Pak Heart J

ASSOCIATION BETWEEN BLOOD CELLS COUNT AND CHEMICAL BIOMARKERS WITH CORONARY ARTERY ECTASIA (CAE)

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Date Received: Jan 29, 2019

Date Revised: March 21, 2019

Date Accepted: April 27, 2019

Contribution

BZ and AS conceived the idea and designed the study. FM, NN, MM and VN did data collection and analysis. SV, AA and MG did final review. All authors contributed equally to be submitted manuscript.

All authors declared no conflict of interest.

This article may be cited as: Shad B, Salari A, Moayerifar M, Mirbolouk F, Naghshbandi M, Nikseresht V, Vahadati S, Ashouri A, Gholipour M. Association between blood cells count and chemical biomarkers with coronary artery ectasia (CAE). Pak Heart J 2019; 52 (03): 212-8

ABSTRACT

Objective: To investigate the relationship between blood cells count and chemical biomarkers with CAE and its severity in patients undergoing coronary angiography.

Methodology: In this retrospective analytical study, patients who underwent coronary angiography in Dr. Hashmat's Heart Centre, Guilan, Iran from 2015-2017, were studied. The patients were divided in three groups: patients with isolated CAE, atherosclerosis patients, and those with normal coronary arteries. In addition, CAE patients were divided into sub-groups, according to the Mark is classification.

Results: Total 302 patients ware included. Among the studied patients, 84 (27.81%) had isolated CAE, 84 (27.81%) had atherosclerosis, and 134 (44.38%) had normal coronary arteries. No statistically significant differences were observed in the demographic characteristics of the patients among the groups except for age. There were no statistically significant differences in the means of the parameters of CBC test and chemical biomarkers among the groups. No statistically significant differences were observed in the demographic characteristics, the parameters of CBC test, and chemical biomarkers in patients with different types of the CAE.

Conclusion: The findings of the present study indicate that blood cell counts and chemical biomarkers could not be considered as appropriate parameters for assessing the presence and/or severity of isolated CAE.

Key Words: Blood cells count, Chemical biomarkers, Coronary artery ectasia, Markis classification.

INTRODUCTION

Coronary artery ectasia (CAE) is defined as dilated coronary artery segments that are greater than 1.5 times the diameter of adjacent normal segments.[1] About 20 t0 30% of the CAE are congenital and the rest are acquired.[1] Atherosclerosis is known as the main cause of more than 50% of the acquired CAE in adult.[1-5] In addition, the most common cause of CAE in children is Kawasaki disease.[6-8] The CAE can be divided in two groups. One group of CAE are those that occurred following atherosclerosis or cardiac and inflammatory diseases. The other group are those that occurred without any preexisting cardiac, coronary, and/or inflammatory diseases. The former is called isolated CAE which may be seen in about 0.1 to 0.32% of angiographies.[1,5,9,10] Since many of the patients with CAE are asymptomatic, the actual incidence of the disease is not known. However, throughout the world, incidence of CAE is between 0.3 to 4.9% in all conducted angiographies and 0.22 to 1.4% in all conducted autopsies[1,5,7] In Iran, as a developing country, the incidence of CAE is about 12.7% which is significantly higher than the average incidence in the world.[11]

Although the exact mechanism of the coronary artery dilation in CAE is unclear, there are some histopathologic similarity with atherosclerosis. The pathologic characteristics of CAE, including the lipid accumulation with foam-like cells, fibrous caps, and functional loss of the musculoelastic components of the coronary artery media are considered as the main findings in the pathogenesis of CAE.[12,13] Other mechanisms that are reported in the literatures are the increased destruction of proteins such as proteoglycans, fibronectin, and types III, IV, V, and IX collagen followed by the increased activity of metalloproteinase (MMP-3), the inflammation of the arteries with high levels of CRP, interlucin-6, vascular cell adhesion molecule (V-CAM), intracellular adhesion molecule (I-CAM), vascular endothelial growth factor (VEGF), and E-selectin.[14,18]

The clinical significance of CAE was studied by Markis et al. who found that the mortality rate in CAE patients was 15%, approximately equal to that of atherosclerosis.[6] In CAE patients, the most common symptom is angina and other signs such as ST elevation MI, non-ST elevation MI, heart failure, and arrhythmia are also reported.[19] The clinical significance of CAE is not well understood. However, it has been observed that CAE has not a good prognosis and may results in other heart diseases with life-threatening risk.[20]

Inflammation is one of the pathologic causes of CAE. Some studies reported the relationship between CAE and Red cell distribution width (RDW), serum bilirubin, neutrophil to lymphocyte ratio (NLR), Mean platelet volume (MPV).[21-26] In contrast, some studies did not find significant associations between these markers and isolated CAE.[27] Therefore, the study of possible relationship between CAE and the inflammatory markers in the blood may be useful for the prediction of the disease and its severity. Since the clinical signs of the heart diseases are similar, the identification of biomarkers that can differentiate isolated CAE from coronary artery disease is of importance.[9]

The present study was performed to investigate the relationship, if any, between blood cells count and chemical biomarkers with CAE and its severity in patients with coronary angiography.

METHODOLOGY

In this retrospective analytical study, patients who underwent coronary angiography , were studied during 2015-2017 in Dr. Heshmat's heart center, Guilan, Iran. The required data were derived from the patients' medical records. Those patients with ischemic heart disease that based on typical chest pain or the invasive tests such as treadmill test or perfusion scan had been candidates for angiography were included in the study. Before angiography, all patients underwent transthoracic echocardiography and its Ejection Fraction (EF) and regional wall abnormality were obtained. Smoking habits, medical history such as diabetes, hypertension, and hyperlipidemia, as well as family history of heart diseases were extracted. Patients with a SBP of higher than 140 mmHg and/or DBP of higher than 90 mmHg were considered as hypertension patients. Those with fast blood sugar (FBS) of more than 126 gm/dl or those who had been received the glucose lowering drugs were considered diabetic.

Patients with kidney and liver insufficiency, pulmonary diseases, hypertension, hypercholesterolemia, anemia, thalassemia, sleep obstructive apneas, preexisting hematologic diseases, thyroid disorders, heart valve diseases, ventricular systolic dysfunction (EF<50%), pregnancy, recent infections and inflammatory disorders, and body temperature of more than 38°C, as well as those with concurrent CAE and atherosclerosis were excluded from the study.

Before commencement of the study, an informed consent was signed by the patients or their families.

The angiography had been performed via the femoral artery without the use of nitroglycerin, adenosine, and/or calcium channel blockers. The recorded angiograms were interpreted by two experienced cardiologist who were unaware of the patients' clinical characteristics and biochemical results. Consequently, the patients were divided in three groups. Group I, the patients with isolated CAE, Group II, those with coronary artery stenosis (more than 50% stenosis of the artery), and group III, those with normal coronary arteries. In addition, CAE patients were divided into sub-groups, according to the Mark is et al., as follow[2]:

Type 1: Diffuse ectasia of two or three vessels

Type 2: Diffuse ectasia in one vessel and localized disease in another

Type 3: Diffuse ectasia in one vessel only

Type 4: Localized or segmental involvement

The systolic and diastolic blood pressures (SBP and DBP), the complete blood count (CBC), and biochemical tests, including, EF, eosinophil MVP, bilirubin, triglyceride (TG), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), FBS, platelet distribution width (PDW), and RDW that had been performed on the day of angiography were extracted from the patients' medical records.

The protocol of the study had been approved by the Ethical Committee of Guilan University of Medical Sciences (Approval No. IR.GUMS.REC.1396.108). The design and objectives of the study were explained to all participants and written informed

consent was obtained from the participants. The data was kept confidential and anonymous in all phases of the study.

Data were analyzed using version 21.0 of SPSS software (SPSS Inc., Chicago, IL, USA). Descriptive results are presented as the mean ± standard deviation (SD). The Kolmogorov-Smirnov goodness of- fit test was used to assess the normality of the distribution of the variables. Variables that had not a normal distribution are presented as median and range. Chi-square or Pearson's correlation coefficient was used for the comparison of quantitative and qualitative variables between the groups. To compare the means of CBC test and biochemical markers among the studied groups, the analysis of variance (ANOVA) test and non-parametric Kruskal-Wallis test was used were applicable. The linear regression model was used to adjust the effects of confounding variables in the comparison of the levels of RDW and PDW in the studied groups. In all statistical tests, a p-value of less than 0.05 was considered significant.

RESULTS

The demographic characteristics of the studied patients. Of the 302 studied patients, 146 (48%) were women and 156 (52%) were men as shown in table 1. The mean age and BMI of the patients were 58.59±11.611 years and 27.98±4.49 kg/m^2, respectively. Only 45 patients (14.9%) were smoker and the rest did not smoke. Of the 302 patients, 84 (27.81%) had the isolated CAE (group I), 84 (27.81%) had atherosclerosis (group II), and 134 (44.38%) had normal coronary arteries (group III). No statistically significant differences were observed in the demographic characteristics of the patients among the groups, but age. The mean age of the patients in group I was significantly higher than group II (p=0.035).

Table 1: Demographic Characteristics of the Studied Patients

Variables	Group I (n=84)	Group II (n=84)	Group III (n=134)	P-value
Age (year)	58.42±12.13	60.25±11.34*	56.10±11.33	0.035
BMI (kg/m^2)	28.51±5.01	27.56±4.30	28.13±4.24	0.30
Gender				
Male	37 (44%)	84 (62.7%) **	25 (41.7%)	0.032
Female	47 (56%)	50 (37.3%)	49 (58.3%)	
Smoking				
Yes	17 (20.23%)	20 (23.80%)	8 (5.97%) ***	0.18
No	67 (79.77%)	64 (76.20%)	126 (94.03)	
SBP (median, range)	120, 90-185	120, 90-185	120, 80-160	0.058
DBP (median, range)	71, 60-120	75, 20-120	70, 60-100	0.14

BMI: body mass index; SBP: systolic blood pressure; DSP: diastolic blood pressure, * Significantly different from group III
** Significantly different from groups II and III, *** significantly different from groups I and II

The CBC test and chemical biomarkers in the studied groups as shown in table 2. The patients in group III, had lower levels of RBC, Hb, WBC, eosinophil, MPV, and PDW and higher levels of uric acid, LDL, and cholesterol than the other groups. However, the differences did not significant. There were no statistically significant differences in the means of the parameters of CBC test and chemical biomarkers among the groups.

Demoghrafic characteristics of the studied patients based on the types of CAE is indicated in table 3. Of the 84 CAE patients, 29 (34.52%), 15(17.86%), 16 (19.04%), and 24 (25.58%) patients had the type 1, type 2, type 3, and type 4 CAE, respectively. Type 4 patients were younger and had the higher BEI than the other patients. However, the differences were not statistically significant. No statistically significant differences were observed in the demographic characteristics of the patients with different types of the CAE.

The CBC test and chemical biomarkers in different CAE types as shown in table 4. As seen, there was no statistically significant difference in the parameters of CBC test and chemical biomarkers between the patients with different types of CAE.

Table 2: The Complete Blood Count Test and Chemical Biomarkers in the Studied Patients

Variables	Group I (n=84)	Group II (n=84)	Group III (n=134)	p-value
EF (median, range)	50, 45-60	50, 40-60	50, 45-60	0.000
RBC (mean±SD)	4.78±0.64	4.70±0.59	4.617±0.55	0.19
Hb (mean±SD)	13.11±1.59	13.19±1.56	12.83±1.93	0.29
MCV (median, range)	86.88, 82.32-98.10	87.44, 82.28-100.47	87.12, 82.0-95.3	0.63
RDW (median, range)	12.9, 12.2-18.0	13.0, 11.3-18.3	12.9, 11.1-17.8	0.52
WBC (median, range)	7900, 4300-14700	8000, 4000-17600	7800, 3200-13500	0.13
Lymphocytes (mean±SD)	34.71±7.97	34.87±7.62	34.89±7.95	0.80
Neutrophils (mean±SD)	58.83±9.67	59.41±8.31	58.66±8.95	0.09
Eosinophil (median, range)	3, 1-25	3, 1-22	2, 1-40	0.44
NLR	1.61,0.66-8.40	1.58, 0.76-4.44	1.62, 0.76-6.07	0.98
MPV (median, range)	10.0, 8.40-13.6	10.0, 8.10-13.6	9.8, 8.8-12.6	0.25
PDW (median, range)	12.9, 9.8-23.2	12.9, 9.5-23.1	12.3, 10-21.5	0.21
FBS (median, range)	113.0, 79-120	100.50, 83-106	104.0, 78-110	0.006
Cholesterol (mean±SD)	150.20±34.86	152.17±43.04	162.33±49.52	0.13
HDL (median, range)	41, 25-197	41.0, 25-80	41, 23-67	0.76
LDL (median, range)	79.20, 30.2-190	86.80, 11.60-216.8	87.2, 23-282	0.35
TG (median, range)	118.5, 49-430	124, 65-534	119, 36-364	0.68
Total bilirubin (median, range)	0.8, 0.5-1.8	0.8, 0.4-1.8	0.8, 0.5-1.6	0.33
Direct bilirubin (median, range)	0.2, 0.1-0.5	0.2, 0.1-0.5	0.2, 0.1-0.4	0.30
Uric acid (mean±SD)	5.11±1.26	5.30±1.21	5.41±1.44	0.12

Table 3: Demoghrafic Characteristics of the Studied Patients Based on the Types of CAE

Variables	Type 1 (n=29)	Type 2 (n=15)	Type 3 (n=16)	Type 4(n=24)	p-value
Age (year)	59.69±9.83	63.27±10.4	59.56±12.31	54.33±14.49	0.13
BMI (kg/m^2)	28.07±5.99	28.15±3.48	27.9±5.75	30.49±4.21	0.27
Gender					
Male	16 (44.4%)	7 (19.4%)	7 (19.4%)	6 (16.7%)	
Female	13 (27.1%)	8 (16.7%)	9 (18.8%)	18 (37.5%)	
Smoking					
Yes	7 (41.2%)	4 (23.5%)	4 (23.5%)	2 (11.8%)	
No	22 (32.8%)	11 (16.4%)	12 (17.9%)	22 (32.8%)	0.18
SBP (median, range)	120, 90-140	100, 90-120	120, 80-130	100, 90-120	0.11
DBP (median, range)	75, 40-90	71, 60-100	70, 60-100	75, 60-90	0.08

* Significantly different from group III, ** Significantly different from groups II and III, *** significantly different from groups I and II

Table 4: The Complete Blood Count Test and Chemical Biomarkers in the Different Types of CAE

Variables	Type 1 (n= 29)	Type 2 (n= 15)	Type 3 (n= 16)	Type 4 (n= 24)	P-value*
RBC (mean±SD)	4.81± 0.62	4.75±0.58	4.86±0.91	4.71±0.51	0.89
Hb (mean±SD)	13.44±1.76	13.18±1.66	12.86±1.6	12.88±1.38	0.53
MCV (mean±SD)	86.81±7.21	86.48±.38	28.53±9.53	84.13±5.72	0.11
RDW (mean±SD)	13.33±1.48	12.85±0.64	13.70±1.76	12.59±1.06	0.22
WBC (mean±SD)	8282±2087	7873±1825	8056±3319	7879±1853	
Lymphocytes (mean±SD)	32.52±7.56	36±5.55	37.44±8.32	35.17±9.17	0.21
Neutrophils (mean±SD)	61.55±10.08	57.8±6.32	55.5±9.54	58.96±9.52	0.20
Eosinophil (median, range)	3, 1-17	2, 2-12	3, 2-20	2.5, 2-15	0.80
NLR	2, 0.9-3.75	1.57, 0.98-2.39	1.5, 0.83-5.4	1.5, 0.66-4.33	0.17
MPV (mean±SD)	10.30±1.13	10.19±0.83	9.97±0.63	10.35±1.21	0.68
PDW (mean±SD)	13.71±3.37	12.87±1.63	13.09±1.80	13.62±2.67	0.71
FBS (mean±SD)	112.41±27.56	106.27±19.91	114.31±26.46	108.25±23.98	0.76
Cholest erol (mean±SD)	150.72±36.03	143.6±27.94	162.06±34.79	149.75±36.84	0.51
HDL (median, range)	43, 25-107	39, 28-68	42, 30-76	39.5, 30-78	0.61
LDL (mean±SD)	80.53±30.54	78.37±24.95	96.93±32.95	82.28±26.01	0.24
TG (mean±SD)	128.21±73.08	127.80±60.53	123.46±49.88	118.21±72.90	0.29
Uric acid (mean±SD)	5.39±1.52	4.93±0.84	4.94±1.23	4.95±0.96	0.45

*No significant difference was observed between the patients with the different types of the CAE

DISCUSSION

In the present study, the relationship between CBC and chemical biomarkers with CAE and its severity in patients with coronary angiography was investigated. The CAE patients divided into three groups of isolated CAE patients (group I), atherosclerotic patients (group II), and patients with normal coronary arteries (group III). In addition, we assessed the CBC and chemical biomarkers in patients with different types of CAE according to Markis et al classification.

Despite advances in cardiology, the pathophysiology of coronary artery ectasia is not well recognized. Although the underlying mechanisms responsible for ectasia formation are not clearly known, previous studies reported that inflammation, neurohormonal process, and cardiovascular risk factors were associated with CAE. On the other hand, its clinical features range from insignificance to cardiac infarction and sudden death. Due to the clinical importance of coronary artery disease, timely diagnosis of disease can be beneficial. In the other hand, coronary artery ectasia which may lead to ischaemic symptoms and findings, could be identified by more sensitive and specific cardiovascular imaging modalities. However, these tools are expensive and time consuming, with potential unwanted effects such as exposure to radiation. By considering these, using non invasive methods can help in the early diagnosis and treatment of the disease. Therefore, using blood cell parameters and chemical biomarkers which is cheap and easily obtained, could be used as an initial criterion, especially in small centers, to determine the need for further imaging modalities in the assessment of CEA.

In our study, in parallel with Demir et al and Balta et al.– studies we found no difference according to BMI, lipid profile, fasting blood sugar and also systolic and diastolic blood pressure.

We found that there was a gender preference in patients in the atherosclerosis group than the other groups and number of men in this group was more than the other group. Kundi et al have reported the same preference in their study investigating the relation between monocyte to high-density lipoprotein cholesterol ratio with presence and severity of isolated coronary artery ectasia.

In the present study, no statistically significant differences were observed in the means of the parameters of CBC test like neutrophile and lymphocyte count, MPV, RDW and PDW, and also chemical biomarkers like uric acid and billirubin levels among the study groups. These findings are in line with those of Demir et al., Balta et al., – and Isik et al. In contrast, Kundi et al. , in a study to assay the relationship between monocyte to high-density lipoprotein cholesterol ratio with presence and severity of isolated CAE found higher levels of blood cells in isolated CAE patients in comparison with control group. In addition, Li et al. demonstrated that patients with isolated CAE had increased lymphocyte, neutrophil and monocyte counts when compared to patients with normal coronary arteries.

Unlike our study, another study by Balta et al.– showed that neutrophil to lymphocyte ratio and MPV were sigificantly higher in patients in both CAE and CAD groups compared to those in the control groups. In the present study, we did not observe a significant relationship in the levels of NLR among the groups. In contrast some studies reported a higher NLR in CAE patients than the control group.– In line with the study of Balta et al., – , we did not found a statistically significant relationship in the levels of NLR between the different types of CAE. These findings were different from Balta et al. and Isik et al which reported rise in NLR in ectasia group compared to control group. Balta et al., – also reported no correlation between the severity of CAE and NLR.

In contrast to our findings, Balta et al – , and Sen et al., found higher levels of MPV in isolated CAE patients than control group. Like our findings, Sarli et al reported no association between coronary artery ectasia and MPV .

In accordance with previous studies we did not found a statistically significant association in the levels of RDW between the studied groups. In another study by Isik et al.[25] RDW was observed to be an independent predictor of both presence and severity of isolated CAE. Li et al.[20] also found no significant difference among control , ectatic and atherosclerotic groups in accordance with RDW measure. We also found no relationship between RDW and severity of isolated ectasia; but Keser et al.[29] reported increased level of RDW in type one coronary artery ectasia.

We found no significant association between PDW levels among the study groups. This is in contrast with Hamur et al. findings which had revealed PDW as an independent predictor of coronary artery ectasia, they also reported significantly higher levels of PDW in isolated CAE patients.

In accordance with previous studies,[21,23,27] we did not found a statistically significant association in the levels of uric acid between the studied groups.

In the present study no significant difference was noted among the study groups concerning bilirubin level. Although, Demir et al., reported significantly lower levels of bilirubin in isolated CAE patients.[23]

The main limitation of our study was the relatively small sample size. We also did not analyze markers of inflammation such as CRP, although the role of inflammation was previously reported in these patients. Finally, intravascular ultrasound (IVUS) provides more precise values about the presence and distribution of atherosclerosis in vessel lumen and throughout the wall. We did not have the opportunity to perform IVUS in this study.

CONCLUSION

The findings of the present study indicate that blood cell counts and chemical biomarkers could not be considered as appropriate parameters for assessing the presence and/or severity of isolated CAE. We believe that further studies with larger sample size are needed to clarify the role of N/L ratio in CAE complicated CAD, especially in relation to angiographic and clinical parameters.

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