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Relationship of Plasma Eosinophil Count with Coronary Artery Disease

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ABSTRACT ***Background:*** Chronic inflammatory disorders that can have systemic effects include allergic asthma and atherosclerosis, a condition in which plaque buildup narrows arteries and increases the risk of coronary artery disease (CAD). Eosinophils (EOS) are inflammatory innate immune cells involved in arterial thrombogenesis. ***Objective:*** To investigate the connection between plasma eosinophil count and coronary artery disease. ***Materials and Methods:*** The case control study was conducted Department of cardiology, TMSS Medical College Hospital, total of 140 patients who underwent coronary angiography were recruited during March 2024 to February 2025. The study case group included 70 patients with isolated CAD and 70 control group consisted of age- and gender-matched subjects with normal coronary angiograms. ***Results:*** Significantly higher were found Creatinin and Total cholesterol 0.75±0.1 and 211±45 in cases in comparison to 0.72±0.2 and 181±36 in controlled respectively ($p < 0.001$). Eosinophils, neutrophils, lymphocytes, NLR and MPV (fL) were significantly investigated ($p<0.05$) comparison with cases and controlled. Gensini scores 32.75 ± 6.94 were in cases and 7.35 ±4.37 were in control ($p < 0.001$). Smoking, ST-segment-elevation myocardial infarction (STEMI), and emergency PCI significantly differed from EOS after ≥0.03×109/L and EOS post <0.03×109/L ($p < 0.05$). Neutrophils, lymphocytes, and NLR were substantially different between EOS post ≥0.03×109/L and EOS post <0.03×109/L ($p < 0.05$). ***Conclusion:*** Patients with coronary artery disease had significantly higher creatinine and cholesterol profiles, as well as a clinical and pathological relationship with decreased Eosinophils (EOS).

Keywords: Eosinophils; ST-Segment Elevation Myocardial Infarction; Outcomes; Coronary Artery Disease.

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INTRODUCTION

Coronary artery disease (CAD), a major contributor to the overall burden of death in the human population, related to pathological process of atherosclerosis, is mainly elucidated as a series of heart disease caused by myocardial ischemia, hypoxia, or necrosis [1]. Constituting 1% to 5% of total peripheral leukocytes, eosinophils are multifunctional granulocytes that are normally involved in allergen-induced allergy or hyperresponsiveness.[2] Accumulating evidence suggests

eosinophil participation in cardiovascular diseases. Eosinophils activate platelets and contribute to stent thrombosis following percutaneous coronary intervention (PCI) or during atherogenesis [2]. Eosinophils are pleiotropic multifunctional leukocytes implicated in the pathogenesis of numerous inflammatory processes and are one of the most important effector cells in allergic asthma progression.[3] So, due to the possible connection between allergic asthma and atherosclerosis, it is important to know whether eosinophils also promote the development of atherosclerosis and, in turn, CAD [3]. However, there are still some studies showing high eosinophil levels are not independently associated with the prevalence and severity of CAD [4]. Additionally, eosinophil cationic protein (ECP) levels, the biomarker of eosinophil activation, are significantly higher in acute coronary syndrome cases and can independently predict CAD severity [5, 6]. ECPs are also associated with atherosclerotic burden and poor prognoses in patients undergoing stent implantation [7]. Therefore, considering these conflicting studies, the direct relationship between eosinophils and CAD remains uncertain [8]. In this present study, we aimed to evaluate the relationship between the amount of eosinophils in peripheral circulation and various CAD subtypes and severities, and the ability of eosinophils to predict severe CAD and acute coronary arterial thrombotic event.

MATERIALS AND METHODS

The case control study was conducted Department of cardiology, TMSS Medical College Hospital, total of 140 patients who underwent coronary angiography were recruited during March 2024 to February 2025. The study case group included 70 patients with isolated CAD without any stenotic lesions under visual assessment. The 70-control group consisted of age- and gender-matched subjects with normal coronary angiograms. In both groups, the indication for coronary angiography was the presence of typical angina or positive or equivocal results for myocardial ischemia on noninvasive screening tests. Their biochemical parameters, including eosinophil count, were measured and their correlation with the severity of coronary artery stenosis, as quantified by the Gensini score system was evaluated. Patients with allergic asthma, autoimmune diseases, a history of allergic diseases, parasitic infections, current infections, malignancies, severe liver or kidney dysfunction, heart failure or shock, rheumatic heart diseases, and valvular heart diseases were excluded in this study. Patients who underwent coronary artery bypass surgery were also not included in this study considering graft complexity. All study participants provided written informed consent. Patients were first categorised by whether they had CAD. CAD was defined as having>50% stenosis in ≥1 major coronary artery. According to the receiver operating characteristic analyses, the best cutoffs of Low EOS post (<0.03×109/L) predicted the 30-day all-cause and cardiac. Patients with combined low EOS post (<0.03×109/L) and (≥0.03×109/L) displayed synergistic risk of 30-day all-cause and cardiac. Continuous variables were expressed as mean±SD (normal distribution) or median and interquartile range (abnormal distribution). The student *t* test was used to determine the differences among groups. Categorical variables were presented as number and percentage. The differences were compared by $\chi 2$ test or Fisher exact test.

RESULTS

Table 1: Comparison of Basic Clinical and Biochemical Features of Patients and Controls

Variables	Study group		
	Case n=70	Control n=70	p value
Age (years)	60.26 ± 10.6	57.86 ± 11.6	0.20
Sex (n.%) males	32 (45.71%)	23 (32.85%)	0.12
Body mass index (BMI) (kg/m2)	29.8 ± 5.4	28.5 ± 4.6	0.12
Smoking	9 (18%)	6 (20%)	0.76
Fasting glucose (mg/dL)	95.7 ± 9	97.6 ± 8.5	0.201
Creatinin (mg/dL)	0.75 ± 0.1	0.72 ± 0.2	<0.001
Total cholesterol (mg/dL)	211 ± 45	181 ± 36	<0.001
Trigliserid (mg/dL)	162.5 ± 65	151.9 ± 41	0.25

TSH (μIU/mL)	1.7 ± 0.6	1.6 ± 0.4	0.24

The basic clinical and demographic characteristics were compared, and there were no statistically significant between-group differences in terms of age, gender distribution, body mass index, smoking status, Fasting glucose, Trigliserid and TSH. Significantly higher were found Creatinin and Total cholesterol 0.75 ± 0.1 and 211 ± 45 in cases in comparison to 0.72 ± 0.2 and 181 ± 36 in controlled respectively (p <0.001).

Table 2: Comparison of Whole Blood Count Features of Patients and Controls

Variables	Study group		
	Case n=70 Mean ±SD	Control n=70 Mean ±SD	p value
Eosinophil	0.19 (±0.15)	0.12 (±0.07)	**0.005**
Neutrophil	6.17 (±3.19)	3.96 (±1.79)	**<0.001**
Lymphocyte	2.29 (±1.36)	4.67 (±3.18)	**<0.001**
NLR	2.69 (±2.47)	1.17 (±1.77)	**<0.001**
MPV (fL)	8.71 (±1.06)	6.34 (±1.57)	**<0.001**
Leukocyte (103/μL)	9.27 (±2.64)	8.61 (±2.13)	**0.10**
Hemoglobin (g/dL)	13.76 (±1.57)	13.46(±1.24)	**0.21**
Hematocrit (%)	41.53 (±3.47)	40.91 (±3.71)	**0.30**
Gensini scores	32.75 ± 6.94	7.35 ±4.37	**<0.001**

Eosinophils were 0.19±0.15 vs. 0.12 ±0.07, neutrophils were 6.17 ±3.19 vs. 3.96 ±1.79, lymphocytes were 2.29 ±1.36 vs. 4.67 ±3.18, NLR was 2.69 ±2.47 vs. 1.17 ±1.77, and MPV (fL) was 8.71 ±1.06 vs. 6.34 ±1.57, all of which were significantly investigated (p<0.05) comparison with cases and controlled. Gensini scores 32.75 ± 6.94 were in cases and 7.35 ±4.37 were in control (p <0.001).

Table 3: Baseline characteristics and comorbidities among the groups divided by EOS post Levels in cases (n=70)

Characteristic	Study group		p value
	EOS post ≥0.03×109/L n=53	EOS post <0.03×109/L n=17	
Smoking	31 (58.49)	5 (29.41)	0.03
Diabetes	17 (32.08)	5 (29.41)	0.83
Hypertension	34 (64.15)	10 (58.82)	0.69
Atrial fibrillation	3 (5.66)	1 (5.88)	0.97
Emergency PCI	19 (35.85)	11 (64.70)	0.03
STEMI	18 (33.96)	11 (64.70)	0.02
Chronic kidney disease	7 (13.21)	2 (11.76)	0.87
Hyperlipemia	11 (20.75)	3 (17.65)	0.78

Smoking and ST-segment-elevation myocardial infarction (STEMI) and emergency PCI were substantially different from EOS post ≥0.03×109/L and EOS post <0.03×109/L (p <0.05).

Table 4: Comparison of Whole Blood Count Features divided by EOS post Levels in cases (n=70)

Investigation	Study group		p value
	EOS post ≥0.03×109/L n=53	EOS post <0.03×109/L n=17	
Neutrophil	6.17 (±3.29)	7.86 (±1.59)	0.04
Lymphocyte	2.87 (±1.26)	2.13 (±1.10)	0.03
NLR	2.14 (±2.25)	3.69 (±1.44)	0.009
MPV (fL)	8.27 (±1.16)	7.94 (±1.37)	0.33
Leukocyte (103/µL)	9.23 (±2.15)	9.62 (±2.11)	0.50
Hemoglobin (g/dL)	13.66 (±1.52)	13.56(±1.21)	0.80
Hematocrit (%)	41.73 (±3.24)	40.54 (±3.73)	0.20
Creatinin (mg/dL)	0.79 ± 0.12	0.73 ± 0.23	0.16
Total cholesterol (mg/dL)	237 ± 45.7	247 ± 46.91	0.43
Trigliserid (mg/dL)	165.45 ± 66.7	157.19 ± 55.7	0.64
Gensini scores	36.47 ± 7.81	32.15 ± 6.94	0.53

Neutrophil and Lymphocyte and NLR were significantly different from EOS post ≥0.03×109/L and EOS post <0.03×109/L (p <0.05).

DISCUSSION

In this study observed that the basic clinical and demographic characteristics were compared, and there were no statistically significant between-group differences in terms of age, gender distribution, body mass index, smoking status, fasting glucose, trigliserid and TSH. Significantly higher were found creatinin and total cholesterol 0.75 ± 0.1 and 211 ± 45 in cases in comparison to 0.72 ± 0.2 and 181 ± 36 in controlled respectively (p <0.001). The Demir *et al*, study revealed (19 males, mean age 65.6±13.7 years) with cases [9]. The control group consisted of 30 age- and gender-matched subjects (10 males, mean age 49.16±9.2 years) with normal coronary angiograms. Clinical and demographic characteristics were compared, and there were no statistically significant between-group differences in terms of age, gender distribution, body mass index, smoking status, or biochemical parameters. Previous studies have demonstrated that the neutrophil lymphocyte ratio (NLR) are greater in coronary artery disease patients than in healthy controls. The NLR increases may suggest that these markers can be used in clinical practice to assess the inflammatory statuses of coronary artery disease patients [10-12].

In this study observed eosinophils were 0.19±0.15 vs. 0.12 ±0.07, neutrophils were 6.17 ±3.19 vs. 3.96 ±1.79, lymphocytes were 2.29 ±1.36 vs. 4.67 ±3.18, NLR was 2.69 ±2.47 vs. 1.17 ±1.77, and MPV (fL) was 8.71 ±1.06 vs. 6.34 ±1.57, all of which were significantly investigated (p<0.05) comparison with cases and controlled. Gensini scores 32.75 ± 6.94 were in cases and 7.35 ±4.37 were in control (p <0.001). Similar observation was found Gao *et al*, they also observed Eosinophil, Neutrophil, Lymphocyte, NLR and MPV (fL) also significant difference in CAD group in comparison with non-CAD. Gao *et al*, reported compared to non-CAD patients, the mean Gensini score was significantly higher (4.35 ± 3.31 *vs.* 39.39 ± 31.95, $p < 0.001$) in CAD patients [3]. Eosinophils contain several granule-associated molecules that play a role in the occurrence of thrombosis and vascular injury. Eosinophils can increase the risk of thrombosis through leukocyte and platelet stimulation and the release of tissue factor [13, 14]. In Demir *et al*, study we have found significantly differences in MPV and NLR between CAE patients and control group [9]. Also, our findings are consistent with previous studies [15, 16]. Additionally, when 2 groups were compared in our study, eosinophil count of the patients having CAE were significantly higher than control groups. Demir et al, reported that the given blood count parameters, in the group of CAE patients' blood eosinophil count, neutrophil lymphocyte ratio (NLR) and MPV value, were significantly higher in comparison with the control group [9]. There was no statistically significant difference between two groups with regard to leukocyte count, platelet count, hemoglobin and hematochrit level.

Present study showed smoking and ST-segment-

elevation myocardial infarction (STEMI) were substantially different from EOS post ≥0.03×109/L and EOS post <0.03×109/L (p <0.05). Similar observation was found Gao *et al*, study [3]. More had tended to have higher blood neutrophils, cardiac troponin I, and creatine kinase MB, but lower systolic blood pressure, blood lymphocyte and monocyte content, and serum creatinine than those with EOSpost≥0.03×109/L. They found no differences in Thrombolysis in Myocardial Infarction flow grades between EOSpost≥0.03×109/L and EOSpost<0.03×109/L before (*P*=0.51) or after the procedure (*P*=0.08) or the Gensini scores (*P*=0.77) and history of allergy (*P*=1.00). The results were same between EOSpost≥0.14×109/L and EOS post<0.14×109/L before (*P*=0.34) and after the procedure (*P*=0.20) or the Gensini scores (*P*=0.84) and history of allergy (*P*=0.70).

Present study was found neutrophil and Lymphocyte and NLR were significantly different from EOS post ≥0.03×109/L and EOS post <0.03×109/L (p <0.05). These findings suggest that eosinophil granule proteins are involved in vascular injury and that eosinophils may affect the cardiovascular system by inducing inflammatory cell infiltration [17, 18]. Few studies have reported that eosinophils are associated with arterial tortuosity, dilatation, thrombosis, cardiac syndrome and non-dipper hypertension [19,20]. Major basic protein, eosinophilic cationic protein, and eosinophil-derived neurotoxin are the primary mediators of eosinophil-associated toxicity to human tissue and may induce eosinophilic myocarditis, pneumonitis, dermatitis, neuropathy, and vasculitis.

CONCLUSION

Patients with coronary artery disease have elevated eosinophil counts, which are strongly and independently linked to the condition. The etiopathogenesis of coronary artery disease and the pathophysiological reasons behind the elevated risk of cardiovascular morbidity and mortality in these people may be better understood as a result of our findings. The thrombosis, endothelial dysfunction, and vascular damage seen in patients with coronary artery disease may be related to the elevated eosinophil concentration. Smoking, ST-segment-elevation myocardial infarction (STEMI), and emergency PCI were all significantly correlated with low EOS counts. As a useful indicator, EOS counts may be able to assist doctors in identifying patients who are at high risk.

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