

An investigation of platelet parameters in smoking patients with coronary slow flow detected during coronary angiography

Koroner anjiyografi sırasında saptanan koroner yavaş akım olan sigara içen hastalarda trombosit parametrelerinin araştırılması

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Abstract

Aim: Coronary slow flow (CSF), which is linked to increased morbidity and mortality, is associated with atherosclerosis, and considered a variant of coronary artery disease (CAD). CSF is more common in smoking patients. We aimed to evaluate laboratory parameters, especially platelet indices, in smoking patients with CSF.

Methods: Patients were selected from those who underwent coronary angiography (CAG) between January 2017 and October 2019. CAG records of 7287 patients were screened retrospectively for our case-control study. Procedures were carried out to identify ischemic heart disease based on clinical indications. CAG was performed in patients with positive non-invasive stress tests and/or high clinical suspicion for atherosclerotic CAD. Smoking patients with CSF (n=226) constituted the study group and matched number (n=226) of smoking patients with NCA were included in the control group. The demographic characteristics of all patients were recorded. Hematologic and biochemical parameters of all subjects were recorded and evaluated.

Results: LDL cholesterol, triglyceride, total cholesterol, ALT, CRP, MCV, RDW, platelet count, PDW, MPV, PCT, and PLR levels were higher in smoking patients with CSF (study group) than normal coronary artery patients (control group) ($P=0.034$, $P=0.015$, $P=0.033$, $P=0.006$, $P<0.001$, $P=0.033$, $P=0.021$, $P=0.039$, $P=0.006$, $P=0.010$, $P=0.021$ and $P=0.008$, respectively). HDL cholesterol was found lower in smoking patients with CSF compared to controls ($P=0.007$).

Conclusion: According to our results, high platelet parameters may play a role in coronary flow pathogenesis. The height of platelet parameters may indicate the presence of CSF. Our findings support the evidence for inflammation and platelet dysfunction in smoking patients with CSF. Extensive studies at a randomized molecular level are needed to demonstrate this relationship.

Keywords: Coronary slow flow, Mean platelet volume, Plateletcrit, Platelet distribution width, Smoking

Öz

Amaç: Artmış morbidite ve mortalite ile ilişkili olan koroner yavaş akış (KYA), ateroskleroz ile ilişkilidir ve koroner arter hastalığının (KAH) bir varyantı olarak kabul edilir. KYA sigara içen hastalarda daha yaygındır. KYA'lı sigara içen hastalarda laboratuvar parametrelerini (özellikle trombosit indeksleri) değerlendirmeyi amaçladık.

Yöntemler: Hastalar Ocak 2017 ile Ekim 2019 arasında koroner anjiyografi (KAG) uygulanan hastalardan seçildi. 7287 hastanın KAG kayıtları retrospektif olarak tarandı. Çalışmamız bir vaka kontrol çalışması olarak tasarlandı. Bu prosedürler, klinik endikasyonlara dayanarak iskemik kalp hastalığını tanımlamak için gerçekleştirildi. KAG pozitif invaziv olmayan stres testleri ve/veya aterosklerotik KAH için yüksek klinik şüphesi olan hastalara uygulandı. KYA'lı sigara içen hastalar (n=226) çalışma grubunu oluşturdu ve NKA'lı eşleşen sayıda sigara içen hastalar (n=226) kontrol grubuna dahil edildi. Tüm hastaların demografik özellikleri kaydedildi. Tüm deneklerin hematolojik ve biyokimyasal parametreleri kaydedildi ve değerlendirildi.

Bulgular: KYA'lı sigara içen hastalarda (çalışma grubu) LDL kolesterol, trigliserit, total kolesterol, ALT, CRP, MCV, RDW, Trombosit sayısı, PDW, MPV, PCT ve PLR düzeyleri normal koroner arter hastalarına (kontrol grubu) göre daha yüksek bulundu (sırasıyla $P=0,034$, $P=0,015$, $P=0,033$, $P=0,006$, $P<0,001$, $P=0,033$, $P=0,021$, $P=0,039$, $P=0,006$, $P=0,010$, $P=0,021$ ve $P=0,008$). HDL kolesterol KYA'lı sigara içen hastalarda kontrol grubuna göre daha düşük bulundu. ($P=0,007$).

Sonuç: Sonuçlarımıza göre, yüksek trombosit parametreleri koroner akım patogeneğinde rol oynayabilir. Trombosit parametrelerinin yüksekliği KYA varlığını gösterebilir. Bulgularımız sigara içen KYA hastalarında inflamasyon ve trombosit fonksiyon bozukluğu kanıtlarını desteklemektedir. Bu ilişkiyi göstermek için randomize moleküler düzeyde kapsamlı çalışmalara ihtiyaç vardır.

Anahtar kelimeler: Koroner yavaş akım, Ortalama trombosit hacmi, Plateletkrit, Trombosit dağılım genişliği, Sigara kullanımı

Introduction

The delayed progression of opaque material in epicardial coronary arteries without stenosis is known as coronary slow flow (CSF) [1]. The pathophysiology of CSF is not fully understood, and its prevalence varies between 1-7% [2]. CSF is considered a variant of coronary artery disease (CAD) [3]. Clinical presentation of CSF patients can range from atypical chest pain to ST-elevated myocardial infarction [4]. Endothelial dysfunction is considered the foundation of CSF pathogenesis [1,5]. Other proposed pathophysiological mechanisms are platelet dysfunction, diffuse atherosclerosis, imbalance of vasoconstrictor and vasodilator functions, small vessel disease, and inflammation [6-10]. Inflammation plays a vital role in atherosclerosis [11], and platelet function disorders are thought to play a role in the development of CSF [12]. Hence, inflammation and platelet dysfunction are shown to be effective in CSF formation [13]. The treatment strategy in CSF patients is also unclear, as the pathophysiology is not fully known [14].

Platelets significantly trigger the formation and progression of atherosclerotic CAD, and play a significant role in fatal thrombosis [15]. In patients with acute coronary syndrome, they were shown to be more active than in healthy controls [16]. The mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) derived from a complete blood count are indices specific to platelet morphology and proliferation kinetics [17].

In this study, we aimed to evaluate laboratory parameters, especially platelet indices, in smoking patients with CSF.

Materials and methods

Patient selection

Patients were selected from those who underwent coronary angiography (CAG) between January 2017 and October 2019. CAG records of 7287 patients were screened for this case-control study. Flow diagram with exclusion criteria is summarized in Figure 1.

A Siemens Axiom Artis diagnostic device (Siemens Healthcare GmbH, Forchheim, Germany) was used to perform CAG in patients with positive non-invasive stress tests and/or high clinical suspicion for atherosclerotic CAD. These procedures were conducted to identify ischemic heart disease based on clinical indications.

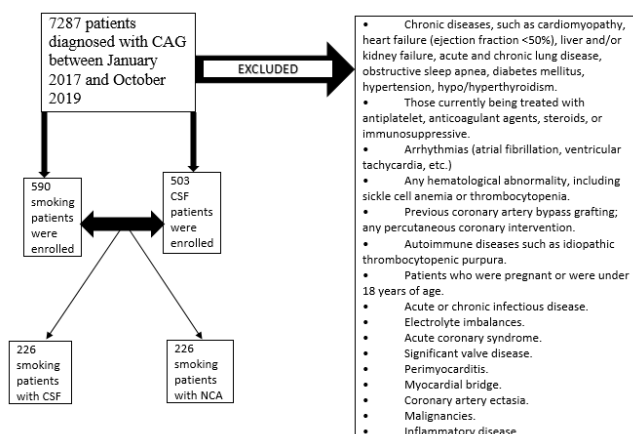


Figure 1: Flow diagram of the study (CAG: Coronary angiography, CSF: Coronary slow flow, NCA: Normal coronary artery)

Various risk factors related to CSF, such as hypertension, obesity, diabetes, smoking status, dyslipidemia, previous history of coronary artery disease, and family history (presence of disease in first-degree relatives) were recorded. Body mass index was calculated by dividing weight in kilograms by the height in meters squared (kg/m²). Hematologic and biochemical parameters of all subjects were recorded and evaluated. Smoking patients with CSF (n=226) constituted the study group and matched number (n=226) of smoking patients with NCA were included in the control group.

Bolu Abant Izzet Baysal University Ethics Committee approved the study (Decision date: 21/11/2019, decision number: 2019/284), which was conducted in accordance with the principles of the Helsinki Declaration.

Statistical analysis

SPSS 20.0 Statistical Package Program for Windows (SPSS Inc, Chicago, Illinois) was used in all statistical analyses. Kolmogorov-Smirnov test was used to evaluate the distribution model. Normally distributed numerical variables were presented as mean (standard deviation) and non-normally distributed ones were presented as median and range. The significance of the difference between the mean values of the groups were evaluated with Student's t-test. Mann-Whitney U test was used to assess the significance of the difference between continuous numerical variables. Categorical variables were compared with the Chi-square test. Confidence interval was accepted as 95%. A *P*-value <0.05 was considered statistically significant.

Results

The demographic characteristics of smoking patients with CSF (study group) and smoking patients with NCA (control group) are presented in Table 1.

LDL cholesterol, triglyceride, total cholesterol, ALT, CRP, MCV, RDW, platelet count, PDW, MPV, PCT, and PLR levels were higher in smoking patients with CSF (study group) than normal coronary artery patients (control group) (*P*=0.034, *P*=0.015, *P*=0.033, *P*=0.006, *P*<0.001, *P*=0.033, *P*=0.021, *P*=0.039, *P*=0.006, *P*=0.010, *P*=0.021, and *P*=0.008 respectively). HDL cholesterol was found lower in smoking patients with CSF compared to the controls (*P*=0.007) (Table 2).

Table 1: General characteristics of the patients

Baseline characteristics	Study group Smoking patients with CSF (n=226)	Control group Smoking Patients with NCA (n=226)	<i>P</i> -value
Age (years)	52.9 (10.2)	53.5 (10.8)	0.534
Male/Female	194/32	182/44	0.132
LVEF (%)	60.6 (3.5)	61.0 (3.5)	0.197
Heart rate	75 (54-98)	74 (53-105)	0.809
SBP (mmHg)	120 (90-140)	120 (90-156)	0.154
DBP (mmHg)	70 (50-92)	70 (58-90)	0.200
BMI	28.3 (4.8)	28.3 (5.5)	0.957

CSF: Coronary slow flow, NCA: Normal coronary artery, LVEF: Left ventricular ejection fraction, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, BMI: Body mass index.

Table 2: Laboratory data of study groups

	Study group Smoking patients with CSF (n=226) Median (Min-Max)	Control group Smoking Patients with NCA (n=226) Median (Min-Max)	P-value
LDL-cholesterol (mg/dL)	109.2 (36.8-221)	111.5 (36.8-174.4)	0.034
Triglyceride (mg/dL)	164 (39-634)	137 (39-634)	0.015
Total cholesterol (mg/dL)	192 (101-287)	186 (101-264)	0.033
HDL-cholesterol (mg/dL)	41.6 (22.8-63.1)	42.3 (22.8-92.8)	0.007
Glomerular Filtration Rate (GFR) (%)	97.5 (71.6-125.4)	97.5 (71.6-125.4)	0.812
Sodium (Na) (mmol/L)	139 (134-146)	139 (134-146)	0.075
Potassium (K) (mmol/L)	4.3 (3.3-5.5)	4.4 (3.7-5.7)	0.159
Alanine aminotransferase (ALT) (u/l)	20 (8-64)	19 (8-61)	0.006
Aspartate aminotransferase (AST) (u/l)	22 (12-50)	20 (12-48)	0.157
Thyroid-stimulating hormone (TSH) (μ IU/mL)	1.3 (0.2-3.7)	1.3 (0.4-3.7)	0.611
C-reactive protein (CRP) (mg/L)	0.4 (0.01-15)	0.4 (0.01-4.9)	<0.001
White blood cell (WBC) (u/mm^3)	8.1 (4.0-12.4)	7.9 (4.0-11.8)	0.930
Hemoglobin (gr/dL)	14.9 (11.1-19.3)	14.6 (10.5-19.4)	0.592
Mean corpuscular volume (MCV) (fL)	89 (64.8-99.1)	88.6 (74.7 -100)	0.033
Red cell distribution width (RDW) (%)	15.4 (12.8-20)	15.2 (12.3-17.8)	0.021
Neutrophil, (u/mm^3)	4.6 (2.2-9.4)	4.6 (2.1-7.7)	0.809
Lymphocyte, (u/mm^3)	2.3 (0.8-5.9)	2.3 (1.3-5.9)	0.281
Monocyte, (u/mm^3)	0.6 (0.006-1.4)	0.5 (0.063-1.4)	0.050
Basophils, (u/mm^3)	0.07 (0.001-0.400)	0.07 (0.001-0.142)	0.604
Eosinophil, (u/mm^3)	0.16 (0.016-0.664)	0.16 (0.002-0.891)	0.966
Platelet counts (Plt) (k/mm^3)	239 (145-442)	234 (115-340)	0.039
Platelet distribution width (PDW) (%)	17.6 (14.8-20.9)	17.5 (12.3-19.8)	0.006
Mean platelet volume (MPV) (fL)	8.0 (5.7-14.0)	7.8 (5.7-9.8)	0.008
Plateletcrit (PCT) (%)	0.19 (0.10-0.41)	0.19 (0.07-0.27)	0.010
Neutrophil Lymphocyte Ratio (NLR)	1.9 (0.2-9.7)	1.9 (0.7-5.3)	0.096
Platelet Lymphocyte Rate (PLR)	99.6 (24.7-528.6)	99.1 (27.7-211.4)	0.008

CSF: Coronary slow flow, NCA: Normal coronary artery

Discussion

This study showed that low-density lipoprotein (LDL) cholesterol, triglyceride, total cholesterol, alanine aminotransferase (ALT), C-reactive protein (CRP), mean corpuscular volume (MCV), red cell distribution width (RDW), platelet count, platelet distribution width (PDW), mean platelet volume (MPV), plateletcrit (PCT), and platelet lymphocyte rate (PLR) levels were higher in smoking patients with CSF than normal coronary artery patients. HDL cholesterol was found lower in smoking patients with CSF compared to the control group. To the best of our knowledge, this is the first study to investigate platelet indices in smoking patients with CSF. In our study, we detected CSF among 6.9% of patients when we retrospectively assessed CAGs. This rate was 3.1% in smoking patients.

CSF is associated with life-threatening arrhythmia, acute coronary syndrome, and significant cardiac vascular events, including sudden cardiac death [18], along with increased cardiovascular mortality [19]. CSF risk factors are found to vary among studies. Beltrame et al. [20] have shown that male gender and smoking are independent risk factors for CSF. In a Chinese study, hyperuricemia, high hsCRP levels, thrombocytosis, and hyperglycemia were reported as independent risk factors [21]. In our study, CRP levels were high in smoking patients with CSF.

Cardiovascular diseases were associated with increased RDW [22]. A strong correlation was observed between RDW, which was reportedly linked with decreased coronary blood flow and inflammatory markers [23]. RDW levels were high in our study.

Platelets play an essential role in atherosclerotic coronary artery disease, atherothrombosis, coagulation, and inflammation processes [24]. MPV represents platelet volume. PDW shows the difference in platelet size. PCT reflects the volume occupied by platelets in a whole blood sample. These parameters are indicators of platelet activation. An increase in MPV is related with the risk of thrombosis [25]. PDW is an indicator of platelet activation in patients with CAD [26]. Gokce et al. [12] reported that the platelet aggregation rate increased significantly in patients with CSF compared to control groups. Işık et al. [27] found the MPV levels high in patients with CSF. On the contrary, Altun et al. [28] reported that MPV and PDW levels were not significantly higher in patients with CSF compared to the controls. PLR is reported to increase in patients with CSF [29]. Platelet count and increased PLR are essential markers of inflammation, and PLR was associated with mortality in ST-elevated myocardial infarction [30]. A study conducted by Çetin et al. [31] in 2016 found that neutrophil lymphocyte ratio (NLR) was high in CSF patients. On the contrary, in our study, compared to the control group, NLR was not higher in smokers with CSF, but platelet count, PDW, MPV, PCT, and PLR levels were.

Limitations

Visual evaluation of angiography images for CSF diagnosis and the inability to use intravascular ultrasound are essential limitations of the study. The presence of large atherosclerotic plaques, demonstrated by intravascular ultrasound, were shown with autopsy studies in CSF patients.

Conclusions

This study showed that LDL cholesterol, triglyceride, total cholesterol, ALT, CRP, MCV, RDW, platelet count, PDW, MPV, PCT, and PLR levels were higher in smoking patients with CSF than NCA patients, while HDL cholesterol was lower.

According to our results, high platelet parameters may play a role in coronary flow pathogenesis. Increased platelet parameters may indicate the presence of CSF. Our findings support the evidence for inflammation and platelet dysfunction in smoking patients with CSF. Extensive, randomized studies at a molecular level are needed to further demonstrate this relationship.

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