Erciyes Med J 2022; 44(3): 293–98 • DOI: 10.14744/etd.2021.70670
ORIGINAL ARTICLE – OPEN ACCESS

Association Between Carotid Artery Disease and ABO Blood Group

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ABSTRACT

Objective: Carotid artery disease (CAD) is a type of cardiovascular disease typically caused by the formation of atherosclerotic plaques in the carotid arteries. Diabetes mellitus, hereditary features, hyperlipidemia, and hypertension are significant risk factors for CAD. There is strong evidence of a relationship between these major risk factors and the ABO blood groups. The aim of this retrospective study was to examine a potential relationship between the ABO blood groups and CAD.

Materials and Methods: The study group comprised 230 consecutive patients diagnosed with CAD using carotid angiography between January 2012 and November 2019 and 136 consecutive subjects without CAD as controls. The data analyzed were collected from patient files: details of demographic characteristics, lipid profiles (total cholesterol, low-density lipoprotein, high-density lipoprotein, very-low-density lipoprotein, and triglyceride levels), and hematological indices (leukocyte, platelet, hemoglobin, mean platelet volume, neutrophil, lymphocyte, monocyte values).

Results: Chi-squared test analysis indicated that there was a statistically significant difference between the distribution of the blood groups in the patient and control groups (p=0.017). Multiple logistic regression analysis demonstrated that the risk of developing CAD was 1.92 times higher in the non-O blood groups than in the O blood group (p=0.032).

Conclusion: A non-O blood group may be another risk factor for CAD. However, the blood group must be evaluated alongside established risk factors to fully understand the risk of developing CAD.

Keywords: Atherosclerosis, cardiovascular disease, carotid artery disease, dyslipidemia, O blood group

Cite this article as:
Kutlay Ö, Yalım Z, Keskin Aktan A, Koca T. Association Between Carotid Artery Disease and ABO Blood Group. Erciyes Med J 2022; 44(3): 293-98.

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Submitted
10.06.2021

Accepted
20.10.2021

Available Online
15.04.2022

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INTRODUCTION

Atherosclerosis is the accumulation of plaques, or atheroma, in the tunica intima layer of an artery. Although the number, size, distribution, and content of these plaques will vary from person to person, they are frequently found in the abdominal aorta, coronary arteries, and internal carotid arteries (1). The subsequent narrowing of the vessel lumen due to accrual and decrease in blood flow to the brain increases the risk of stroke. Atherosclerosis is a primary cause of carotid artery disease (CAD). CAD often doesn't produce symptoms until the narrowing of the carotid arteries has become severe or obstruction occurs. Some characteristics, diseases, and habits add to risk of CAD development. These include age, obesity, familial history, insulin resistance and diabetes mellitus (DM), hyperlipidemia (HLD), hypertension (HT), and smoking (2).

Blood group antigen proteins are found on the cell membrane of human red blood cells. These antigens and their reaction to antibodies for type A and B blood determine an individual's blood group (3). Some ABO phenotypes are associated with cardiovascular risk factors. Studies have shown that those with B blood type have a greater risk of developing HT (4–6). Some research has also indicated that individuals of the AB blood group may be more at risk for HT than those with type O blood. In addition the B blood group has a greater risk of type 2 diabetes compared with the O blood group (7).

A greater risk of developing coronary heart disease (CHD) has been noted in the non-O blood groups in several studies (8). One evaluation of the relationship between ABO blood groups and coronary plaque characteristics indicated that non-O blood groups had more severe coronary artery stenosis (9). DM, HT, and dyslipidemia are the most common causes of atherosclerotic plaques and CAD. There is strong evidence that these risk factors may be related to blood group. The objective of this study was to identify whether the occurrence of CAD in an individual may be related to their blood group.

MATERIALS and METHODS

Ethical Considerations

This study was approved by the Afyonkarahisar Health Sciences University, Faculty of Medicine Clinical Research Ethics Committee on 01. 11. 2019, (no: 2019/11). The research was performed according to the principles of the Helsinki Declaration.

Table 1. Demographic and clinical features of the patient and control groups

	Patients (n=230)	Controls (n=136)	p
Age (years)	68±13	67.5±17.75	0.574[a]
Gender			
Female/male	73/157 (31.17/68.3)	55/81 (40.5/59.5)	0.092[b]
History of			
Hypertension	173 (76.5)	34 (25)	<0.001[b]
Diabetes	101 (43.9)	15 (13.2)	<0.001[b]
CVE/TIA	28 (12.1)	3 (2.2)	<0.001[b]
Smoker	159 (69.1)	33 (24.2)	0.001[b]
Plasma			
Glucose (mg/dL)	109.9±56.5	96.9±18.9	<0.001[a]
HDL (mg/dL)	36.2±16.65	41.8±17.15	0.005[a]
LDL (mg/dL)	110.16±36.48	96.06±25.34	0.002[c]
VLDL (mg/dL)	27.58±16.78	22.3±13.67	0.045[a]
Triglycerides (mg/dL)	135.25±84.13	106.05±61.05	0.009[a]
Total cholesterol (mg/dL)	168.1±40.78	165.24±34.24	0.652[c]
Leukocyte (10^3/µL)	7.98±3.31	7.65±2.66	0.077[a]
Platelets (10^3/µL)	227±84.5	264±102.25	0.001[a]
Hemoglobin (g/dL)	13.13±1.97	12.32±2.09	0.008[c]
MPV (fL)	9.52±1.4	9.74±1.39	0.294[c]
Neutrophils (%)	5.12±3.08	5.02±2.03	0.169[a]
Lymphocytes (%)	1.9±0.8	1.9±0.89	0.988[c]
Monocytes (10^3/µL)	0.6±0.26	0.55±0.25	0.045[a]

[a]: Results were presented as median±interquartile range. Mann-Whitney U test. P<0.05 was considered statistically significant. [b]: Results were presented as n (%). Pearson chi-squared test. P<0.05 was considered statistically significant. [c]: Results were presented mean±SD. Independent samples t-test. P<0.05 was considered statistically significant. CVE: Cerebrovascular event; TIA: Transient ischemic attack; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very-low-density lipoprotein; MPV: Mean platelet volume

Study Design and Data Collection

This retrospective study was carried out by members of the department of cardiology and the department of physiology. The files of patients diagnosed with CAD who presented at the cardiology clinic between January 2012 and November 2019 and who underwent carotid angiography were reviewed. The patient group consisted of 230 consecutive cases with 30% or more carotid artery stenosis detected with carotid artery duplex scanning. The control group comprised 136 consecutive individuals with normal carotid arteries observed on angiography and no other heart disease. A sample size of 344 was calculated using power and sample size software (Type I error α=0.05, effects size medium, power=80%).

The study data retrieved from the university's electronic database included demographic information, such as age and gender, and clinical background details, such as history of HT, HLD, DM, smoking, cerebrovascular events (CVE), transient ischemic attack (TIA), and blood group. HT was defined as a systolic blood pressure ≥140 mmHg and a diastolic blood pressure ≥90 mmHg, or use of antihypertensive drugs. DM was defined as a fasting blood glucose value of ≥126 mg/dL or use of oral antidiabetic or insulin treatment. HLD was defined as a total cholesterol value of ≥200 mg/dL or lipid-lowering therapy.

Statistical Analysis

The data were analyzed using IBM SPSS Statistics for Windows, Version 22.0 (IBM Corp., Armonk, NY, USA). The Kolmogorov-Smirnov test was used to determine the normality of the distribution of numeric variables (p>0.05). Data meeting the normality and variance homogeneity assumptions were presented as mean±SD. Pairwise comparisons were conducted with independent samples t-tests. Non-normally distributed variables (i.e., age, plasma level of glucose, high-density lipoprotein [HDL], very-low-density lipoprotein [VLDL], total cholesterol [TC], leukocytes, platelets, neutrophils, monocytes) were presented as the median±interquartile range. Pairwise comparisons were conducted using the Mann-Whitney U test. For the categorical variables (i.e., gender, blood type [O and non-O], history of HT, DM, CVE/TIA, and smoking), frequencies and percentages were used and Pearson chi-squared tests were used for comparisons. Multiple logistic regression analysis was run to examine whether the occurrence of CAD could be predicted by blood type (O and non-O) and adjustment variables (i.e., gender, age, history of HT, DM, CVE/TIA, and smoking). All of the hypothesis tests were 2-tailed. A 95% confidence interval was presented for odds ratios and p<0.05 was considered statistically significant.

Table 2. Distribution of O and non-O blood groups in patient and control groups

Blood group	Patient		Control		p
	n	%	n	%	
O	56	24.3	49	36	0.017[a]
Non-O	174	75.7	87	64	

[a]: Results were presented as n (%). Pearson chi-squared test. P<0.05 was considered statistically significant

RESULTS

There was no statistically significant difference between the patients and the controls in terms of age (respectively, 68±13 years and 67.5±17.75 years, p=0.574) or gender (p=0.092). Of the study group, 68.3% of the patients were male, 31.17% were female, while 59.5% of the controls were male and 40.5% were female (Table 1). As shown in Table 1, there was a high prevalence of HT, DM, smoking, CVE/TIA, and CAD in the patient group when compared with the control group. The mean fasting glucose, LDL, VLDL, triglyceride, and TC values were also higher in the patient group, and the HDL value was lower. The differences were statistically significant, with the exception of TC. The complete blood count results indicated that the mean platelet count was lower in the patient group and the hemoglobin and monocyte values were higher; the differences were statistically significant (p<0.05).

In the patient group, 50% of participants had blood type A, 19.6% had type B, 24.3% had type O, and 6.1% had type AB. In the control group, 44.9% had type A, 13.2% had type B, 36% had type O, and 5.9% had type AB. A Pearson chi-squared test revealed that the distribution of O and non-O blood groups differed significantly between patients and controls (p=0.017, Table 2). The risk of CAD in the non-O blood group was found to be 1.75 times higher than in the O blood group. Multiple logistic regression analysis of the association between CAD (i.e., patient or control) and blood type (i.e., O and non-O) revealed that the relationship was still significant even when adjustment variables (i.e., gender, age, history of HT, DM, and CVE/TIA, and smoking) were controlled. The predictors in the model explained 48% of the variance (Nagelkerke R squared) and the model fit the data (Hosmer-Lemeshow (8)= 5.474, p=0.706). Beta coefficients indicated that all of the predictors, except age and a history of smoking were significantly related to the occurrence of CAD (Table 3). Odds ratios revealed that the risk of CAD for individuals with a history of HT, DM, CVE/TIA was 5.35, 3.98, and 4.53 times higher, respectively, than that of individuals without such history. The CAD risk for males was 2.02 times higher than that of females. The risk was also 1.92 times higher among individuals with a non-O blood type when compared with the O blood type, indicating that blood type was still a significant predictor even after controlling for other CAD predictors.

Table 4 shows the expected and observed stenosis rates according to blood group in the patients diagnosed with CAD. The expected stenosis rates were determined using a chi-squared test consistent with the model that included other parameters used in the study, and the statistical software. Stenosis percentages were divided into 3 groups: ≤50%, 50–70%, and >70%. Comparison of the number of cases seen and expected with regard to stenosis percentage in the O blood group revealed fewer cases observed than expected (p<0.05). The lipid profiles and hematological indexes were also compared between the O blood group and the non-O blood group to assess the relationship between blood group and CAD (Table 5).

DISCUSSION

Atherosclerosis is the most common cause of CAD. The atherosclerotic process leads to the narrowing of the carotid arteries and stenosis. The stenosis can cause cranial thromboembolisms in the carotid artery, resulting in ischemic stroke or mortality. Various treatment options have been developed to prevent or control these circumstances. However, since ischemic stroke can occur despite medical treatment, the scientific world has been compelled to seek alternative treatment methods (10).

Since the discovery of ABO blood groups in 1901, many studies have examined potential relationships between blood groups and various diseases (11). Research has revealed that the ABO blood group type may be related to the risk of development of diseases such as cardiovascular disease, hematological disorders, metabolic diseases, cancer, cognitive disorders, and severe acute respiratory

Table 3. Multiple logistic regression of CAD on blood type and adjustment variables

	Beta	p	Odds ratio	95% CI for odds ratio	
				Lower	Upper
Blood type (O and non-O)	0.654	0.032	1.923	1.058	3.496
Gender	0.704	0.023	2.022	1.101	3.714
Age	0.008	0.536	1.008	0.983	1.033
Hypertension	1.677	0.000	5.350	2.986	9.585
Diabetes	1.381	0.000	3.981	1.981	8.000
CVE/TIA	1.511	0.000	4.532	2.600	7.900
Smokers	1.266	0.065	3.547	0.924	13.619

CAD: Coronary artery disease; CI: Confidence interval; CVE: Cerebrovascular event; TIA: Transient ischemic attack

Table 4. Distribution of stenosis percentage by blood group

Blood Group	<50%	50–70%	>70%	Total
O				
Count	20	26	10	56
Expected count	12.7	20.5	22.9	56
A				
Count	8	41	66	115
Expected count	26	42	47	115
B				
Count	16	14	15	45
Expected count	10.2	16.4	18.4	45
AB				
Count	8	3	3	14
Expected count	3.2	5.1	5.7	14

syndrome-related coronavirus 2 infection (12–14). In some population studies, non-O blood groups have been identified as a potential risk factor for cardiovascular disease (11). Atherosclerosis, dyslipidemia, HT, and DM are among the risk factors determined in the etiology of carotid artery disease (10). Although researchers have been evaluating the relationship between ABO blood groups and various diseases for years, no study to define the relationship between the ABO group and CAD was found in a literature review. To the best of our knowledge, the present study is original and represents a valuable contribution to the literature.

Some studies have examined the relationship between CHD and ABO blood groups (15–17) and we know that CHD has a similar etiology to CAD. It was reported in 1 study that the non-O blood group had more severe coronary artery stenosis and that these individuals could be at risk for CHD (9). CHD has been reorted to be less common among people with an O blood group (15). Compared with the O blood group, A, B, or AB blood groups have been said to have a greater risk of coronary heart disease of 5%, 11%, and 23%, respectively (18). The findings obtained in our study appear to be consistent.

The ABO blood group antigens may be a potential independent risk factor for atherosclerotic plaque formation given the different incidence of thrombotic events, higher systolic and diastolic blood pressure, and dyslipidemia (18). Some researchers have found that high plasma levels of Von Willebrand Factor (VWF) and Factor VIII, which are coagulation factors, were related to the clinic prevalence of thrombotic events (19). It has been demonstrated that patients with an AB blood group had a high VWF level, which was associated with thrombus formation (20). Supporting studies reported that VWF levels were lower in those with the O blood group than non-O groups (21, 22). Vlot et al. (23) also noted that VWF levels were lower in the O blood group. The researchers found that the plasma half-life of VWF was shorter in the O blood group than other blood groups.

Biswas et al. (24) evaluated the distribution of the ABO blood groups in a segment of CHD patients in India. They found that individuals in the AB blood group had a lower risk of developing cardiovascular disease than other blood groups and the O group had a greater risk. They linked this finding to lower HDL cholesterol in the O blood group, as well as other factors, and the higher concentration of HDL cholesterol in the AB blood group. Several studies have addressed a positive correlation between individuals with CHD and non-O blood groups and high serum cholesterol.

Table 5. Clinical variables of O and non-O blood groups in patients with carotid artery disease

	O	Non-O	p
Age (years)	67±14	69±13	0.281[a]
Plasma			
Glucose (mg/dL)	111.7±93.85	107±48	0.323[a]
HDL (mg/dL)	33.2±17.55	37.4±16.8	0.359[a]
LDL (mg/dL)	110.75±35.14	109.96±37.04	0.891[b]
VLDL (mg/dL)	26.66±18.82	26.92±16.56	0.505[a]
Triglycerides (mg/dL)	133.3±93.85	19.6±82.65	0.371[a]
Total cholesterol (mg/dL)	165.73±36.81	168.9±42.1	0.625[b]
Leukocytes (10^3/µL)	7.85±3.125	8.1±3.28	0.568[a]
Platelets (10^3/µL)	229±99	228±80.5	0.907[a]
Hemoglobin (g/dL)	13.1±2.21	13.14±1.89	0.907[b]
MPV (fL)	9.65±1.27	9.47±1.44	0.411[b]
Neutrophils (%)	5.43±3.35	5.14±2.97	0.845[a]
Lymphocytes (%)	1.99±0.97	1.87±0.74	0.425[b]
Monocytes (10^3/µL)	0.56±0.33	0.6±0.24	0.501[a]

[a]: Results were presented as median±interquartile range. Mann-Whitney U test. P<0.05 was considered statistically significant. [b]: Results were presented as mean±SD. Independent samples t-test. P<0.05 was considered statistically significant. HDL: High-density lipoprotein; LDL: Low-density lipoprotein; VLDL: Very-low-density lipoprotein; MPV: Mean platelet volume

The correlation was stronger than in individuals with an O blood group (25, 11). In our study, there was no statistically significant difference found in the lipid profiles of the O blood group and the non-O blood group in patients with CAD.

There is also a contradiction in the literature findings regarding the association between blood groups and type 2 DM, another important risk factor for CAD. Although it has been reported that the frequency of type 2 DM was greater in some blood groups, a precise identification remains inconclusive (18). Our results indicated that the CAD group had a greater proprotion of patients with DM and HT in comparison with the controls. According to the results of our study, 0 blood group, which we think has a protective effect on CAD, reduces the percentage of stenosis in carotid artery patients to 70% and below. Huang et al. (9) also evaluated the relationship between ABO blood groups and coronary plaque characteristics. They found that non-O blood group patients had more severe coronary artery stenosis than the O blood group patients (9). This finding supports the low percentage of stenosis in O blood group patients with CAD in the present study.

Limitations

The fact that this was a single-center study constitutes a limitation to the interpretaion of the findings. The power analysis conducted suggests that the sample size was sufficient, but a larger sample would have enabled us to obtain more robust results.

CONCLUSION

The findings of this study with control for risk factors indicated that non-O blood group patients had a 1.92 greater risk of developing CAD. The non-O blood group had a higher percentage of stenosis in the carotid arteries. This result supports the possibility that the O blood group could be a protective factor for CAD. The contributing factors are complex. Racial and ethnic distribution of blood groups and sample size are important factors in predicting the risk of atherosclerosis. Understanding the patient's risk, the blood type, and other risk factors must all be considered comprehensively. Although blood group appears to be an important factor in predicting the risk of atherosclerosis, to understand the patient's risk, the blood group must be considered in conjunction with other variables.

The data from this study may also be beneficial to health planners and help deal with future regional health problems. Greater knowledge of blood groups and associated diseases and a related database would not only provide data of the availability of human blood that may be useful in the event of a regional disaster, but also to learn about the possibile future burden of disease.

Acknowledgements: The authors would like to thank Hatice Ülkü, Bahriye Nur Kaya, Doğukan Özen, Zeynep Yağmur Girgin, Aleyna Durgun, and Hasan Samet Kurt from third-year Medical Faculty students who helped to collect the data of the article.

Ethics Committee Approval: The Afyonkarahisar University of Health Sciences Clinical Research Ethics Committee granted approval for this study (date: 01.11.2019, number: 2019/11).

Informed Consent: Written informed consent was obtained from patients who participated in this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept – ÖK, TK; Design – ÖK, ZY; Supervision – ÖK, AKA; Resource – ÖK, ZY; Materials – ÖK, ZY; Data Collection and/or Processing – ÖK, ZY, AKA; Analysis and/or Interpretation – ÖK, ZY, AKA; Literature Search – ÖK, TK; Writing – ÖK, ZY, AKA; Critical Reviews – ÖK, ZY, AKA, TK.

Conflict of Interest: The authors have no conflict of interest to declare.

Financial Disclosure: The authors declared that this study has received no financial support.

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