, Complications, and Statin Use

As shown in **Table 4**, the results showed there was no significant difference in serum APOB/APOA1 ratio in patients with AF by different subtypes, complications, and statin use (P>0.05).

Correlation Between Serum APOB/APOA1 Ratio and AF-Related Metabolic Factors

Figure 1 shows the serum APOB/APOA1 ratio and AF-related metabolic factors. Specifically, serum APOB/APOA1 ratio was positively correlated with TG (r=0.146, P<0.05; **Figure 1A**), TC (r=0.227, P<0.05; **Figure 1B**), ALB (r=-0.128, P<0.05; **Figure 1C**), and PAB (r=-0.107, P<0.05; **Figure 1D**).

Subgroup Analysis of the Relationship Between APOB/APOA1 Ratio and Metabolic Factors in Patients with AF

Table 5 shows the subgroup analysis of the relationship between APOB/APOA1 ratio and metabolic factors in patients with AF. The results showed that male and female patients with lower serum APOB/APOA1 ratios also had lower TG, TC, HDL-C, and APOB had higher LDL-C, APOA1, and PAB (P<0.05). In addition, men with a lower serum APOB/APOA1 ratio had higher ALB (P<0.05), and women with a lower APOB/APOA1 ratio had lower SUA (P<0.05).

Discussion

We used a retrospective matched case-control study design to investigate the relationship between the serum APOB/APOA1 ratio and AF by sex in the Chinese population. The present results showed that a low serum APOB/APOA1 ratio in men and women was significantly related to AF. Further results indicated that serum APOB/APOA1 ratio was positively correlated with TG, TC, and APOB and was negatively correlated with ALB and PAB. These significant findings imply that a low serum APOB/APOA1 ratio may be associated with causes of AF.

Many studies have indicated that the serum APOB/APOA1 ratio is a significant marker of risk for cardiovascular events [28], but the relationship between the serum APOB/APOA1 ratio and the risk of AF has not been consistently shown. In this study, the serum ApoB/ApoA1 ratio of patients with AF was decreased due to the decrease in APOB production. Studies have shown that there are low serum levels of APOB in patients with AF.

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Table 3. Differences of serum APOB/APOA1 ratio in male and female patients with atrial fibrillation.

Variable	n	APOB/APOA1 ratio	P value
Paroxysmal AF			
Men	174	0.83±0.74	0.099
Women	146	0.73±0.27	
Permanent AF			
Men	186	0.73±0.27	0.455
Women	314	0.71±0.30	
AF+hypertension			
Men	293	0.77±0.62	0.208
Women	345	0.72±0.30	
AF+CHD			
Men	393	0.77±0.54	0.107
Women	419	0.72±0.30	
AF+diabetes			
Men	138	0.84±0.83	0.159
Women	136	0.73±0.38	
AF patients using statins			
Men	304	0.76±0.59	0.110
Women	301	0.70±0.28	
AF patients non-using statins			
Men	156	0.76±0.28	0.368
Women	159	0.73±0.31	

Data are presented as mean±SD. AF – atrial fibrillation; APOA1 – apolipoprotein A1; APOB – apolipoprotein B; CHD – coronary heart disease.

A cohort study showed that low serum APOB was a dominant factor in the occurrence of AF in men and women [29]. Nevertheless, it has been reported that the APOB/APOA1 ratio is significantly elevated in people with inflammation and atherosclerosis [30-33], which is inconsistent with our findings. According to recent studies, there are several possible reasons for this difference. First, sleep quality in the study population was a significant factor. Ren et al reported that a long sleep duration was associated with a low APOB/APOA1 ratio [34]. Second, the patients used certain drugs. Hamedi-Kalajahi et al reported that oral L-carnitine supplementation caused a decrease in the APOB/APOA1 ratio [35]. Additionally,

Table 4. Differences in serum APOB/APOA1 ratio by different subtypes, complications, and statins use in patients with atrial fibrillation.

Variable	n	APOB/APOA1 ratio	P value
Subtypes of AF			
Paroxysmal AF	320	0.78±0.58	0.082
Permanent AF	600	0.72±0.28	
Complications of AF			
AF+hypertension	618	0.74±0.47	0.296
AF+CHD	812	0.74±0.43	
AF+diabetes	274	0.79±0.65	
Whether or not statins were used			
Yes	605	0.73±0.46	0.421
No	315	0.75±0.29	

Data are presented as mean±SD. AF – atrial fibrillation; APOA1 – apolipoprotein A1; APOB – apolipoprotein B; CHD – coronary heart disease.

dietary habits, physical exercise, and family history were also possible factors.

Our main finding in this study was that a low serum APOB/APOA1 ratio in men and women was associated with AF in a Chinese population. Specifically, there were several potential mechanisms that could explain this finding. Increasing recognition that inflammation and oxidative stress contribute significantly to AF has elucidated their mechanistic links [36-38]. Inflammation, in particular, is thought to be closely associated with oxidative stress, apoptosis, and fibrosis that promote the formation of AF substrates [39], thereby increasing the vulnerability to AF. There is also evidence to support the relationship between blood lipid profiles and AF, based on the mechanisms of inflammation and oxidative stress [40,41]. Serum APOA1, a key protein element of HDL, is a main initiator and promoter of cholesterol reverse transport [42,43]. It has strong anti-inflammatory and anti-oxidant properties and anti-atherogenic effects, which can promote the production of nitric oxide and the release of prostacyclin [44,45]. Research has shown that serum APOA1 exerts anti-inflammatory effects mainly by inhibiting cytokine production by monocytes/macrophages [46-48]. Previous studies also reported that serum APOA1 was reduced in acute inflammation and may induce the production of TNF-α and IL-1β [46,48]. Serum APOB, the LDL-matching protein [49], has also been shown to be associated with inflammation. Faraj et al reported that serum APOB is a strongly

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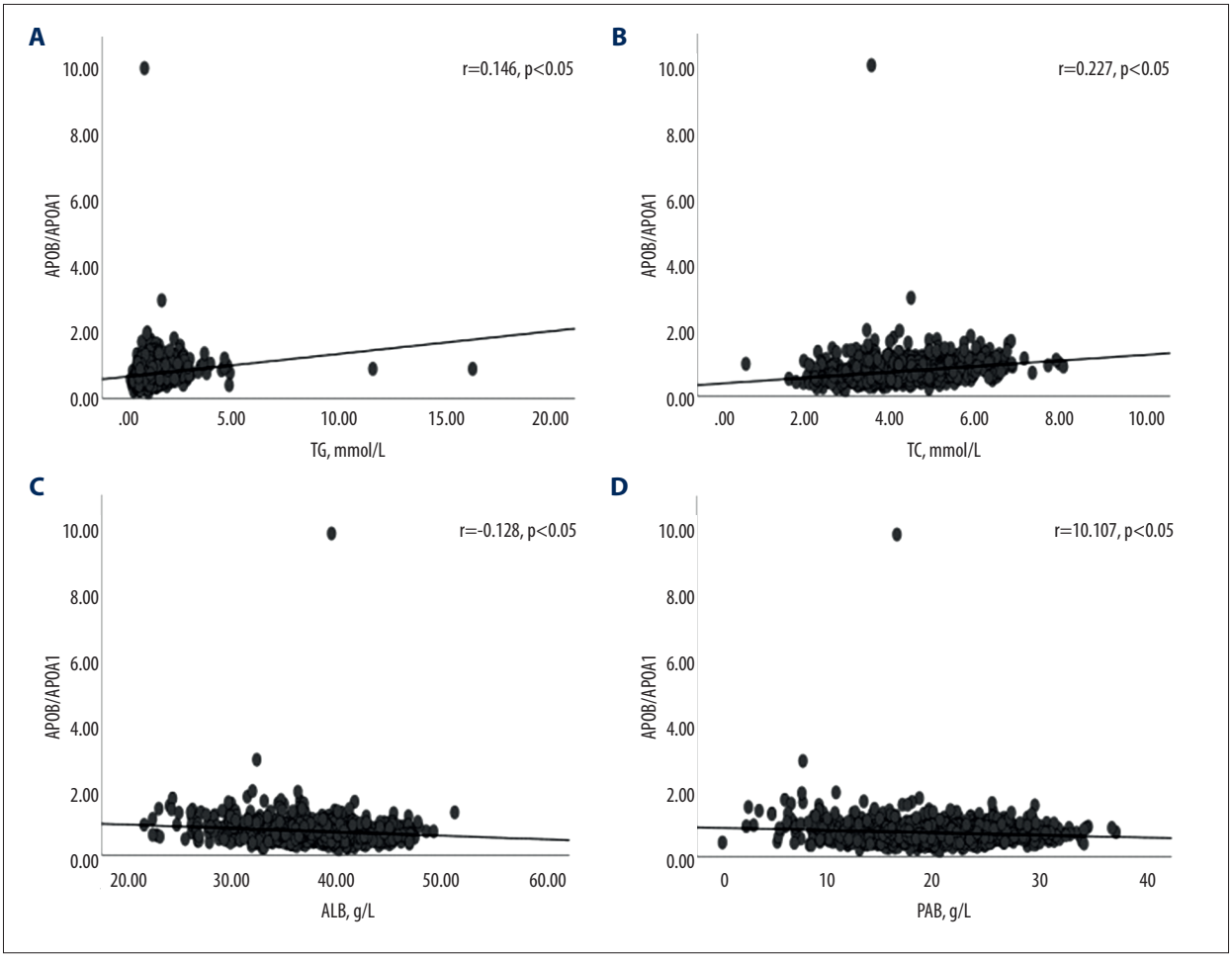

Figure 1. The scatter plots showed the correlation between serum APOB/APOA1 ratio and metabolic factors in patients with atrial fibrillation (AF). (**A**) Correlation between serum APOB/APOA1 and TG in patients with AF (r=0.146, *P*<0.05). (**B**) Correlation between serum APOB/APOA1 and TC in patients with AF (r=0.227, *P*<0.05). (**C**) Correlation between serum APOB/APOA1 and ALB in patients with AF (r=-0.128, *P*<0.05). (**D**) Correlation between serum APOB/APOA1 and PAB in patients with AF (r=-0.107, *P*<0.05). These figures were drawn by SPSS software (version 26.0, SPSS Inc., Chicago, IL, USA). TG – triglyceride; TC – total cholesterol; ALB – albumin; PAB – prealbumin.

correlated predictor of inflammatory markers, such as interleukin-6 and CRP, and results from further studies suggested that reduced serum APOB may contribute to inflammation [26,50]. Additionally, the relationship between atherosclerosis and inflammation has been increasingly recognized [51,52]. It can be said that atherosclerosis has been included in the category of chronic inflammatory diseases [53]. The serum APOB/APOA1 ratio has been shown to be an effective indicator to evaluate the balance between pro-atherogenic LDL particles and anti-atherogenic HDL particles [54]. Generally speaking, the higher the serum APOB/APOA1 ratio is, the more likely it is that atherosclerosis will occur [55]. However, our results suggested that a lower APOB/APOA1 ratio was more likely to contribute to inflammation, thereby increasing the likelihood of AF. We hypothesized that the reduction of HDL-C accompanied by the loss of anti-inflammatory, anti-oxidant, and anti-atherosclerosis effects was a possible factor in the increase of AF matrix formation [56-58]. Certainly, other potential confounding factors, such as lifestyle, regional environment, and aging, may also influence the results. Further studies should be conducted to explore these potential correlations and mechanisms.

In addition, the relationship between the serum APOB/APOA1 ratio and metabolic syndrome by sex has been confirmed. A study showed that there was a positive linear correlation between the number of metabolic syndrome components and the APOB/APOA1 ratio in men [59]. Another study reported that the serum APOB/APOA1 ratio was also correlated with metabolic syndrome and its components in women, and further suggested that this relationship was independent of patient sex [60]. Our present results were consistent with those of earlier studies and confirmed the relationship between the

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Table 5. Subgroup analysis of the relationship between APOB/APOA1 ratio and metabolic factors in patients with atrial fibrillation.

Variable	Men (n=460)				Women (n=460)			
	≤0.61	0.61-0.83	≥0.83	P value	≤0.56	0.56-0.77	≥0.77	P value
Number, n	156	153	151		152	158	150	
TG, mmol/L	1.24±0.88	1.20±0.59	1.49±1.09	<0.05*	0.98±0.39	1.33±0.62	1.53±1.42	<0.05**
TC, mmol/L	4.19±1.09	4.15±0.89	4.47±1.13	<0.05*	3.78±0.80	4.46±1.01	4.87±1.19	<0.05*
LDL-C, mmol/L	1.08±0.30	1.02±0.20	0.86±0.19	<0.05*	1.28±0.28	1.16±0.26	0.96±0.32	<0.05*
HDL-C, mmol/L	2.50±0.89	2.56±0.71	2.87±0.79	<0.05*	1.96±0.53	2.68±0.92	3.17±0.88	<0.05*
APOA1, g/L	1.13±0.26	1.10±0.21	0.92±0.21	<0.05*	1.32±0.24	1.24±0.22	1.01±0.25	<0.05*
APOB, g/L	0.79±0.38	0.78±0.16	1.01±0.74	<0.05*	0.60±0.13	0.82±0.16	1.00±0.22	<0.05*
AST, U/L	25.42±46.31	24.78±19.94	26.61±31.39	0.895	24.01±20.94	23.87±18.35	21.89±13.62	0.516
PAB, g/L	19.55±5.92	21.38±5.62	18.83±7.45	<0.05*	19.39±5.00	20.19±5.39	17.90±5.82	<0.05*
ALB, g/L	38.02±4.64	38.80±4.76	36.73±5.65	<0.05*	38.39±4.21	38.64±4.26	37.19±4.90	<0.05*
SCr, μmol/L	78.55±52.13	84.33±26.66	81.58±22.24	0.377	76.41±97.30	71.42±49.42	75.37±47.52	0.795
SUA,mg/dL	5.89±1.76	6.14±1.69	6.32±2.09	0.957	5.48±1.42	5.71±1.44	5.94±1.83	<0.05*

Data are presented as mean±SD. TG – triglyceride; TC – total cholesterol; ALB – albumin; PAB – prealbumin; LDL-C – low-density lipoprotein cholesterol; HDL-C – high-density lipoprotein cholesterol; APOA1 – apolipoprotein A1; APOB – apolipoprotein B; AST – aspartate aminotransferase; PAB – prealbumin; ALB – albumin; SCr – serum creatinine; SUA – serum uric acid. * Statistically significant value ($P<0.05$).

serum APOB/APOA1 ratio and metabolic factors in patients with AF. Specifically, the serum APOB/APOA1 ratio was positively correlated with TG, TC, HDL-C, and APOB and was negatively correlated with LDL-C, APOA1, ALB, and PAB. Also, we further investigated the sex distribution of this association. We found that patients with a lower serum APOB/APOA1 ratio had lower TG, TC, HDL-C, and APOB and higher LDL-C, APOA1, and PAB in both sexes, higher ALB in men, and lower SUA in women. These findings may help explain the relationship between APOB/APOA1 and AF-related metabolic factors, and it is essential to further study its internal mechanism.

This study had some limitations as a retrospective study from a single center, using data from patient medical records, which relied on accurate data input. First, the retrospective case-control study design cannot determine causality. Second, a smaller sample size, single-center design, and higher age distribution may have had a certain impact on our results. Third, this study did not involve clinical data on persistent AF; therefore, our results are not applicable to all patients with AF. Fourth, several potential confounding factors may also have been ignored, such as the markers of inflammation and oxidative stress. Finally, previous researchers have stated that patients should be evaluated for stenosis, prognosis, and ischemic heart disease; we did not evaluate these variables [61,62]. Nevertheless, this study did provide a new perspective on the pathology of AF. Further studies are encouraged to systematically assess the association between the serum APOB/APOA1 ratio and AF, as well as AF-related factors.

Conclusions

In conclusion, our findings show that a low serum APOB/APOA1 ratio in men and women from China was significantly related to AF. This finding suggests that a low serum APOB/APOA1 ratio may be related to the causes of AF. We recommend future prospective cohort investigations to confirm these results.

Acknowledgements

I would like to express my special thanks to my partners and our funding agency for the encouragement and support they gave me during this study.

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