Prognostic value of platelet and neutrophil to lymphocyte ratio in COPD patients

Ahmed Gouda El-Gazzar, Mohammed Hussein Kamel, Ola Kamal Mohammad Elbahnasy & Marwa El-Sayed El-Naggar

To cite this article: Ahmed Gouda El-Gazzar, Mohammed Hussein Kamel, Ola Kamal Mohammad Elbahnasy & Marwa El-Sayed El-Naggar (2020) Prognostic value of platelet and neutrophil to lymphocyte ratio in COPD patients, Expert Review of Respiratory Medicine, 14:1, 111-116, DOI: 10.1080/17476348.2019.1675517

To link to this article: https://doi.org/10.1080/17476348.2019.1675517

Accepted author version posted online: 02 Oct 2019.
Published online: 13 Oct 2019.
Prognostic value of platelet and neutrophil to lymphocyte ratio in COPD patients

Ahmed Gouda El-Gazzar, Mohammed Hussein Kamel, Ola Kamal Mohammad Elbahnasy and Marwa El-Sayed El-Naggar

Chest department, Faculty of Medicine, Benha University, Benha, Egypt; Outpatient clinic department, Benha Chest Hospital, Benha, Egypt

ABSTRACT

Background: Chronic obstructive pulmonary disease (COPD) is the third driving reason for death around the world and a real number of patients suffers from disease exacerbation. Platelet lymphocyte ratio (PLR) and neutrophil lymphocyte ratio (NLR) are novel biomarkers in acute exacerbation of COPD (AECOPD) and related to expanded 90-day mortality in patients with COPD.

Objectives: This work aimed to assess NLR and PLR in COPD patients.

Methods: This case-control study was carried out on 100 COPD patients and 60 healthy subjects. Complete blood count (CBC) with differential was made during and after exacerbation to define NLR and PLR.

Results: The cases and controls groups were matched as regards age, sex, and body mass index (BMI) (P-values: 0.3, 0.2, and 0.06 respectively). NLR and PLR were increased significantly in COPD patients (2.24 ± 0.56 and 157.1 ± 28.36) compared to control group (1.31 ± 0.23 and 102.82 ± 3.99) (P-value < 0.0001). During exacerbation NLR and PLR were elevated significantly compared to stable condition (P-value < 0.0001). NLR and PLR show a significant positive correlation with smoking index, COPD stage, and dyspnea severity.

Conclusion: NLR and PLR increased in stable COPD patients and further increased during exacerbation that can predict in hospital mortality.

1. Introduction

Chronic obstructive pulmonary disease (COPD) is the third driving reason for death worldwide [1]. It is characterized as being a preventable and treatable disease that has constant respiratory symptoms and airflow limitation due to abnormalities in the airway and/or alveoli resulting from exposure to harmful particle or gases [2]. As COPD is a chronic disease, large number of patients experience the ill effects of exacerbation, which characterized by an acute aggravation of respiratory symptoms that make an adjustment in treatment necessary [3]. The utility of blood-based biomarkers as diagnostic and prognostic instruments for patients with acute exacerbation of COPD (AECOPD) have been formerly assessed [4]. White blood cell (WBC) count and its subtypes have a role in predicting the future risk of exacerbation and mortality [5]. Thrombocytosis can expect in-hospital and 1-year mortality in patients with AECOPD. Also, lymphopenia accompanied with adverse consequences in these patients [6]. Platelet lymphocyte ratio (PLR) and neutrophil lymphocyte ratio (NLR) have been identified as novel biomarkers in AECOPD. Higher PLR is related to expanded 90-day mortality in COPD patients [7]. Additionally, NLR recognized as subclinical inflammatory biomarker and COPD patients with high NLR have greater exacerbation risk that necessitates hospitalization [8,9].

2. Aim of the work

Aim of the work is to assess NLR and PLR in COPD patients.

3. Subjects and methods

3.1. Study selection

This prospective case-control study was carried out in chest department, Benha university hospitals on 100 COPD patients (same COPD patients enrolled during exacerbation and after being stable; the patient has chronic symptoms and persistent airflow limitation without exacerbation) and 60 healthy controls (apparently healthy age and sex matched group who did not suffer from COPD or other disease that can affect complete blood count (CBC) value) in the period between October 2016 and May 2018.

3.2. Exclusion criteria

The following patients were excluded: Age less than 18 years, pregnant females, acute cerebrovascular event, acute coronary syndrome, hematological disease, inflammatory bowel disease, rheumatic diseases with pulmonary involvement, chronic liver disease and renal disease, thrombocytopenia and patients with known malignancies. These conditions may have an effect on respiratory symptoms or CBC values.

3.3. Study description

All patients included in this work were subjected to the following.
Figure 1−increased 60 mmHg. Moderate shows the relationship between is the correlation coefficient.

Non-life-threatening acute respiratory failure (2.9 ± 0.19) and (194.4 ± 3.9), respectively (P < 0.05) PLR cut off values in predicting AECOPD. The degree of significance in this work started below 0.05 (P < 0.05) was judged to be significant (S), P value > 0.05 is non-significant (NS), and P ≤ 0.001 is highly significant (HS).

4. Results

There were no significant dissimilarity between cases and controls in relation to (age, sex, weight, height, and BMI) (Table 1). FEV1 and smoking index were highly significantly raised in COPD cases compared to control group (Some of COPD quitted smoking and some did not. Also, all patients during exacerbation did not smoke for at least 2 weeks). NLR and PLR were significantly increased in COPD patients (2.24 ± 0.56 and 157.1 ± 28.36) compared to control group (1.31 ± 0.23 and 102.82 ± 3.99), respectively (P value < 0.0001). These two ratios were further increased during AECOPD. Among different degree of severity of AECOPD, the highest levels of NLR and PLR were found in AECOPD with life threatening acute respiratory failure (2.9 ± 0.19) and (194.4 ± 3.9), respectively (Table 2). Table 3 shows the relationship between mean value of NLR, PLR and clinical characteristics in COPD. They increased in smokers, patients with cardiovascular (CVS) comorbidities, C+D GOLD class of COPD. Also, NLR and PLR increased in patients with FEV1 < 50%, in stages III and IV.

4.3. Statistical analysis [13]

The collected data tabulated and analyzed using SPSS version 23 software (IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.). Categorical data were presented as number and percentages. Comparisons between two groups for numerical variables were done using independent t test for normally distributed variables or Mann Whitney U test for non-normally distributed variables. F test was used for comparison of NLR and PLR according to exacerbation severity. Correlations were done using Pearson’s correlation; ‘r’ is the correlation coefficient. It ranged from −1 to +1. The receiver operating characteristic (ROC) curve was used to detect NLR and PLR cut off values in predicting AECOPD. The degree of significance in this work started below 0.05 (P < 0.05) was judged to be significant (S), P value > 0.05 is non-significant (NS), and P ≤ 0.001 is highly significant (HS).

3.4. Statistical analysis [13]

The collected data tabulated and analyzed using SPSS version 23 software (IBM SPSS Statistics for Windows, Version 23.0. Armonk, NY: IBM Corp.). Categorical data were presented as number and percentages. Comparisons between two groups for numerical variables were done using independent t test for normally distributed variables or Mann Whitney U test for non-normally distributed variables. F test was used for comparison of NLR and PLR according to exacerbation severity. Correlations were done using Pearson’s correlation; ‘r’ is the correlation coefficient. It ranged from −1 to +1. The receiver operating characteristic (ROC) curve was used to detect NLR and PLR cut off values in predicting AECOPD. The degree of significance in this work started below 0.05 (P < 0.05) was judged to be significant (S), P value > 0.05 is non-significant (NS), and P ≤ 0.001 is highly significant (HS).

4. Results

There were no significant dissimilarity between cases and controls in relation to (age, sex, weight, height, and BMI) (Table 1). FEV1 and smoking index were highly significantly raised in COPD cases compared to control group (Some of COPD quitted smoking and some did not. Also, all patients during exacerbation did not smoke for at least 2 weeks). NLR and PLR were significantly increased in COPD patients (2.24 ± 0.56 and 157.1 ± 28.36) compared to control group (1.31 ± 0.23 and 102.82 ± 3.99), respectively (P value < 0.0001). These two ratios were further increased during AECOPD. Among different degree of severity of AECOPD, the highest levels of NLR and PLR were found in AECOPD with life threatening acute respiratory failure (2.9 ± 0.19) and (194.4 ± 3.9), respectively (Table 2). Table 3 shows the relationship between mean value of NLR, PLR and clinical characteristics in COPD. They increased in smokers, patients with cardiovascular (CVS) comorbidities, C+D GOLD class of COPD. Also, NLR and PLR increased in patients with FEV1 < 50%, in stages III and IV.

4. Results

There were no significant dissimilarity between cases and controls in relation to (age, sex, weight, height, and BMI) (Table 1). FEV1 and smoking index were highly significantly raised in COPD cases compared to control group (Some of COPD quitted smoking and some did not. Also, all patients during exacerbation did not smoke for at least 2 weeks). NLR and PLR were significantly increased in COPD patients (2.24 ± 0.56 and 157.1 ± 28.36) compared to control group (1.31 ± 0.23 and 102.82 ± 3.99), respectively (P value < 0.0001). These two ratios were further increased during AECOPD. Among different degree of severity of AECOPD, the highest levels of NLR and PLR were found in AECOPD with life threatening acute respiratory failure (2.9 ± 0.19) and (194.4 ± 3.9), respectively (Table 2). Table 3 shows the relationship between mean value of NLR, PLR and clinical characteristics in COPD. They increased in smokers, patients with cardiovascular (CVS) comorbidities, C+D GOLD class of COPD. Also, NLR and PLR increased in patients with FEV1 < 50%, in stages III and IV. ROC curve analysis for utilizing NLR alone to anticipate in-hospital mortality shows an ideal NLR cut-off of 1.505, area under the curve (AUC) of 0.9945 with 94% sensitivity and 86.7% specificity. For PLR, the ROC curve showed an ideal cut-off PLR of 113.495, AUC of 1 with 100% sensitivity and 100% specificity (Table 4; Figure 1). NLR and PLR shows a significantly positive correlation with smoking index, COPD stage, and mMRC score while they were negatively correlated with FEV1% values (Table 5).
Table 1. Statistical analysis of demographic data (Age, sex, weight, height, BMI, FEV1, and smoking index among the studied groups.

<table>
<thead>
<tr>
<th></th>
<th>Cases (n = 100)</th>
<th>Controls (n = 60)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>Mean ± SD</td>
<td>59.6 ± 11</td>
<td>56.8 ± 9.5</td>
</tr>
<tr>
<td>Sex</td>
<td>Male, n (%)</td>
<td>84 (84.0)</td>
<td>44 (73.3)</td>
</tr>
<tr>
<td></td>
<td>Female, n (%)</td>
<td>16 (16.0)</td>
<td>16 (26.7)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>Mean ± SD</td>
<td>80.7 ± 15.1</td>
<td>76.9 ± 12.3</td>
</tr>
<tr>
<td>Height (m)</td>
<td>Mean ± SD</td>
<td>169.5 ± 7</td>
<td>171.7 ± 6.2</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>Mean ± SD</td>
<td>28.58 ± 6.18</td>
<td>26.06 ± 4.44</td>
</tr>
<tr>
<td>FEV1</td>
<td>Mean ± SD</td>
<td>41 ± 17.6</td>
<td>89.6 ± 9</td>
</tr>
<tr>
<td>Smoking index</td>
<td>Mean ± SD</td>
<td>487.7 ± 347.8</td>
<td>124.3 ± 156.3</td>
</tr>
</tbody>
</table>

BMI: body mass index, FEV1: forced expiratory volume in the first second.

Table 2. Statistical analysis of NLR and PLR in stable COPD and during exacerbation; and comparison between different degrees of severity of AECOPD.

<table>
<thead>
<tr>
<th></th>
<th>Stable COPD (N = 100)</th>
<th>During AECOPD (N = 100)</th>
<th>P-value</th>
<th>Mild AECOPD (N = 16)</th>
<th>Moderate AECOPD (N = 14)</th>
<th>Severe AECOPD without respiratory failure (N = 28)</th>
<th>AECOPD with non-life-threatening acute respiratory failure (N = 18)</th>
<th>AECOPD with life-threatening acute respiratory failure (N = 24)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>NLR (mean ± SD)</td>
<td>2.24 ± 0.56</td>
<td>2.65 ± 0.41</td>
<td>&lt;0.0001</td>
<td>1.6 ± 0.23</td>
<td>1.5 ± 0.14</td>
<td>2.2 ± 0.24</td>
<td>2.6 ± 0.07</td>
<td>2.9 ± 0.19</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PLR (mean ± SD)</td>
<td>157.11 ± 28.36</td>
<td>180.25 ± 22.16</td>
<td>&lt;0.0001</td>
<td>120.8 ± 2.4</td>
<td>122.4 ± 1.5</td>
<td>155.9 ± 3.7</td>
<td>166.8 ± 15.4</td>
<td>194.4 ± 3.9</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

Table 3. The relationship between mean value of NLR, PLR, and clinical characteristics in COPD.

<table>
<thead>
<tr>
<th></th>
<th>Smoking status</th>
<th>N (%)</th>
<th>NLR (Mean ± SD)</th>
<th>P-value*</th>
<th>PLR (Mean ± SD)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking status</td>
<td>Quitted</td>
<td>32(32)</td>
<td>1.68 ± 0.35</td>
<td>&lt;0.0001</td>
<td>128.14 ± 13.96</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>Still</td>
<td>68(68)</td>
<td>2.5 ± 0.42</td>
<td>&lt;0.0001</td>
<td>170.74 ± 22.53</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>CVS Comorbidities</td>
<td>Yes</td>
<td>40(40)</td>
<td>2.59 ± 0.41</td>
<td>&lt;0.0001</td>
<td>178.83 ± 21.86</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>60(60)</td>
<td>2.01 ± 0.52</td>
<td>&lt;0.0001</td>
<td>142.63 ± 22.44</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>GOLD stage</td>
<td>A + B</td>
<td>30(30)</td>
<td>1.59 ± 0.18</td>
<td>&lt;0.0001</td>
<td>123.6 ± 8.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>C + D</td>
<td>70(70)</td>
<td>2.52 ± 0.4</td>
<td>&lt;0.0001</td>
<td>171.47 ± 20.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>mMrc</td>
<td>&lt; 2</td>
<td>28(28)</td>
<td>1.68 ± 0.37</td>
<td>&lt;0.0001</td>
<td>129.02 ± 20.4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>≥ 2</td>
<td>72(72)</td>
<td>2.46 ± 0.46</td>
<td>&lt;0.0001</td>
<td>168.44 ± 23.29</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>FEV1</td>
<td>&lt;50%</td>
<td>70(70)</td>
<td>2.51 ± 0.4</td>
<td>&lt;0.0001</td>
<td>171.22 ± 20.82</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>≥ 50%</td>
<td>30(30)</td>
<td>1.61 ± 0.3</td>
<td>&lt;0.0001</td>
<td>124.18 ± 10.12</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Stage</td>
<td>I &amp; II</td>
<td>30(30)</td>
<td>1.55 ± 0.17</td>
<td>&lt;0.0001</td>
<td>124.18 ± 10.12</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td></td>
<td>III &amp; IV</td>
<td>70(70)</td>
<td>2.54 ± 0.36</td>
<td>&lt;0.0001</td>
<td>173.8 ± 18.9</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

*Numerical variables were done using independent t test for normally distributed variables or Mann Whitney U test for non-normally distributed variables.

5. Discussion

Both NLR and PLR are pointers of immune reaction to different stress stimuli [14]. During AECOPD, the inflammation severity is seriously elevated leading to increment of the NLR and PLR, which might be utilized as a sign of inflammation and prognosis for COPD patients [15]. In this work, NLR and PLR were increased significantly in COPD patients in contrast to control group and their levels increased seriously during exacerbation. Also, their levels increased significantly with increase severity of acute exacerbation (Table 2). Yao et al., in a study on 303 COPD patients, found that the mean levels of NLR and PLR were elevated during acute exacerbation (7.92 and 207.21, respectively) [16]. Also Kurtipek et al. found in 94 patients with COPD that mean NLR and PLR increased in stable condition (2.75 ± 1.11 and 137.39 ± 65.42) and increased more significantly during exacerbation (7.99 ± 5.72 and 231.18 ± 141.36) [17]. Furthermore, Shah and Mishra evaluated the utility of NLR in patients with AECOPD, stable disease, and healthy controls (n = 149, 153, 71, respectively). They found that elevated NLR was strongly associated with AECOPD compared to stable disease and controls and they concluded that NLR is a strong biomarker of AECOPD that may aid as an indicator for hospital admission [18]. In another study, NLR and PLR had used to anticipate the severity of the exacerbation. NLR values in conjunction with clinical findings were helpful in determining where to admit the patient. If NLR is 3.5–4.0, the patient is for outpatient follow-up. If it is 6.5–7.0, hospital admission is recommended and

Table 4. Sensitivity, specificity, and cut off point of NLR and PLR in diagnosis of COPD exacerbation.

<table>
<thead>
<tr>
<th></th>
<th>AUC</th>
<th>SE</th>
<th>95% CI</th>
<th>Best cutoff</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>NLR</td>
<td>0.945</td>
<td>0.023</td>
<td>(0.9–0.991)</td>
<td>1.505</td>
<td>94</td>
<td>86.7</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>PLR</td>
<td>1</td>
<td>0</td>
<td>113.495</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

* Receiver operating characteristic (ROC) curve analysis was used to evaluate the sensitivity and specificity.
if it is 13.0–14, admission to the ICU should be reviewed [19]. In this study, NLR and PLR were elevated significantly in currently smoking, COPD patients with CVS comorbidities, patients with mMRC > 2, and in C + D GOLD class of COPD. Also, NLR and PLR had increased significantly in patients with FEV1 < 50% compared to FEV1 > 50% and in stages III and IV compared to stages I and II in patients with COPD (Table 3). In a study on 243 patients, Tulgar et al. found that NLR had increased significantly among smokers compared to non-smokers (2.14 ± 0.19 versus 1.66 ± 0.6, p: 0.001) as effect of smoking is one of the pathogenesis of COPD and decrease smoking lead to decreases in inflammatory burden in the lung [20]. While Duyar and his colleagues investigated the relation among NLR, PLR, and the seriousness of stable COPD patients regarding stage of the disease, airflow obstruction, mMRC dyspnea scale, and frequency of exacerbation. They found that PLR was raised in male patients with serious airflow obstruction (FEV1 less than 50%) and in GOLD stage C and D. So, in stable disease, PLR could identify COPD patients with increased risk of exacerbation [21]. Also, Günay et al. had evaluated the role of neutrophil-to-lymphocyte ratio in COPD patients and compared it with other inflammatory biomarkers. From 269 COPD patients, they established that NLR in stable disease were significantly higher than those of the controls. During acute exacerbation there was further increase in comparison to stable state [22]. Paliogiannis et al. concluded that the NLR has a valuable role in COPD patients in anticipating exacerbation and mortality [23]. Furthermore, Taylan et al. declared that expanded NLR is as helpful as C-reactive protein (CRP) in the assessment of inflammation in AECOPD, and it is valuable for early recognition of exacerbation in patients with normal levels of conventional markers [24]. Also, Yao et al., found that NLR and PLR were remarkably higher in patients who died in hospital than among those who remained alive [16]. In this study, ROC curve analysis for utilizing NLR alone to anticipate in-hospital mortality showed an ideal NLR cutoff of 1.505, AUC of 0.9045 with 94% sensitivity and 86.7% specificity. For PLR, the ROC curve showed an ideal cutoff PLR of 113.495, AUC of 1 with 100% sensitivity and 100% specificity (Table 4; Figure 1), which suggest that the NLR and PLR are straightforward and valuable biomarker for anticipating in-hospital mortality in patients with COPD. In similar study, Yao et al. found that ROC curve analysis for utilizing NLR to foresee in-hospital mortality demonstrated cutoff NLR of 6.24, AUC of 0.803 with 81.08% sensitivity and 69.1% specificity. Its predictive capacity is greater than CRP or PLR separately. They concluded that the conjunction of PLR, CRP and NLR expanded the prognostic sensitivity for in-hospital mortality in exacerbated patients [16]. Babaoğlu et al. found that NLR was significantly

![Figure 1. ROC for analysis of using NLR and PLR in diagnosis of COPD exacerbation.](image)

Table 5. Correlation between NLR, PLR, and different parameter in COPD patients.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>NLR</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>0.222</td>
<td>0.223</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>0.012</td>
<td>0.05</td>
</tr>
<tr>
<td>Height (m)</td>
<td>0.029</td>
<td>0.082</td>
</tr>
<tr>
<td>BMI</td>
<td>0.142</td>
<td>0.174</td>
</tr>
<tr>
<td>FEV1</td>
<td>-0.754**</td>
<td>-0.796**</td>
</tr>
<tr>
<td>Smoking index</td>
<td>0.519**</td>
<td>0.513**</td>
</tr>
<tr>
<td>mMRC</td>
<td>0.872**</td>
<td>0.931**</td>
</tr>
</tbody>
</table>

BMI: body mass index, FEV1: forced expiratory volume in the first second, mMRC: modified medical research council, NLR: neutrophil lymphocyte ratio, PLR: platelet lymphocyte ratio.

* Pearson’s correlation was used.
higher in dead patients with AECOPD ($p = 0.009$) and recommended that it can be used as a prognostic marker in hospital [25]. Also, Rahimia et al. studied 315 patients with AECOPD, they found in ROC curve analysis that NLR had the highest AUC of 0.717 with a cutoff value of 4, 87% sensitivity and 40% specificity [26]. Xiong et al. supported that raised NLR might be related to lifelong mortality in patients with COPD. Thus, NLR has a role in COPD as an indicator for hospitalization, inflammation assessment and mortality [27]. In this work, NLR and PLR showed a significant positive correlation with the smoking index, COPD stage and mMRC score while they were negatively correlated with FEV1 values (Table 5). This result matched Furutate et al., who found in 141 stable and 49 exacerbated COPD patients that NLR was correlated significantly with FEV1, BODE (BMI, airflow obstruction, dyspnea, exercise capacity) index and mMRC dyspnea scale score which recommend the possible role of the NLR to anticipate elements of disease severity [28]. Yasar et al., found a negative correlation between NLR and FEV1, while there was positive correlation with dyspnea score [29]. Um and his colleagues concluded that NLR showed a significant opposite relationship to airflow limitation, and patients with higher NLRs liable to exacerbate frequently during past and future 1 year follow-up [30]. Also Lee et al. studied 61 patients with stable COPD and 59 with acute exacerbation and found that NLR showed remarkable correlations with BMI, dyspnea, level of airway obstruction, BODE index, 6-minute walk test, exercise tolerance, and scale of mMRC [31]. In a retrospective study done by Kalemci et al., based on data collected from 153 patients with COPD, a significant increase was found in platelet distribution width (PDW) and PLR as the severity of COPD increases [32]. While Ergun and Ergan studied the predictive role of NLR and PLR in hospitalized COPD patients and found that the mean values of NLR and PLR were increased in non-survivors [33].

6. Conclusions

From this study, it concluded that NLR and PLR increased in stable COPD patients and further increased during exacerbation which can predict in hospital mortality. Further studies on the job of the NLR and PLR in dealing with patients with COPD are recommended.

Funding

This paper was not funded.

Author contributions

All authors were involved in the study design, analysis, interpretation of the data and revising its content. All authors agree to be accountable for all aspects of the work.

Declaration of interest

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

Ethical approval

The Research Ethics Committee at the Faculty of Medicine, Benha University has approved the study. Informed consent was obtained from every single individual member incorporated into the study.

Reviewer disclosures

Peer reviewers on this manuscript have no relevant financial or other relationships to disclose.

References

Papers of special note have been highlighted as either of interest (-) or of considerable interest (-) to readers.


- This reference discusses COPD successfully.


- Those references support the idea of this study.


- Those references support the idea of this study.


- Those references support the idea of this study.


