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Original Article

Evaluation of hemogram parameters in smoking patients with coronary artery ectasia

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Abstract

Aim: We aimed to compare hemogram parameters (especially platelet indices) in smoking patients with coronary artery ectasia and the normal coronary artery.

Material and Methods: The records of 7287 patients who underwent coronary angiography between January 2017 and October 2019 were reviewed. After appropriate exclusions, smoking patients were divided into coronary artery ectasia and normal coronary artery groups. A total of 292 patients were included in the study and hemogram parameters of these two groups were compared.

Results: Compared to the control group triglyceride cholesterol (136 (43-835) vs. 152 (30-835) mg/dL, p:0.030), Hemoglobin (14.6 (11.5-16.9) vs. 14.9 (11.5-17.1) gr/dL, p: 0.012), RDW (15.5 (12.9-19.9) vs. 15.2 (12.3-17.5) %, p: 0.016), Eosinophil count, (0.172 (0.002-0.877) vs. 0.143 (0.039-0.385) u/mm^3, p: 0.035), Platelet counts (Plt) (226 (143-442) vs. 218 (125-294) k/mm^3, p: 0.013), PDW (17.7 (16.1-20.9) vs. 17.5 (15.9-18.9) %, p: 0.003), MPV (8.2 (6.2-11.2) vs. 7.8 (6.1-10.6) Fl, p: 0.001), PCT (0.18 (0.07-0.32) vs. 0.17 (0.08-0.24), p: 0.017), NLR (2.1 (0.9-12.8) vs. 2.0 (1.1-4.5), p: 0.012), and PLR (102.8 (50.8-339.3) vs. 97.1 (46.0-194.3), p: 0.004), were significantly in CAE patients. There was no significant difference between the two groups in terms of other biochemical and hemogram values.

Conclusion: Hemogram parameters that are common, simple and inexpensive are increased smoking patients with coronary artery ectasia.

Keywords: Atherosclerosis; coronary artery ectasia; hemogram parameters; inflammation; smoking

INTRODUCTION

Coronary artery ectasia (CAE) is the dilation of the coronary artery lumen, and focal coronary dilation is called "coronary aneurysm" (1). The incidence of CAE ranges from 0.3–5.3 % (2). CAE occurs at a similar frequency in men and women (3). The diameter of the CAE dilated segment is 1.5 times that of normal adjacent coronary artery segments (4). CAE is related to increased morbidity and mortality (5). The etiopathogenesis of CAE is not fully understood (6). The most common cause of CAE is atherosclerosis (7). CAE can be caused by genetic diseases (Marfan syndrome, apical hypertrophic cardiomyopathy, etc.), rheumatologic disorders (polyarteritis nodosa, systemic lupus erythematosus, etc.), hypertension, smoking, cocaine use, percutaneous transluminal coronary angioplasty, stent placement, and directional coronary atherectomy (8). CAE often occurs in patients with coronary artery disease (CAD). Some studies have shown that CAE is associated with an increased risk of myocardial infarction (9). The clinical presentation of CAE patients can vary, which may affect its timely diagnosis (10). CAE may cause acute coronary syndrome, ventricular arrhythmias, and sudden cardiac death due to severe coronary artery stenosis [10]. The gold standard for diagnosing CAE is coronary angiography (CAG).

One of the most important precursors of atherosclerotic processes is endothelial dysfunction (11). The key role of inflammation in the development of atherosclerosis is well known (12). Smoking triggers endothelial dysfunction (13) and is one of the most important preventable risk factors in the development of atherosclerosis. The World Health Organization states that smoking is responsible for 10% of all CVD cases (14).

Cigaret is an addictive substance that causes many diseases in today's world. It is known that smoking affects inflammatory processes and blood viscosity. In this study, we aimed to evaluate the hemogram parameters of smoking patients with CAE.

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MATERIAL and METHODS

Patient selection

We reviewed 7287 angiograms performed between January 2017 and October 2019 at Bolu Abant Izzet Baysal University Medical Faculty Hospital. A Siemens Axiom Artis diagnostic device (Siemens Healthcare GmbH, Forchheim, Germany) was used to perform CAG procedures to investigate ischemic heart diseases based on clinical indications. The study received the ethical approval of the University Ethics Committee (Date: 21/11/2019 Decision number: 2019/285). Data about patients were obtained from the institution's database and patient files. CAG images recorded in digital format were evaluated visually by two blinded cardiologists, and patients they diagnosed with CAE were included in the study. These participants were selected from patients with chronic coronary syndrome. Patients with clear CAE evidence were selected. The baseline demographic data and clinical cardiovascular risk factors were obtained from hospital records and included hypertension, diabetes mellitus, current or former smoking, family history of CAD, dyslipidemia, weight, and height. CAE patients and a control group were matched for their demographic parameters.

Exclusion criteria

- •Hypertension
- •Diabetes mellitus
- •Heart failure (ejection fraction <50%)
- •Acute coronary syndrome (ACS)
- •Arrhythmias (atrial fibrillation, ventricular tachycardia, etc.)
- •Previous coronary artery bypass grafting
- •Percutaneous coronary intervention
- •Significant heart valve disease
- •Antiplatelet/anticoagulant agents and steroid users
- •Electrolyte imbalance
- •Any hematological abnormality (sickle cell anemia, thrombocytopenia, etc.)
- •Immunosuppressive therapy
- •Thyroid diseases (hypo/hyperthyroidism)
- •Stroke
- •Liver failure
- •Kidney failure
- •Acute and chronic lung disease
- •Patients under 18 years of age
- •Autoimmune diseases
- •Iatrogenic ectasia
- •Pregnancy
- •Pericarditis
- •Myocarditis
- •Obstructive sleep apnea
- •Chronic inflammation
- •Active infection
- •Cancer

Statistical analysis

The statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS) software (SPSS version 20.0 for Windows, IBM Co., Chicago, IL, USA). A Kolmogorov-Smirnov test was used to determine the distribution's normality. Normal variables were compared with a T-test and expressed as the mean ± standard deviation. A Mann-Whitney U test was employed for variables with an abnormal distribution and was expressed as the median (IQR: interquartile interval). A chi-square test was used to compare nonparametric variables. A p-value < 0.05 was considered statistically significant.

RESULTS

All patients were current smokers. We enrolled 292 individuals, including 146 CAE patients (mean age: 54.5±9.4 years) and 146 control participants (mean age: 53.6±8.8 years). The mean age, frequencies of sex, and body mass index were not significantly different between the study patients and the control group (Table1).

Table 1. General characteristics of the study groups

Baseline characteristics	Smoking Patients with CAE (n=146)	Smoking Patients with NCA (n=146)	P value
Age (years)	54.5±9.4	53.6±8.8	0.484
Male/female	80/66	74/72	0.725
LVEF (%)	60.8±3.4	61.1±3.4	0.253
Heart rate	73 (48-120)	74 (50-98)	0.253
SBP (mmHg)	120 (90-164)	120 (90-150)	0.081
DBP (mmHg)	71 (60-100)	70 (50-90)	0.067
BMI	27.8 (20.5-46.9)	27.2 (21.1-39.4)	0.154

CAE: Coronary Artery Ectasia, NCA: Normal Coronary Artery, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: body mass index

Laboratory test results were compared between the CAE patients and the control group. Statistical significance was found for the CAE patients for triglyceride cholesterol (136 mg/dL [range: 43–835 mg/dL] vs. 152 [range: 30–835mg/dL] for controls, p=0.030), hemoglobin (14.6 g/dL [range: 11.5–16.9 mg/dL] vs. 14.9 g/dL [range: 11.5–17.1 g/dL]

in controls, p= 0.012), RDW (15.5%[range: 12.9–19.9%] vs. 15.2%[range: 12.3–17.5%] in controls, p = 0.016), eosinophil count (0.172 u/mm3[range: 0.002–0.877u/mm^3] vs. 0.143 u/mm^3[range: 0.039–0.385 u/mm^3] for controls, p= 0.035), platelet count (226 k/mm^3[range: 143–442k/mm^3] vs. 218 k/mm3[range: 125–294 k/mm^3] in controls, p= 0.013), PDW (17.7%[range: 16.1–20.9%] vs. 17.5%[range: 15.9–18.9] in controls, p= 0.003), MPV (8.2 fL [range: 6.2–11.2 fL] vs. 7.8 fL [range: 6.1–10.6 fL] in controls, p= 0.001), PCT (0.18 [range: 0.07–0.32] vs. 0.17 [range: 0.08–0.24] in controls,p= 0.017), NLR (2.1 [range: 0.9–12.8] vs. 2.0 [1.1-4.5] in controls, p= 0.012), and PLR (102.8 [range: 50.8–339.3] vs. 97.1 [range: 46.0–194.3] in controls, p= 0.004). There was no significant difference between the two groups in terms of other biochemical and hemogram values (Table 2).

Table 2. Laboratory data of study groups

	Smoking Patients with CAE (n=146)	Smoking Patients with NCA (n=146)	p
	MEDIAN (Min-Max.)		
LDL-cholesterol (mg/dL)	114 (20.3-221)	113.1 (44.9-207)	0.403
Triglyceride (mg/dL)	136 (43-835)	152 (30-835)	0.030
Total cholesterol (mg/dL)	181 (84-395)	186 (84-395)	0.316
HDL-cholesterol (mg/dL)	41.9 (21.2-71.2)	39.9 (22.8-76)	0.201
Glomerular Filtration Rate (%)	90.7 (61.8-123.1)	96 (54.9-125.4)	0.092
ALT (u/l)	17 (4-41)	18 (8-41)	0.531
AST (u/l)	21 (12-40)	21 (12-38)	0.652
TSH	1.2 (0.5-3.7)	1.2 (0.5-3.5)	0.768
CRP (mg/L)	0.3 (0.01-20)	0.3 (0.01-15)	0.415
WBC, (u/mm^3)	8.1 (4-11.8)	7.7 (5.7-11.8)	0.311
Hemoglobin (gr/dL)	14.6 (11.5-16.9)	14.9 (11.5-17.1)	0.012
MCV	88.4 (64.8-104)	89.3 (77.3-99.1)	0.291
RDW (%)	15.5 (12.9-19.9)	15.2 (12.3-17.5)	0.016
Neutrophil, (u/mm^3)	4.6 (2.1-8.6)	4.6 (3.2-6.9)	0.129
Lymphocyte, (u/mm^3)	2.2 (0.6-5)	2.3 (1.3-3.8)	0.731
Monocyte, (u/mm^3)	0.6 (0.03-1.4)	0,6 (0.2-1.0)	0.703
Basophils, (u/mm^3)	0.07 (0.001-0.4)	0.07 (0.001-0.1)	0.057
Eosinophil, (u/mm^3)	0.172 (0.002-0.877)	0.143 (0.039-0.385)	0.035
Platelet counts (Plt) (k/mm^3)	226 (143-442)	218 (125-294)	0.013
PDW (%)	17.7 (16.1-20.9)	17.5 (15.9-18.9)	0.003
MPV (Fl)	8.2 (6.2-11.2)	7.8 (6.1-10.6)	0.001
PCT	0.18 (0.07-0.32)	0.17 (0.08-0.24)	0.017
Neutrophil Lymphocyte Ratio (NLR)	2.1 (0.9-12.8)	2.0 (1.1-4.5)	0.012
Platelet Lymphocyte Rate (PLR)	102.8 (50.8-339.3)	97.1 (46.0-194.3)	0.004

CAE: Coronary Artery Ectasia, NCA: Normal Coronary Artery, GFR: Glomerular filtration rate, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, PDW: Platelet distribution width; RDW: Red cell distribution width; MPV: Mean platelet volume; HDL: high-density lipoprotein; LDL: low-density lipoprotein; WBC: White blood count; PCT: plateletcrit.

DISCUSSION

This study showed that hemogram parameter levels were different between patients with CAE who were current smokers and control participants. To the best of our knowledge, this is the first study to evaluate hemogram parameters in smokers with CAE.

CAE is a variant of atypical coronary atherosclerosis characterized by disruption of the elastic lamina (15). Atherosclerosis (7,16) and inflammation [8] both play a role in the etiology of CAE. It is estimated that 20–30% of all CAE cases are associated with inflammatory diseases (17).

The endothelium controls blood flow, coagulation, and inflammatory processes (18). Smoking has been shown to have an effect on all stages of atherogenesis (19). The key process involved at the onset of smoking-induced atherogenesis is endothelial dysfunction (14).

Previous studies in CAE patients have shown a high neutrophil-lymphocyte ratio (NLR) (20). In our study, the smoking subgroup of CAE patients was found to have a high NLR, and the platelet-lymphocyte ratio (PLR) was also elevated. Turhan et al. (21) reported high levels of C-reactive protein (CRP) in CAE patients. The present study also found high levels of CRP in patients with CAE who were current smokers.

The mean platelet volume (MPV) is an indicator of platelet activation and has an independent effect on the pathophysiology of atherosclerosis. MPV levels are high in patients with acute myocardial infarction, unstable angina pectoris, and congestive heart failure (22). CAE patients in particular demonstrate high MPV levels (23), and this trend was also identified in the present study. All CAE patients in our study were current smokers, and they showed higher MPV levels than normal coronary arteries.

The critical role of platelets in inflammation is well established (24). The platelet distribution width (PDW) indicates the difference in platelet size, while the plateletcrit (PCT) reflects the proportion of volume that platelets make up in a whole blood sample. Studies have shown an association between PCT and PDW and inflammation (25). Our study revealed that the platelet count, PDW, and PCT were higher in smoking patients with CAE.

CONCLUSION

There is no consensus on the best treatment for CAE, which is an important cause of mortality and morbidity. Close monitoring of patients with CAE is vital. Routine hematological analyses are important tests that are also simple, effortless, and cost-effective. These tests may help predict which current smokers will go on to develop CAE, and future prospective large-scale randomized controlled trials will be required to identify the relationships between CAE and laboratory parameters in these patients.

Competing interests: The authors declare that they have no competing interest.

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Ethical approval: The study received the ethical approval of the University Ethics Committee (Date: 21/11/2019 Decision number: 2019/285).

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