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Original Article

Does Blood group A predict slow coronary flow in patients undergoing elective coronary angiography?

A Kan Grubu Elektif Koroner Anjiyografi Uygulanan Hastalarda Yavaş Koroner Akım Öngörüyor Mu?

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ABSTRACT

Aim: Coronary slow flow (CSF) is an angiographic phenomenon characterized by slow progression of contrast in the coronary arteries in the absence of coronary artery obstruction. As it is not considered as benign finding and there is still no convincible pathophysiologic explanation, further research is needed. In the present study, we aimed to evaluate the relationship between ABO blood groups and coronary flow characteristics.

Material and Methods: The clinical, laboratory and angiographic data of 230 patients with SCF were collected retrospectively from our institutional databases. A total of 250age- and gender-matched subjects with normal coronary flow (NCF) were used as a control group. Coronary flow was assessed by using the thrombolysis in myocardial infarction frame count (TFC). Coronary flow characteristics were evaluated according to ABO blood groups.

Results: A total of230 patients with SCF (125 males, mean age: 54.6±9.1 years) and 250 subjects with NCF (134 males, mean age: 53.7±10.1 years) were included. In SCF group, cigarette smoking (33.5% vs %23.2; p=0.012), platelet count (257.2±73.3 x103/mm3 vs. 240.5±63.7 x103/mm3, p=0.011) and mean MPV (9.2±1.4 fL vs. 8.8±1.2 fL, p=0.001) were higher than subjects with NCF. Having blood group A was more common in SCF group than subjects with NCF (53.5% vs 41.2%; p=0.039).In the regression analysis, blood group A (OR=1.94; p=0.003), cigarette smoking (OR=1.57; p=0.033), platelet count (OR=1.03; p=0.008) and MPV (OR=1.27; p=0.002) were found to be as independent predictors of SCF.

Conclusion: Blood group A is more common in SCF group and independently predicts SCF.Further studies are needed to evaluate the underlying mechanisms, but the relationship between blood groups and SCF seems multifactorial.

Keywords: Blood group antigens; complete blood count; coronary slow flow; platelets; TIMI frame count.

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ÖZ

Amaç: Koroner yavaş akım (KYA), koroner arter tıkanıklığı yokluğunda, koroner arterlerde kontrastın yavaş ilerlemesi ile karakterize anjiyografik bir fenomendir. Benign bulgu olarak görülmediğinden ve hala ikna edici patofizyolojik bir açıklama olmadığı için daha fazla araştırmaya ihtiyaç vardır. Bu çalışmada, ABO kan grupları ve koroner akış karakteristikleri arasındaki ilişkiyi değerlendirmeyi amaçladık.

Gereç ve Yöntemler: KYA'lı 230 hastanın klinik, laboratuvar ve anjiyografik verileri retrospektif olarak kurumsal veri tabanlarımızdan toplandı.250 yaş cinsiyet uyumlu normal koroner akımı olan hasta kontrol grubu olarak kullanıldı.Koroner akım TIMI kare sayısı kullanılarak değerlendirildi.Koroner akım özellikleri ABO kan gruplarına göre değerlendirildi.

Bulgular: KYA'ltoplam 230 hasta (125 erkek, ortalama yaş: 54,6 ± 9,1 yıl) ve normal koroner akım(NKA)'lı 250 hasta (134 erkek, ortalama yaş: 53,7 ± 10,1 yıl) çalışmaya dahil edildi.KYA grubunda sigara içiciliği (33.5% vs %23.2; p=0.012), trombosit sayısı (257.2±73.3 x103/mm3 vs. 240.5±63.7 x103/mm3, p=0.011)ve ortalama trombosit hacmi(9.2±1.4 fL vs. 8.8±1.2 fL, p=0.001)NKA'lılardandaha yüksekti.A kan grubuna sahip olmak KYA grubunda,NKA'lı bireylerden daha yaygındı.Regresyon analizinde kan grubu A (OR = 1.94; p = 0.003), sigara içimi (OR = 1.57; p = 0.033), trombosit sayısı (OR = 1.03; p = 0.008) ve ortalama trombosit hacmi (OR = 1.27; p = 0.002) KYA'nınbağımsız belirleyicileri olarak bulundu.

Sonuçlar: A Kan grubu, KYA grubunda daha yaygındır ve bağımsız olarak KYA'yıöngörür. Altta yatan mekanizmaları değerlendirmek için daha ileri çalışmalara ihtiyaç vardır, ancak kan grupları ve SCF arasındaki ilişki çok yönlü görünmektedir.

Anahtar kelimeler: Kan grubu antijenleri; tam kan sayımı; koroner yavaş akım; trombositler; TIMI kare sayısı

Introduction

The slow coronary flow (SCF) is an angiographic finding characterized by delayed distal coronary vasculature opacification in the absence of significant epicardial coronary atherosclerosis(1). Although it is well-known clinical entity, the pathogenic mechanisms are incompletely understood.SCF is a frequent angiographic finding, with a reported incidence of 1%-7% in patients undergoing diagnostic coronary angiography (CAG) because of clinical suspicion of coronary artery disease (CAD)[1-3]. Rather than representing a simple angiographic finding, SCF has direct clinical manifestations, as it has been linkedto electrocardiographic changes, myocardial ischemia, lifethreateningarrhythmias, sudden cardiac death, and recurrentacute coronary syndromes[4-6]. Endothelial dysfunction, microvascular abnormalities, occult atherosclerosis and inflammatory processes are among the proposed responsible factors that contribute to the pathogenesis of SCF [1,7].

Several studies have raised the possibility of ABO blood groups antigens in pathogenesis of CAD[8,9]. However, most of these studies reported a significant relationship between ABO blood groups and acute coronary syndromes or sudden cardiac death[8-11]. Increased prothrombotic state, higher prevalence of conventional cardiovascular risk factors, and higher level of systemic inflammatory responsein patients with different ABO blood groups were the possible underlying factors in development of cardiovascular diseases[12,13]. The non-O blood groups (A, B, and AB) have been shown to be more prone to arterial and venous thrombotic diseases [11,14]. The pathophysiology of SCF is still not fully understood and the role of blood groupsthat increase thrombotic susceptibility seems to be worth further researching. Considering these facts, herein, we aimed to evaluate the relationship between ABO blood groups and coronary flow characteristics.

Material and Methods

Study Population

The clinical, laboratory and angiographic data of 230 patients with SCF were collected retrospectively from the Turkey Yuksek Ihtisas Training and Research Hospital registry and Ankara Numune Research and Training Hospital registry between January 2007 and August 2016. A total of 250 age- and gender-matched subjects with normal coronary flow (NCF) and normal coronary artery were used as a control group. These subjects in control group were collected from our last 2 years CAG database. Exclusion criteria were decompensated heart failure, significant valvular heart disease, history of acute coronary syndrome, previous revascularization, recent infection, cancer, autoimmune diseases, renal and/or liver failure. Hypertension was defined as a systolic blood pressure ≥ 140 mm Hg and/or a diastolic blood pressure ≥90 mm Hg in at least 2 measurements or current use of any antihypertensive. Diabetes mellitus was defined as a fasting plasma glucose level of >126 mg/dL or >200 mg/dL at any measurement or use of any oral antidiabetic. Smoking was defined as current smoking. The study complies with the principles outlined in the Declaration of Helsinki and approved by Institutional Ethics Committee. Informed constents were collected from all patients.

Coronary Angiography

All patients underwent CAG with radial or femoral approach using the Judkins technique. None of the patients experienced arterial hypotension during the procedure. Indications for CAG were made according to the results of non-invasive stress tests or high clinical suspicion for CAD. Selective cine-angiographic images of the coronary arteries were recorded using a digital angiographic system (AXIOM Sensis; Siemens AG, Munich, Germany). The contrast agent used was iopromide (Omnipaque; GE Healthcare, Cork, Ireland). All CAGswere independently quantified by 2 experienced invasive cardiologists blinded to the clinical details of the study groups. Coronary flow was assessed by using the thrombolysis in myocardial infarction frame count (TFC). Briefly, the number of cine frames that was recorded at 25 frames/s required for the contrast to first reach standard distal coronary landmark in the left anterior descending (LAD) artery, left circumflex (LCX) artery, and right coronary artery (RCA) were measured. The distal ends for the coronary vessels were defined as distal bifurcation for the LAD, distal bifurcation of the major branch for the LCX, and the first side branch of the right posterolateral artery for the RCA. The LAD artery is usually longer than the other major coronary arteries, and the TFC for this vessel is often higher. TFC was divided by 1.7 to obtain corrected TFC (cTFC) for LAD (15). The average cTFC values were used for analysis. The standard mean values for normal visualization of coronary arteries are defined as previously described (15). TFC ≥2 standard deviations from the normal published range of any one of the 3 coronary arteries were considered to have SCF (15). Intra-observer and inter-observer variabilities were determined from first30 patients from the study sample. The κ value for intra-observer reliability was 0.95 ($p < 0.001$). The interobserver reliability was a κ of 0.92 ($p < 0.001$) for the assessment of cTFC values.

Laboratory Measurements

In our hospital for preparation before angiography, venous blood samples were obtained from patients in the morning, after 12 hours of complete fasting, for measurement of serum biochemistry panel including lipid parameters. Samples for the complete blood count analysis were collected in EDTA anticoagulated Monovette tubes (Sarstedt, Leicester, United Kingdom). Calibration was assessed daily with the commercially available calibrant (Beckman Coulter; Fullerton, California), and monitored 3 times daily with internal quality control material. The ABO blood groups were tested using agglutination techniques.

Statistical Analysis

Statistical analyses were performed by Statistical Package for Social Sciences (SPSS) for Windows 20 (IBM SPSS Inc., Chicago, IL). Kolmogorov-Smirnov test was used for normal distribution of data. Normally distributed continuous variables were analyzed using T test and expressed as mean±standard deviation. Abnormally distributed continuous variables were analyzed using Mann-Whitney U test and expressed as median (min-max). Categorical variables were expressed in numbers and percentage. Chi-square test and Fisher's exact chi-square test were used compare the categorical variables. Independent predictors for SCF by stepwise method logistic regression. $p<0.05$ was considered significant for statistical analyses.

Results

A total of 230 patients with SCF (125 males, mean age:54.6±9.1 years) and 250 subjects with NCF(134 males, mean age:53.7±10.1 years) were included.Cigarette smoking were common in SCF group with respect to subjects with NCF (33.5% vs %23.2; p=0.012). There was no significant difference regarding with other demographic and angiographic findings. Detailed results are given in the table1. Having blood group A was more common in SCF group than subjects with NCF (53.5% vs 41.2%; p=0.039) (Figure 1).

Table 1. Comparison of patients with and those without coronary slow-flow phenomenon regarding demographic and CAG findings.

Variables	Patients with SCF	Patients with NCF	P
	n=230	n=250	
Clinical parameters			
Gender, n(%)			
Male	125(54.3)	134(53.6)	0.870
Female	105(45.7)	116(46.4)	
Age, years	54.6±9.1	53.7±10.1	0.305
Diabetes mellitus, n (%)	31(13.5)	30(12.0)	0.627
Hypertension, n (%)	83(36.1)	80(32.0)	0.345
Current smoking, n (%)	77(33.5)	58(23.2)	0.012*
Angiographic parameters			
LAD TFC	47.3±2.3	22.8±2.3	<0.001*
CX TFC	34.1±2.6	19.1±2.4	<0.001*
RCA TFC	31.2±1.9	15.3±2.3	<0.001*

Values are mean ± SD or median (min-max) or percentage
*significant at the 0.05 level
Abbreviations: CX= circumflex; CAG=coronary angiography ;LAD= left anterior descending; LVEF= left ventricular ejection fraction; NCF= normal coronary flow; RCA= right coronary artery; SD=standard deviation; SCF= slow coronary flow; TFC= TIMI frame count; TIMI=thrombolysis in myocardial infarction.

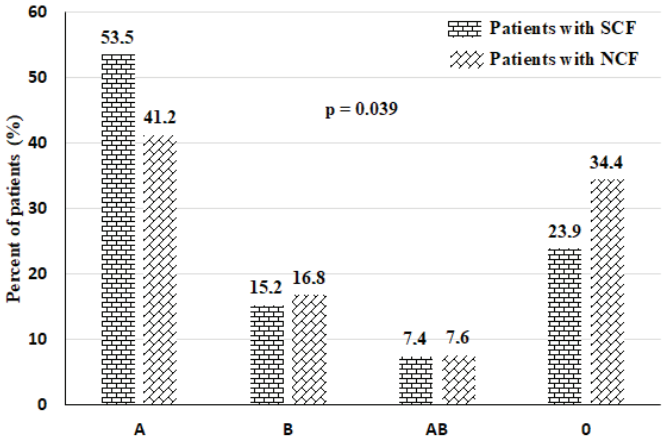

Figure 1: *Distribution of ABO bloodgroups in studypopulation (NCF= normal coronaryflow; SCF=slowcoronaryflow).*

Among subjects with SCF, TFC were higher in subjects with blood group A than the ones with non-A blood group (LAD 48.5±2.3 vs 45.8±1.8; p<0.001, Cx35.2±2.8 vs 32.8±2.1; p<0.001and RCA 34.6±2.2 vs 32.3±1.4; p<0.001). Distribution of TFC of coronary arteries with respect to different blood group antigens in study population were shown in Figure 2.

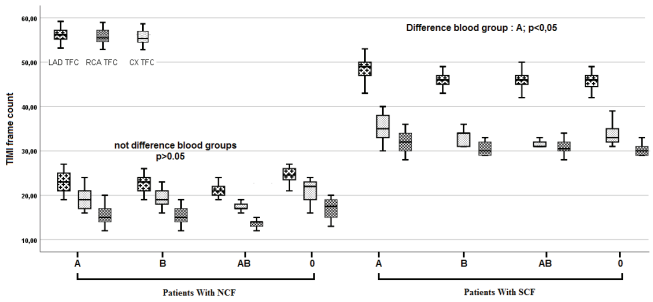

Figure 2: *Distribution of TFC of coronaryarterieswithrespect-todifferentbloodgroup in studypopulation. No difference in bloodgroupswith NCF (p>0.05). Significantdifferencebetween A andnon-A bloodgroupswith SCF (p<0.05). (NCF= normal coronaryflow; SCF= slowcoronaryflow).*

In SCF group, platelet count (257.2±73.3 x103/mm3vs. 240.5±63.7 x103/mm3, p=0.011) and mean MPV(9.2±1.4 fL vs.8.8±1.2 fL, p=0.001)were higher than subjects with NCF. There was no significant difference between groups with respect to other laboratory findings (Table 2).

Independent predictors of SCF that were determined from univariate logistic regression analysis, namely type of blood group, smoking, platelet count and MPV were assessed in stepwise regression model. Having blood group A (OR=1.94; p=0.003), cigarette smoking(OR=1.57; p=0.033), platelet count (OR=1.03; p=0.008) and MPV (OR=1.27; p=0.002) were found to be as independent predictors of SCFmultivariate logistic regression analysis. Details of regression analysis were shown in Table 3.

Table 2. Comparison of laboratoryfindings in patientswith-andthosewithoutcoronaryslow-flowphenomenon.

Variables	Patients-With SCF n=230	PatientsWith NCF n=250	p
Glucose, mg/dL	91.0±19.2	89.1±15.2	0.217
Serum creatinine, mg/dL	0.86±0.10	0.85±0.08	0.084
Urea, mg/dL	35.0±3.1	34.5±3.7	0.108
Total cholesterol, mg/dL	197.4±38.3	194.4±37.2	0.394
LDL-C, mg/dL	118.0 ±33.6	115.7±32.5	0.445
HDL-C, mg/dL	47.8±11.3	48.6±11.4	0.566
Triglyceride, mg/dL	150(52-394)	142(34-360)	0.761
WBC, 103/mm3	7.3±2.0	7.0±1.8	0.106
Hemoglobin, g/dL	13.4±1.8	13.7±2.3	0.200
Neutrophil, x103mL	3.9(1.1-9.7)	3.6(1.0-9.6)	0.337
Lymphocyte, x103mL	1.7(0.1-6.3)	1.6(0.1-6.7)	0.832
Monocyte, x103mL	0.5±0.1	0.5±0.1	0.100
Platelets, 103/mm3	257.2±73.3	240.5±63.7	0.011*
Hematocrit (%)	35.4±6.3	35.0±6.4	0.431
RDW, %	13.8±1.4	13.6±1.5	0.363
MPV, fL	9.2±1.4	8.8±1.2	0.001*
PDW, %	12.9±1.4	12.6±1.5	0.080

Valuesaremean± SD ormedian (min-max).
*significant at the 0.05 level
Abbreviations:HDL-C=high-densitylipoproteincholesterol;LDL-C=low-densitylipoproteincholesterol;MPV=Meanplateletvolume; PDW=Plateletdistributionwidth; RDW=Reddistributionwidth; WBC= White bloodcell

Table 3. Independent Predictors of the SCF Phenomenon

Risk factor	Univariable				Multivariable			
	OR	95% CI		P	OR	95% CI		p
		lower	upper			lower	upper	
Blood groups								
0	ref				ref			
A	1.87	1.22	2.86	0.004*	1.94	1.25	3.01	0.003*
B	1.30	0.74	2.29	0.356	1.48	0.83	2.65	0.183
AB	1.40	0.67	2.92	0.372	1.41	0.67	3.00	0.366
Smoking	1.67	1.12	2.50	0.013*	1.57	1.04	2.38	0.033*
Platelets	1.03	1.01	1.06	0.011*	1.04	1.01	1.06	0.008*
MPV	1.28	1.10	1.48	0.001*	1.27	1.09	1.48	0.002*
					Nagelkerke R2: 0.385; p<0.001*			

*significant at the 0.05 level
Abbreviations:CI=Confidenceinterval; MPV=Meanplateletvolume; PDW=Plateletdistributionwidth; OR=Oddsratio

Discussion

To the best of our knowledge, this is the first study to assess relationship between ABO blood group and CSF. The main findings of the present study are as follows: (i) blood group A was more common in SCF group than subjects with NCF, (ii) platelet count and mean MPV were higher in patients with SCF than subjects with NCF, (iii) having blood group A, cigarette smoking, platelet count and MPV were found to be as independent predictors of SCF. In addition, there is no significant difference in patients with blood group 0, B, or AB.

SCF has a diverse presentation from mild chest discomfort to ST-segment elevation myocardial infarction [1,16]. Different pathophysiological mechanismsfor SCF have been proposed previously such as propensity to thrombosis, microvascular injury, microvascular disease, endothelial dysfunction, and atherosclerosis [17].

In the literature, it has been shown that non-O blood groups associated with increased risk for arterial and venous thromboembolic events, intermittent claudication and cerebral ischemia [11]. Recently, Sun et al.[14] reported significant correlation between non-O blood type and risk of VTE in a hospital-based study. Moreover, several studies investigated the relationship between ABO blood groups and coronary artery disease (CAD) [11]. In a study conducted in the Portuguese population, authors found that there is significantly more frequent left main stenosis in patients with blood group A, and single-vessel disease in patients with blood group AB [18]. In patients from Northern Finland, undergoing coronary artery by-pass grafting surgery, blood group B was associated with a higher angiographic score [19]. In the optic coherence tomography study, it was shown that the plaques of O type blood group were exhibited more stably compared with non-O type blood group [20]. In a relatively recent meta-analysis in which 225,810 participants were included, authors reported that both blood group A and non-O were the risk factors of CAD [21]. Although it is often stated that risk increases in non-O blood group patients, some studies have focused on blood group A. Namely, Whincup et al. [22] found that the incidence of ischemic CAD was higher in those with blood group A than that with blood group non-A. In addition, a study conducted by Wazirali et al. [23] suggested that blood group A was associated with a substantially increased risk of CAD, which is independent of conventional cardiovascular risk factors.In a study reported by Lee et al. [9], a significant association was observed between blood group A and an increased risk of CAD, in both univariate and multivariate analyses.

Although the mechanisms underlying the associations between ABO blood group and cardiovascular risk remain unclear, several lines of evidence support its potential cardiovascular effects. Blood group antigens may modulate the risk of CAD and myocardial blood supply by influencing the levels of hemostasis and inflammatory proteins in circulation [9,24]. It was reported that plasma levels of factor VIII- von Willebrand factor (vWf) complex in non-O individuals were approximately 25% higher than in group O individuals [24]. The vWf has an important role in hemostasis and thrombosis by mediating platelet adhesion to the vascular wall and participates in platelet aggregation [25,26]. In a relatively recent study, the relationship between blood groups and endothelial dysfunction, one of the important mechanisms thought to play a role in SCF has been demonstrated [27]. In addition, it was demonstrated that non-O blood groups associated with severity of CAD and microvascular thrombosis [27]. Higher risk of atherosclerosis, microvascular disease, propensity to thrombosis and endothelial dysfunction might be the underlying factors explaining the SCF and blood group A antigen relationship found in our study. Importantly, our study has not been designed to explain the underlying mechanism, but the relationship between blood groups and SCF seems multifactorial.

CSF is commonly seen patients who are current smokers [1,2]. Different theories have been postulated about the effects of smoking on coronary flow.It includes small vessel dysfunction based on observations including microvascular tone dysfunction, endothelial thickening in small vessels and impaired endothelial release of nitric oxide (NO) [1,2]. In accordance with this, we found that cigarette smoking as an independent predictor of SCF.

Platelets are important blood cells that participate in the processes of atherothrombotic events, coagulation, and inflammation [16,28]. MPV is and indicator of platelet size, volume, and activation[28]. Elevated MPV value has been shown to be involved in the pathogenesis of atherosclerosis and thrombogenesis [16,28]. As another important finding, we found that platelet count and mean MPV were higher in subjects with SCF.

Our study should be evaluated in the light of several limitations. The presented study was conducted on a retrospective basis and represented multi-center experience. We used only a one-spot blood sample for laboratory analysis with no information regarding the temporal trend of changes during follow-up.

Conclusion

In the present study, our results suggest that prevalence of blood group A, cigarette smoking, platelet count and mean MPV are higher in patients with SCF than subjects with NCF. The association between blood group A and SCF was independent from confounding parameters. Further studies are needed to evaluate the underlying mechanisms, but the relationship between blood groups and SCF seems multifactorial. Higher risk of atherosclerosis, microvascular disease, propensity to thrombosis and endothelial dysfunction are possible underlying factors explaining the propensity of SCF in subjects with blood group A.

Declaration of conflict of interest

The authors received no financial support for the research and/or authorship of this article. There is no conflict of interest.

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