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Original Article

Neutrophil-to-Lymphocyte Ratio: Is it Higher in Bronchiectasis than in COPD?

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ABSTRACT

Objective: There are similarities in the symptoms and attacks of bronchiectasis and chronic obstructive pulmonary disease (COPD). Chronic systemic inflammation in COPD and destructive pulmonary inflammation in bronchiectasis lead to increases in inflammatory markers. The presence of peripheral blood eosinophil (PBE)(>2%) and the neutrophil-to-lymphocyte ratio (NLR) affect the type of attack and the treatment approach. The aim of this study was to investigate differences in inflammatory markers and the presence of PBE in COPD and bronchiectasis.

Methods: A retrospective, cross-sectional study of patients diagnosed in 2014 with bronchiectasis (J47.0-ICD) or COPD (J44.0-J44.9-ICD) at the inpatient or outpatient clinics of a chest disease hospital was performed. Patients with cancer, hematological disease, renal disease, or COPD with bronchiectasis were excluded. Demographic details and the C-reactive protein (CRP) levels were recorded, and the NLR was calculated. The study groups were then sub-classified according to PBE.

Results: In all, 2664 patients (outpatient bronchiectasis: n=1024, outpatient COPD: 775; inpatient bronchiectasis: n=180, inpatient COPD n=685) were included. The NLR was significantly lower in the bronchiectasis group. The median CRP level was lower in those with bronchiectasis than in the COPD inpatient group. The PBE values in the bronchiectasis and COPD inpatient groups was 24% and 40%, respectively and 18% and 40% in outpatient groups, respectively. The NLR in patients with PBE was lower than that observed in non-eosinophilic patients.

Conclusion: Inflammatory markers were lower in patients with bronchiectasis than in those with COPD. Continuous anti-inflammatory treatment may be recommended for patients with COPD. NLR and PBE may be able to indicate the nature of advisable treatment in further studies.

INTRODUCTION

Chronic obstructive pulmonary diseases, which are generally defined as emphysema and bronchitis, are grouped into 5 categories: chronic obstructive pulmonary disease (COPD), asthma, bronchiectasis, cystic fibrosis, and bronchiolitis obliterans.[1] In general, the symptoms of these diseases are similar; however, the underlying mechanisms are different. COPD can be described as a small airway disease in adults with exposure to harmful dust, gas, or fumes, while asthma can be defined as a major airway disease associated with earlier exposure to an allergen.[2] Although the frequency of COPD and asthma is high in the community, bronchiectasis with a structural disorder in the bronchi due to untreated infection during lung development is less common. The more rarely observed cystic fibrosis, defined as a genetic disorder, also demonstrates a radiological similarity to bronchiectasis with structural lung injury.[3] Bronchiectasis, a systemic inflammatory disease that is more common in developing countries due to inadequate treatment and pulmonary damage, is very similar to COPD regarding symptoms and attacks observed. Structural damage to the

lungs is widespread in bronchiectasis, and mortality rate increases during exacerbation when appropriate antibiotics are not provided.[4] A bronchiectasis attack is a consequence of pulmonary infection, while infectious attacks are observed in two-thirds of COPD patients.[2] Diffuse pulmonary injury is less common in COPD.

There is limited data comparing inflammatory markers in COPD and bronchiectasis. A few studies have been conducted at our center to investigate the utility of neutrophil-to-lymphocyte ratio (NLR) to evaluate the severity and diagnosis of acute exacerbation of COPD with a limited number of patients.[5,6] This retrospective study was planned to investigate the hypothesis that the inflammatory marker of NLR would be higher in cases of bronchiectasis than exacerbated COPD, due to pulmonary parenchymal damage.

MATERIAL AND METHODS

The study was approved by by the local ethics committee (12.10.2016/119) and was conducted in accordance with the Declaration of Helsinki. Since the study was retrospective, written consent was not obtained from the patients.

Patients

Patients who had previously been diagnosed with bronchiectasis or COPD and followed up in the outpatient clinic were evaluated. Records of a diagnosis of bronchiectasis were obtained from the hospital database electronically using the J47.0 ICD code, and the J44 (J44.0-J44.9) ICD code was used for a diagnosis of COPD. The patient inclusion and exclusion criteria are summarized in Figure 1.

Bronchiectasis and COPD patients with hemogram values at the time of admission were included in the study. The hemogram, NLR, C-reactive protein (CRP), and platelet/mean platelet volume (PLT/MPV) values of outpatient clinic and hospitalized patients with COPD and bronchiectasis were compared.

Definitions

COPD: A COPD diagnosis was made according to the guidelines of the Global Initiative for Chronic Obstructive Lung Disease, the American Thoracic Society and Turkish Thoracic Society. The COPD diagnosis was established by a pulmonologist who evaluated airflow obstruction on spirometry (i.e., forced expiratory volume in one second [FEV_1] ≤70% predicted and FEV_1 and forced vital capacity [FVC] ratio ≤70%) in patients with a compatible history for COPD.[2,7,8] However, since no spirometry values were found in the system, the related data could not be recorded in this study.

Bronchiectasis: The diagnosis of bronchiectasis was made by a radiologist and pulmonary specialists using the findings of computerized tomography (CT) and high resolution CT (HRCT) to evaluate the presence of an internal bronchial diameter wider than the adjacent pulmonary artery; no thinning in the bronchi (based on a bronchus with the same diameter at a distance of 2 cm or more from the separated bronchus); costal pleura within 1 cm of the mediastinal pleura; bronchial thickening and thickening of the bronchial wall; assessment of tubular, varicose, and cystic bronchial dilatations; and the clinical diagnosis of chest disease specialists. Bronchiectasis types were not recorded in the study due to the absence of CT/HRCT images in the hospital electronic system.

NLR: The ratio was obtained by dividing the absolute number of neutrophils by the absolute number of lymphocytes. The data were expressed as a percentage value (i.e.: 2.25%).

PLT/MPV: The platelet to mean platelet volume ratio was calculated and written as an absolute value.

Neutrophilic exacerbation of COPD/bronchiectasis: This condition was defined as the presence of less than 2% peripheral blood eosinophils.

Eosinophilic exacerbation of COPD/bronchiectasis: This was defined as a percentage of peripheral blood eosinophils of more than 2%.

Recorded data

The hemogram, serum biochemistry, and CRP values at admission to the hospital were recorded from the information in the hospital's electronic database. Due to the lack of a procalcitonin test in the hospital during the study period, no procalcitonin values were recorded.

Statistical analysis

In this retrospective cross-sectional study, the demographics of patients with bronchiectasis and COPD were summarized with descriptive statistics. IBM SPSS Statistics for Windows, Version 20.0 (IBM Corp., Armonk, NY, USA) was used for the statistical analysis. Comparisons were made using the Mann-Whitney U Test (nonparametric) if the population showed uniform distribution of continuous numerical data (i.e., age, hemogram, serum biochemistry, CRP, NLR values), and when the values were parametric, Student's t-test was used. The groups were analyzed using a chi-square test for dichotomized values of gender, additional disease presence, CRP, NLR, and eosinophil groups. A p value <0.05 was considered significant.

RESULTS

The selection process used for bronchiectasis and COPD patients admitted to the ward and to the outpatient clinic during the study period is illustrated in Figure 1. Table 1

summarizes the demographic characteristics of the 1204 patients with bronchiectasis who were evaluated in the outpatient clinic (n=1024) and those who were hospitalized (n=180), as well as the 1460 patients with COPD in the 2 groups (n=685 and n=775, respectively). In the bronchiectasis group, there were significantly more female patients and they were younger than the COPD patients. The comparison of outpatient clinic and inpatient groups revealed that the hemogram values were statistically significantly different, but not clinically significant.

Table 2 demonstrates the comparison of inflammatory markers in bronchiectasis and COPD patients according to outpatient and hospital admission status. The inflammatory markers (NLR, PLT/MPV, CRP, sedimentation) of the 2 groups were compared. The NLR and CRP values were found to be clinically and statistically significant in the COPD group that was hospitalized. The sedimentation rates and PLT/MPV values were similar in both groups. In the outpatient group, the NLR values in COPD patients were high, and the PLT/MPV values were low, with clinical and statistical significance.

Table 3 shows the NLR, CRP, and sedimentation values according to eosinophilic or noneosinophilic status in the COPD and bronchiectasis patients. The inflammatory markers were statistically higher in the noneosinophilic groups of COPD and bronchiectasis patients.

DISCUSSION

The results of this study indicated that the NLR value, which was used as a new inflammatory marker, was significantly higher in hospitalized patients with bronchiectasis and COPD compared with those who were not hospitalized. In contrast to our hypothesis regarding acute ex-

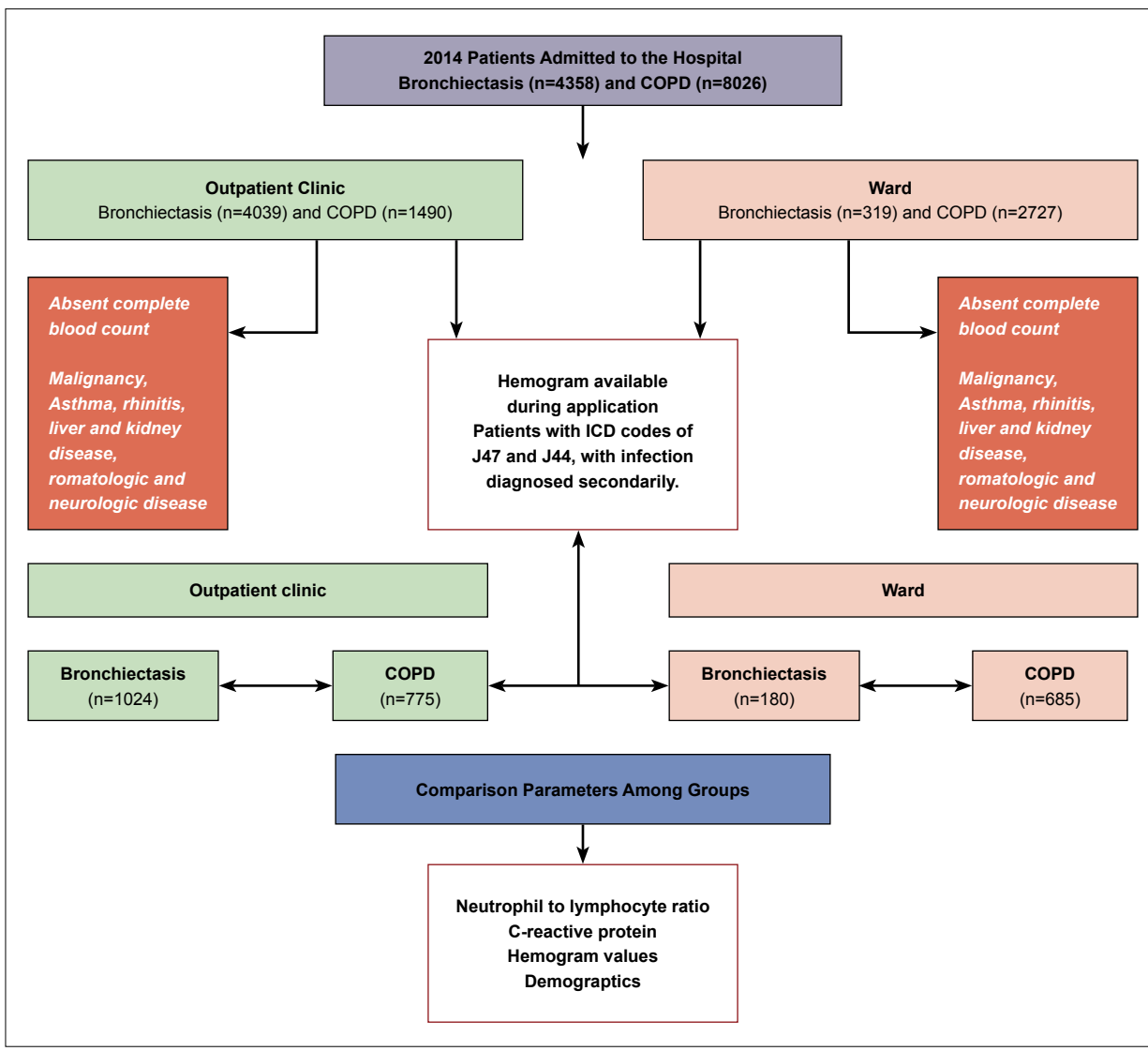

Figure 1. Flow chart of patient enrollment.

Table 1. Comparison of demographic details and hemogram values of the study groups

	Ward			Outpatient clinic		
	Bronchiectasis (n=180) Median*	COPD (n=685) Median*	p	Bronchiectasis (n=1024) Median*	COPD (n=775) Median*	P
Age, mean±SD (years)	43±16	69±10	0.001	45±15	67±11	0.001
Female, n (%)	90 (50)	195 (29)	0.001	570 (56)	262 (34)	0.001
Complete blood count						
Leukocyte (×10^9/L)	11.3 (7.4–14.2)	10.7 (8.1–13.9)	0.98	8.7 (6.9–11.5)	9.7 (7.6–12.3)	0.001
Neutrophil (×10^9/L)	8.13 (5.40–12.36)	8.41 (6.01–11.53)	0.47	5.68 (4.12–8.18)	6.67 (4.97–9.18)	0.001
Lymphocyte (×10^9/L)	1.40 (0.90–2.01)	1.14 (0.73–1.71)	0.001	1.96 (1.48–2.49)	1.58 (1.08–2.17)	0.001
Monocyte (×10^9/L)	0.63 (0.47–0.90)	0.58 (0.36–0.87)	0.025	0.58 (0.43–0.80)	0.67 (0.48–0.95)	0.001
Neutrophil, (%)	77 (68–86)	80 (72–89)	0.004	66 (58–75)	71 (62–79)	0.001
Monocyte (%)	6.4 (4.5–8.2)	5.7 (3.6–7.9)	0.019	6.6 (5.2–8.3)	7.2 (5.4–9.4)	0.001
Eosinophil (%)	0.8 (0.2–1.9)	0.4 (0.1–1.5)	0.020	1.7 (0.9–2.6)	1.5 (0.8–2.7)	0.14
Basophil %	0.3 (0.1–0.5)	0.4 (0.1–0.7)	0.006	0.4 (0.2–0.7)	0.6 (0.3–1.1)	0.001
Lymphocyte (%)	14 (8–22)	11 (7–18)	0.005	23 (15–31)	17 (11–23)	0.001
Erythrocyte (×10^{12}/L)	4.2 (3.8–4.7)	4.4 (3.9–4.8)	0.011	4.6 (4.2–4.9)	4.6 (4.3–5.0)	0.16
Hemoglobin (g/dL)	11.8 (10.4–13.1)	12.5 (11.3–13.7)	0.001	12.9 (11.7–14.6)	13.2 (11.9–14.6)	0.001
Hematocrit (%)	35 (31–40)	38 (34–41)	0.001	39 (35–42)	40 (36–43)	0.001
MCV (fL)	84 (80–88)	86 (82–90)	0.001	85 (81–89)	86 (82–90)	0.001
Platelet (×10^9/L)	265 (212–339)	259 (210–332)	0.83	280 (229–343)	248 (2001–313)	0.001
MPV	8.3 (7.8–9.2)	8.4 (7.8–9.1)	0.69	8.3 (7.7–9.0)	8.2 (7.6–8.9)	0.07

COPD: Chronic obstructive pulmonary disease; MCV: Mean corpuscular volume; MPV: Mean platelet volume; SD: Standard deviation.
Median* : Median (interquartile range).

Table 2. Comparison of inflammatory markers in the study groups

	Ward			Outpatient clinic		
	Bronchiectasis (n=180) Median*	COPD (n=685) Median*	p	Bronchiectasis (n=1024) Median*	COPD (n=775) Median*	P
NLR	5.84 (3.0–10.96)	7.39 (4.04–12.97)	0.006	2.84 (1.91–4.79)	4.30 (2.71–7.03)	0.001
PLT/MPV	31 (24–42)	31 (24–40)	0.79	33 (27–43)	30 (24–39)	0.001
CRP (mg/L)	18.6 (8.5–84)	45.0 (14.2–121.0)	0.001	12.3 (3.3–36.1)	12.8 (3.3–32.2)	0.91
ESR (mm/h)	62 (28–87)	49 (27–70)	0.33	36 (18–65)	54 (29–61)	0.36

COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; NLR: Neutrophil-to-lymphocyte ratio; PLT/MPV: Platelet/mean platelet volume. Median* : Median (interquartile range).

acerbation of bronchiectasis with extensive pulmonary parenchymal damage, the NLR was found to be higher in COPD patients.

The similarities between COPD and bronchiectasis symptoms have been investigated in a few studies, and among the patients with moderate and severe COPD, bronchiectasis was detected in 28% to 57%.[9–11] Exacerbation caused by infection was observed in patients with bronchiectasis and in nearly 40% of COPD patients. In a recent study performed by Novosad et al.,[12] it was suggested that COPD is a risk factor for bronchiectasis.

Potential pathogenic microorganisms, which play an important role in the pathogenesis of bronchiectasis, have also been identified in severe COPD. In these studies, it has been pointed out that long-term and severe exacerbation of the bronchial mucosa may lead to an aggravated clinical presentation and increase mucosal damage due to bronchiectasis.[12]

Table 3. Results of a hemogram in the study groups according to eosinophil values

	Bronhiectasis					Chronic obstructive pulmonary disease				
	Outpatients' clinic (n=1024)		Wards (n=180)		p	Outpatients' clinic (n=775)		Wards (n=685)		p
Eosinophil (%)	<%2	>=%2	<%2	>=%2		<%2	>=%2	<%2	>=%2	
NLR	3.8 (2.3–6.6)	2.1 (1.6–2.9)	7.5 (4.7–13.8)	2.9 (1.9–4.19)	<0.001	5.5 (3.4–9.0)	3.0 (2.1–4.4)	8.6 (4.9–15.3)	3.3 (2.6–4.5)	<0.001
CRP (mg/L)	18.1 (4.2–66.3)	6.1 (3.3–20.1)	27.6 (10.2–88.9)	8.8 (3.3–79.9)	0.001	16 (6.3–44.4)	7.8 (3.3–30.9)	53.5 (15.8–128)	22.0 (6.4–67.9)	<0.001
ESR (mm/h)	39 (18–70)	34 (18–57)	69 (30–107)	33 (8–63)	0.031	55 (29–61)	42 (28–69)	50 (27–70)	44 (30–63)	0.83

NLR, CRP, ESR: Median and interquartile range (25th-75th percentile). CRP: C-reactive protein; ESR: Erythrocyte sedimentation rate; NLR: Neutrophil-to-lymphocyte ratio.

In the present study, cases of COPD with bronchiectasis were excluded in order to analyze the difference in inflammatory markers in COPD and bronchiectasis separately. The demographic characteristics of the 2 groups were significantly different in the present study. In our study, two-thirds of the COPD patients were male, while the rate of female patients was twice that of males in those with bronchiectasis. Furthermore, bronchiectasis patients were nearly 2 decades younger than COPD patients. This finding was compatible with the results presented in other studies.[9]

Wilson et al.[13] studied different CRP tests in patients with bronchiectasis; among 87 patients with bronchiectasis there was a median CRP value of 6.0 mg/L while a high sensitive CRP of 4.5 mg/L was observed in similar patient groups. In our study, a standard CRP measurement was used, and it was as twice high as CRP value in the study of Wilson et al. Wilson et al. also found a median leukocyte value of 7700 and a median neutrophil value of 4.9 in bronchiectasis patients, and in our study, a median leukocyte value was 8700 and a median neutrophil value was 5.68.

Martínez-García et al.[10] found a significant difference in the median CRP value (sensitive CRP) of 8.3 mg/dL in a COPD patient group with bronchiectasis and 5.3 mg/dL in a COPD patient group without bronchiectasis (p=0.018). In our study, although COPD patients with bronchiectasis were excluded, the median CRP value in COPD patients without bronchiectasis and the COPD patients who were admitted to the outpatient clinic with the symptom of fever had higher values than seen in Martinez-Garcia's study. In one of the studies performed by Jin et al.,[14] factors related to the presence of bronchiectasis in COPD patients were researched and CRP values were similar to those of our study: 11.5 mg/L in COPD patients without bronchiectasis.

During our study period, to the best of our knowledge, there was no data about NLR values in bronchiectasis patients in the English-language literature. In a few studies of non-cystic fibrosis bronchiectasis patients admitted to the outpatient clinic, NLR values were calculated by dividing the neutrophil to leukocyte values and the NLR value s were calculated as 2, which was lower than our findings.[15]

NLR in COPD in our country has been studied to some degree and offered some contribution to the literature. Günay et al.[16] first examined COPD patients who were in stable condition and not hospitalized and demonstrated that the NLR value increased during attack periods.

In addition, Saltürk et al.[6] found higher NLR values in cases of COPD exacerbation requiring intensive care unit assistance. Karakurt et al.[17] similarly studied the relationship between NLR value and the length of stay in hospital due to COPD exacerbation. In that study, the authors re-

Table 4. NLR and CRP in attack period and stable COPD

	NLR		CRP	
Studies	**Attack**	**Stable\Discharged**	**Attack**	**Stable\Discharged**
Günay[16] (Ward & outpatient clinic)	4.6	2.3	4.0	2.0
Kurtipek[20] (Outpatient clinic)	7.9	2.7	57.6	5.0
Our study (Outpatient clinic)	4.3	–	12.8	–
Our study (Ward)	7.3	–	45.0	–

COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein (mg/L); NLR: Neutrophil-to-lymphocyte ratio.

Table 5. Blood eosinophilia in attack periods and stable COPD

	Attack\Stay in hospital				Stable period\Discharged patient			
	Eosinophil ≥2%		Eosinophil <2%		Eosinophil ≥2%		Eosinophil <2%	
	NLR	CRP	NLR	CRP	NLR	CRP	NLR	CRP
Duman (Ward discharged)[5]	3.6	19.3	8.0	35.1	3.7	12.2	6.0	12.2
Saltürk (Intensive care unit)[6]	4.6	39.3	13.0	52.7	3.7	41.5	6.8	43.0
Karakurt (Ward)[17]	3.4–3.7	17.4–28.9	7.7–8.5	28.2–40.7	3.7–3.7	12.8–12.7	6.4–6.1	16.6–10.4
Our study (Ward)	3.3	22.0	8.6	53.5	–	–	–	–
Çoban (Outpatient clinic)[18]	2.9	9.0	5.1	10.0	–	–	–	–
Our study (Outpatient clinic)	3.0	7.8	5.5	16.0	–	–	–	–

COPD: Chronic obstructive pulmonary disease; CRP: C-reactive protein (mg/L); NLR: Neutrophil-to-lymphocyte ratio.

ported that NLR value decreased with attack treatment, and that the hospitalization period was more than 7 days with an increased NLR value.

Duman et al.[5] evaluated NLR value and survival time in COPD patients who were hospitalized and discharged from hospital due to acute exacerbation of COPD (AECOPD). They showed that as the severity of the exacerbation increases, NLR values also increased. In a large patient population, Çoban et al.[18] found an NLR value of 4 in in an outpatient group who did not require hospitalization due to AECOPD.

Table 4 shows a comparison of NLR and CRP values according to attack severity. In these studies, when eosinophilia was greater than or equal to 2%, it was demonstrated that NLR values did not increase much and remained constant during treatment of the exacerbation.

In the study conducted by Kang et al.,[19,20] eosinophilic COPD exacerbation outcomes and prognosis were much better than that seen in neutrophilic COPD exacerbations. Recent studies of neutrophilic (peripheral blood eosinophilia [PBE] <2%) and eosinophilic (PBE ≥2%) COPD attacks offered suggestions for the treatment of exacerbation. These studies predict that these markers will be utilized in COPD treatment guidelines for treatment options such as antibiotics, steroids, or both, in the future.

Table 5 summarizes the results of the study regarding NLR and CRP values according to PBE and follow-up of exacerbation in bronchiectasis patients. This represents a novel contribution to the literature.

In our study, among bronchiectasis patients who presented at the outpatient clinic, the NLR value was lower in the eosinophilic group than in the non-eosinophilic group, which made us think that the inflammatory response may be similar to that of COPD patients.

The existence of bronchiectasis was ignored in our study; however, there are some arguments focusing on a new phenotype of COPD existing with bronchiectasis. Although COPD existing with bronchiectasis was not considered in this study, in the presence of eosinophilia, NLR values of bronchiectasis patients were lower, as in the COPD patients. The inflammatory response in bronchiectasis appeared to be similar that (the inflammatory response) in COPD in the presence of eosinophilia.

Although knowledge of the CRP value in cases of both in-

flammation and infection is beneficial, NLR together with PBE can allow easier, cheaper, prompt treatment that will reflect the clinical progression of the patient instantly and guide steroid and antibiotic choices.

Limitations

The first limitation of the present study is the single-center and retrospective design. However, the large number of patients evaluated by a pulmonary specialist and institutional faculty, as well as the results, should be valuable.

Though radiology and spirometry records are the gold standards in the definition of bronchiectasis and COPD, respectively, the lack of these parameters is a second limitation. The diagnoses of COPD and bronchiectasis were made by a pulmonary specialist in a chest diseases training hospital. Third, while NLR is studied as a marker of infection and inflammation in COPD and bronchiectasis, NLR values may not be generalized as a marker for other infectious and inflammatory diseases. We think the study will add to other studies about different diseases.

The strength of the study is that a large volume of patient data was obtained from the electronic database and the errors related to data entry were minimized.

CONCLUSIONS

In COPD, chronic systemic inflammation may explain an increase in NLR value. This may be lower than that seen in patients admitted to the hospital due to a COPD attack in comparison with what might be thought to be a bronchiectasis attack, a common parenchymal injury of the lungs.

Infection is seen in two-thirds of patients experiencing a COPD attack when the bronchi have been previously infected. One-third of COPD exacerbations have noninfectious causes (exposure to dust, gas, smoke, cigarettes). When noninfectious sources are present for extended periods of time it may cause progression in the COPD pathogenesis and and increase of inflammatory markers. It is thought that this is the reason COPD patients had a higher NLR value than that seen in bronchiectasis patients with airway and parenchymal lung disease.

The NLR and PBE values can guide clinicians in the determination of both the type of attack and treatment options to be used in COPD and bronchiectasis cases, as well as in the follow-up of these diseases.

In the presence of bronchiectasis, the use of NLR and PBE may represent a new approach in treatment, but additional studies are required.

Continuous systemic inflammation occurs in both the attack phase and stable COPD; therefore, permanent use of anti-inflammatory therapy is crucial in COPD patients.

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Ethics Committee Approval

Approved by the local ethics committee (12.10.2016/119).

Informed Consent

Retrospective study.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: P.S., Z.K., S.G.; Design: P.S., E.T.; Data collection &/or processing: İ.İ., N.Ç.G, E.A.; Analysis and/or interpretation: Z.K., G.G.; Literature search: M.Ç.A., İ.Ö.; Writing: Z.K., S.G.; Critical review: Z.K., G.G.

Conflict of Interest

None declared.

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Nötrofil Lenfosit Oranı: Bronşektazide KOAH'dan Daha mı Yüksektir?

Amaç: Bronşektazi ile kronik obstrüktif akciğer hastalığı (KOAH) semptom ve ataklarda benzerlik gösterir. Kronik obstrüktif akciğer hastalığında kronik sistemik enflamasyon ve broşiektazide yıkıcı pulmoner enflamasyon, enflamatuvar belirteçlerin artmasına neden olur. Periferik kan eozinofili (PKE) (>%2) ve nötrofil lenfosit oranı (NLO) atak tipi ve tedavi yaklaşımını belirlemede etkilidir. Bu çalışmada, enflamatuvar belirteçlerin farklılıkları ve KOAH ve bronşektazide PKE varlığının araştırılması amaçlanmıştır.

Gereç ve Yöntem: Geriye dönük kesitsel bu çalışmada 2014 yılında Göğüs Hastalıkları Hastanesinde poliklinikte ve yatırılarak tedavi edilen bronşektazi (ICD J47.0) ve KOAH (ICD J.44.0-44.9) hastaları değerlendirildi. Kanser, hematolojik ve renal bozukluklar, bronşektazi ile beraber KOAH olan olgular dışlandı. Hemogram değerleri olanlar çalışmaya dahil edildi, demografik özellikleri, C-reaktif protein (CRP) kaydedildi, NLO hesaplandı. Çalışma grupları PKE'ye göre gruplandırıldı.

Bulgular: Çalışmaya 2664 hasta alındı (bronşektazi n=1204, bronşektazi yatan hasta=180; KOAH=685). Bronşektazi grubunda NLR anlamlı olarak daha düşüktü. C-reaktif protein, yatan hasta grubunda KOAH'dan daha düşüktü. Bronşektazi ve KOAH hastalarında yatan ve ayaktan hasta gruplarında eozinofil oranı sırasıyla %24, %40 ve %18, %40 idi. Periferik kan eozinofili hastalarında NLO değerleri eozinofilik olmayan hastalardan daha düşüktü.

Sonuç: Bronşektazide, enflamatuvar belirteçler KOAH hastalarından daha düşük saptandı. Bu durum, KOAH için sürekli anti-enflamatuvar tedavi kullanımı açısıdan önemlidir. Nötrofil lenfosit oranı ve PKE ile ilgili ileride yapılacak çalışmalar tedavinin doğasını gösterebilir.

Anahtar Sözcükler: Bronşektazi; enflamatuvar belirteçler; kronik obstrüktif akciğer hastalığı; netrofil lenfosit oranı.