Neutrophil to Lymphocyte Ratio as a Predictor of Left Ventricular Hypertrophy in Patients with Newly Diagnosed Hypertension

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Abstract

Objective: Concentric or eccentric left ventricular hypertrophy (LVH) is an independent prognostic factor of major cardiovascular events in hypertension (HT). A high neutrophil to lymphocyte ratio (NLR) is correlated with high mortality and poor prognosis in cardiovascular disease. This study was performed to investigate the associations between NLR and different left ventricular (LV) geometric patterns in patients with newly diagnosed HT.

Methods: The study population consisted of 222 patients with newly diagnosed HT (mean age: 53.2 ± 10.0 years). Echocardiographic examination was performed in all patients. Four different geometric patterns were determined in hypertensive patients according to the left ventricular mass index (LVMI) and relative wall thickness (RWT).

Results: The baseline demographic characteristics were similar in all groups. The NLR and platelet to lymphocyte ratio (PLR) were higher in the eccentric hypertrophy and concentric hypertrophy groups compared to the normal geometry and concentric remodeling groups (p < 0.05, for all). NLR was positively and significantly correlated with LVMI (r = 0.508, p < 0.001). Linear regression analysis showed that LVMI was independently correlated with NLR (β = 5.440, p < 0.001), systolic blood pressure (β = 0.284, p < 0.001), ejection fraction (β = -0.201, p < 0.001), E/A (β = -2.270, p = 0.24), and high-density lipoprotein cholesterol (β = -0.245, p < 0.001).

Conclusions: We demonstrated that patients with newly diagnosed HT with LVH had significantly higher NLR and PLR compared to those without LVH. In addition, NLR predicted LVH in hypertensive patients. The results of this study suggested that inflammation plays a role in the pathogenesis of LVH in hypertensive subjects.

Introduction

The prevalence of hypertension (HT) is increasing across the world irrespective of income status [1]. HT persists as a major public health problem that the global prevalence of hypertension was estimated to be 1.13 billion in 2015, with a prevalence of over 150 million in central and Eastern Europe [2]. It also affects more than one third of the adult population in USA [3].

There are four different geometric patterns of left ventricle (LV) in HT. These geometric patterns include normal LV geometry (NG), concentric remodeling (CR), eccentric hypertrophy (EH) and concentric hypertrophy (CH). Left ventricular hypertrophy (LVH) is traditionally classified as concentric or eccentric, which is one of the substantial HT-mediated organ damage. It is a fundamental process of adaptation to an increased hemodynamic overload [4]. LVH is an independent prognostic factor for major cardiovascular events including sudden cardiac death, acute myocardial infarction, stroke, and congestive heart failure in hypertensive subjects [5]. For this reason, hypertensive patients with LVH have an increased risk of cardiovascular events compared to hypertensive patients without LVH [6].

It is well known that low-grade inflammation plays a significant pathophysiological role in HT and cardiovascular disease [7,8]. Several study have been demonstrated that LVH is a low-grade inflammatory state, which is predominantly managed by various inflamma-
tory cascades [9,10]. Animal studies demonstrated that inflammatory markers in fibrotic process are the main component in ventricular remodeling [11,12].

The neutrophil to lymphocyte ratio (NLR) is mostly used as an easily biomarker of systemic inflammatory status [13]. NLR is a simple and readily available marker for chronic low-grade inflammation that can be easily obtained from differential counts of white blood cell (WBC) subtypes [14]. Based on aforementioned results, the present study aimed to investigate whether NLR are associated with LV remodeling in newly diagnosed hypertensive patients.

**Methods**

**Study population**

Between January 2018 and February 2019, consecutive subjects who admitted our outpatient clinic and having newly diagnosed essential HT were enrolled to the study. HT was defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg (blood pressure taken from three separate measurements in office in seated position) and SBP ≥ 130 mmHg and/or DBP ≥ 80 mmHg based on the mean 24-h circadian ambulatory blood pressure monitoring [15].

The exclusion criteria were defined as any condition that has a capability to alter cardiac structures and functions: patients with a history of anti-hypertensive drug therapy, secondary HT, history of coronary artery disease, cardiomyopathy, a body mass index (BMI) over 30 kg/m², diabetes mellitus, renal failure, chronic inflammatory disease, gestational HT, congenital heart disease, LV systolic dysfunction (ejection fraction < 50%), atrial fibrillation, liver disease, obstructive sleep apnea, valvular heart disease, patients with active infection, and WBC count of > 12 × 10³/µl or < 4 × 10³/µl.

**Study protocol**

All patients underwent a complete medical assessment, physical examination, blood analysis, electrocardiography (ECG), and transthoracic echocardiography (TTE). The institutional ethics committee approved the study protocol.

All echocardiographic examinations were performed using a Vivid 5 Pro device (General Electric, Horten, Norway) with a 2.5 MHz transducer. The measurements were performed in the left lateral decubitus position as recommended by current guideline American Society of Echocardiography [16], and three consecutive cycles were avaraged for each parameter. Standard echocardiographic analysis included two dimensional, M-mode, Doppler flow, and tissue Doppler flow measurements. Diastolic interventricular septum thickness (IVS), diastolic posterior wall thickness (PWT), left atrial (LA) diameter, left ventricle end systolic (LVESD) and end diastolic dimensions (LVEDD) were measured from parasternal long-axis view. Ejection fraction (EF) was calculated by using modified Simpson method. Mitral early diastolic velocity (E), mitral late diastolic velocity (A) were recorded from the apical transducer position of the sample volume situated between the mitral leaflet tips, and the ratio of E to A (E/A ratio) was calculated. Myocardial early diastolic velocity (Em) and myocardial late diastolic velocity (Am) wave velocities were measured with the sample volume using PWD from the mitral lateral and septal annulus.

LV mass (LVM) was calculated from M-mode echocardiography using the American Society of Echocardiography recommended Cube formula as following [16]:

\[ LVM (g) = 0.8 \times 1.04 \left( [IVS + PWT + LVEDD]^3 - LVEDD^3 \right) + 0.6 \]

LVM was divided by body surface area to obtain the LV index (LVMI, g/m²), which cut-off values of 115 g/m² and 95 g/m² for men and women respectively. Body surface area (m²) was calculated using the Du Bois formula \([\text{weight (kg)}^{0.425} \times \text{height (cm)}^{0.725} \times 0.007184]\). Relative wall thickness (RWT = 2 × PWT in end diastole/ LV diastolic diameter in end diastole) was calculated. Normal RWT was defined as values ≤ 0.42 and increased RWT as > 0.42 [16]. Patients with increased LVM and increased RWT were considered to have CH, and those with increased LVM and normal RWT were considered to have EH. Those with normal LVM and increased or normal RWT were considered to have concentric remodeling CR or NG, respectively.

Diastolic dysfunction was classified by mitral inflow pattern according to recent guidelines [17]. Grade I diastolic dysfunction was defined as E/A ratio of less than 0.8 along with a peak velocity of ≤ 50 cm/sec and normal left atrial pressure (LAP); grade II diastolic dysfunction was characterized by an E/A ratio > 0.8 but < 2 and increased LAP; and grade III diastolic dysfunction was defined as an E/A ratio of greater than 2 and increased LAP.

After a 12-h fasting period, bloods samples were obtained from the cephalic vein using a traumatic venipuncture and mixed with EDTA. Complete WBC counts, including neutrophil and lymphocyte counts, were measured using an automated hematology analyzer CELL-DYN Ruby (Abbott Diagnostics, Abbott Park, IL, USA) and expressed as × 1.000 cells/mm³. Hemoglobin and platelet count were also calculated. NLR was calculated by dividing the neutrophil count by the lymphocyte count, and platelet to lymphocyte ratio (PLR) was calculated as the number platelets divided by the lymphocyte count, both of which were obtained from the same blood samples. Plasma triglyceride, low-density lipoprotein, high-density lipoprotein, glucose, creatinine, uric acid and C-reactive protein (CRP) were analyzed on the Architect c8000 Chemistry System (Abbott Diagnostics, USA) using commercial kits (Abbott).
Statistical analysis

All analyses were performed using SPSS 17.0 (SPSS for Windows 17.0, Chiacago, IL). Continuous variables were presented as the mean ± standard deviation. Categorical variables were presented as frequencies and percentages. Kolmogorov Smirnov test was used to determine whether the continuous variables were distributed normally. Comparisons of categorical variables between the groups were conducted using the chi-square test. Analysis of variance (ANOVA) was used in the analysis of continuous variables. Pearson’s correlation test was used for the variables with a linear correlation and Spearman’s correlation test was used for those without a linear correlation. Linear regression analysis was used to determine which variables affects LVMI. 95.0% confidence intervals (95.0% CI) were determined. A two-tailed p-value of 0.05 was considered statistically significant.

Results

A total of 383 consecutive subjects with newly diagnosed essential HT were initially enrolled to the study. Forty-five patients were excluded owing to diabetes mellitus, 20 patients due to chronic renal insufficiency, 60 patients due to ischemic heart disease, 25 patients due to moderate or severe valvular heart disease, and 11 patients due to secondary HT. Consequently, measurements were obtained from 222 patients with newly diagnosed essential HT in this study (mean age: 53.2 ± 10.0 years, male: 112 patients). In the present study, four different LV geometric patterns were determined according to LVMI and RWT: (i) 58 patients with NG group, (ii) 60 patients with CR group, (iii) 50 patients with EH group, and (iv) 54 patients with CH group.

Table 1: Clinical and echocardiographic characteristics of different left ventricular geometry.

<table>
<thead>
<tr>
<th>Variables</th>
<th>NG group</th>
<th>CR group</th>
<th>EH group</th>
<th>CH group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>53.2 ± 10</td>
<td>54.1 ± 9.5</td>
<td>50.6 ± 12.1</td>
<td>54.6 ± 7.9</td>
<td>0.181</td>
</tr>
<tr>
<td>Sex (male), n(%)</td>
<td>31 (%53.4)</td>
<td>33 (%55)</td>
<td>24 (%48)</td>
<td>24 (%44.4)</td>
<td>0.658</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>17 (%29.3)</td>
<td>19 (%31.7)</td>
<td>21 (%42.6)</td>
<td>22 (%40)</td>
<td>0.404</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.3 ± 2.3</td>
<td>27.1 ± 2.3</td>
<td>26.8 ± 2.1</td>
<td>27.1 ± 2.4</td>
<td>0.560</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>83.6 ± 8.5</td>
<td>86.1 ± 10.5</td>
<td>90.0 ± 9.1</td>
<td>91.4 ± 9.8</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>142.3 ± 9.6</td>
<td>147.2 ± 14.0</td>
<td>154.0 ± 11.3</td>
<td>157.5 ± 16.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>EF (%)</td>
<td>60.8 ± 4.8</td>
<td>61.1 ± 3.9</td>
<td>58.5 ± 5.2</td>
<td>57.9 ± 4.6</td>
<td>0.001</td>
</tr>
<tr>
<td>LVEDD (cm)</td>
<td>4.6 ± 0.3</td>
<td>4.4 ± 0.3</td>
<td>5.1 ± 0.3</td>
<td>4.8 ± 0.2</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LVESD (cm)</td>
<td>3.1 ± 0.2</td>
<td>3.0 ± 0.2</td>
<td>3.3 ± 0.3</td>
<td>3.2 ± 0.3</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>IVS (cm)</td>
<td>1.00 ± 0.10</td>
<td>1.11 ± 0.12</td>
<td>1.17 ± 0.7</td>
<td>1.23 ± 0.13</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>PW (cm)</td>
<td>0.90 ± 0.10</td>
<td>1.04 ± 0.11</td>
<td>1.05 ± 0.5</td>
<td>1.17 ± 0.14</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LAD (cm)</td>
<td>3.3 ± 0.3</td>
<td>3.4 ± 0.3</td>
<td>3.7 ± 0.3</td>
<td>3.6 ± 0.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>RWT (g/m²)</td>
<td>0.38 ± 0.02</td>
<td>0.47 ± 0.05</td>
<td>0.40 ± 0.02</td>
<td>0.49 ± 0.06</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>152.8 ± 38.1</td>
<td>166.8 ± 34.8</td>
<td>225.5 ± 32.5</td>
<td>223.2 ± 43.3</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>LVMI (g/m²)</td>
<td>79.9 ± 18.2</td>
<td>85.9 ± 16.1</td>
<td>121.5 ± 17.1</td>
<td>126.7 ± 29.7</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>E/A</td>
<td>1.1 ± 0.5</td>
<td>0.8 ± 0.3</td>
<td>0.9 ± 0.4</td>
<td>0.9 ± 0.4</td>
<td>0.002</td>
</tr>
<tr>
<td>E/Em</td>
<td>8.0 ± 2.1</td>
<td>8.6 ± 3.0</td>
<td>9.2 ± 2.2</td>
<td>10.3 ± 3.9</td>
<td>0.001</td>
</tr>
</tbody>
</table>

Note: NG: Normal Geometry; CR: Concentric Remodelling; EH: Eccentric Hypertrophy; CH: Concentric Hypertrophy; BMI: Body Mass Index; DBP: Diastolic Blood Pressure; SBP: Systolic Blood Pressure; EF: Ejection Fraction; LVEDD: Left Ventricular End-Diastolic Dimension; LVESD: Left Ventricular End-Systolic Dimension; IVS: Interventricular Septal Thickness; PW: Posterior Wall Thickness; LAD: Left Atrial Diameter; RWT: Relative Wall Thickness; LV: Left Ventricular; LVMI: Left Ventricular Mass Index; E/A: Peak Velocity of Early Diastolic Flow/Peak Velocity of Late Diastolic Flow; E/Em: Peak Velocity Of Early Diastolic Flow Across Mitral Valve/Myocardial Peak Velocity of Early Diastole.
Comparison of baseline and echocardiographic characteristics of the groups were presented in Table 1. There was no statistically difference in terms of age, sex, smoking, and BMI among the groups (p > 0.05, for all). SBP, DBP, EF, LVEDD, LVESD, IVS, PWT, LA diameter, RWT, LV mass, E/A, and E/Em values were different among the groups (p < 0.05, for all). The measurements of SBP, DBP, LVMI and the ratio E/Em were increasing, while the ratio of E/A was decreasing from NG group to the CH group.

Comparison of laboratory characteristics of the groups were demonstrated in Table 2. No significant dif-

<table>
<thead>
<tr>
<th>Variables</th>
<th>NG group n = 58</th>
<th>CR group n = 60</th>
<th>EH group n = 50</th>
<th>CH group n = 54</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>13.7 ± 1.3</td>
<td>13.9 ± 1.6</td>
<td>13.7 ± 1.5</td>
<td>13.6 ± 1.4</td>
<td>0.737</td>
</tr>
<tr>
<td>WBC (10^3/µL)</td>
<td>8.7 ± 2.0</td>
<td>8.9 ± 1.7</td>
<td>8.9 ± 1.6</td>
<td>9.1 ± 1.8</td>
<td>0.737</td>
</tr>
<tr>
<td>Platelet (10^3/µL)</td>
<td>256.3 ± 82.0</td>
<td>254.1 ± 75.5</td>
<td>268.2 ± 85.3</td>
<td>265.0 ± 61.5</td>
<td>0.733</td>
</tr>
<tr>
<td>Neutrophils (10^3/µL)</td>
<td>4.8 ± 1.2</td>
<td>4.9 ± 1.1</td>
<td>5.4 ± 1.1</td>
<td>5.8 ± 1.2</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Lymphocytes (10^3/µL)</td>
<td>2.7 ± 0.7</td>
<td>2.6 ± 0.7</td>
<td>2.1 ± 0.6</td>
<td>2.3 ± 0.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>NLR</td>
<td>1.89 ± 0.44</td>
<td>1.93 ± 0.47</td>
<td>2.69 ± 0.59</td>
<td>2.63 ± 0.53</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>PLR</td>
<td>103.2 ± 46.2</td>
<td>100.9 ± 35.5</td>
<td>135.9 ± 53.9</td>
<td>120.6 ± 29.0</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Blood glucose (mg/dL)</td>
<td>95.7 ± 5.6</td>
<td>95.6 ± 8.0</td>
<td>95.3 ± 6.6</td>
<td>97.3 ± 6.9</td>
<td>0.436</td>
</tr>
<tr>
<td>Creatinine (mg/dL)</td>
<td>0.87 ± 0.19</td>
<td>0.92 ± 0.18</td>
<td>0.90 ± 0.16</td>
<td>0.92 ± 0.20</td>
<td>0.550</td>
</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>189.5 ± 25.2</td>
<td>190.0 ± 29.8</td>
<td>190.6 ± 33.3</td>
<td>204.6 ± 34.7</td>
<td>0.030</td>
</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>114.3 ± 23.7</td>
<td>113.4 ± 28.7</td>
<td>118.7 ± 26.7</td>
<td>126.0 ± 28.7</td>
<td>0.057</td>
</tr>
<tr>
<td>HDL-cholesterol (mg/dL)</td>
<td>24.2 ± 7.7</td>
<td>43.7 ± 8.5</td>
<td>40.7 ± 6.9</td>
<td>39.1 ± 5.7</td>
<td>0.006</td>
</tr>
<tr>
<td>Triglyceride (mg/dL)</td>
<td>193.9 ± 102.4</td>
<td>203.3 ± 95.9</td>
<td>170.0 ± 64.31</td>
<td>181.1 ± 85.0</td>
<td>0.224</td>
</tr>
<tr>
<td>Uric acid (mg/dL)</td>
<td>4.5 ± 0.9</td>
<td>4.6 ± 0.7</td>
<td>4.9 ± 0.9</td>
<td>5.1 ± 0.9</td>
<td>0.003</td>
</tr>
<tr>
<td>C-reactive protein (mg/dL)</td>
<td>0.43 ± 0.11</td>
<td>0.39 ± 0.13</td>
<td>0.40 ± 0.10</td>
<td>0.40 ± 0.12</td>
<td>0.342</td>
</tr>
</tbody>
</table>

*p < 0.001 between NG and CH groups; **p < 0.001 between CR and CH groups; *p < 0.001 between NG and EH groups, p = 0.002 between NG and CH groups; #p < 0.001 between CR and EH groups, p = 0.003 between CR and CH groups; $p < 0.001 between NG, EH and CH groups; **p < 0.001 between CR, EH and CH groups; #p = 0.006 between NG and EH groups; $p = 0.001 between CR and EH groups, p = 0.008 between CR and CH groups; #p = 0.049 between NG and CH groups; $p = 0.004 between NG and CH groups; *p = 0.003 between NG and CH groups; **p = 0.028 between CR and CH groups.


Figure 1: The relationship between neutrophil to lymphocyte ratio and different left ventricle geometry patterns.
Pearson correlation analyses showed that LVMI was correlated positively and moderately with NLR (r = 0.508, p < 0.001) and weakly with PLR (r = 0.229, p = 0.001) (Figure 2). In addition, Sperman’s correlation analyses demonstrated that SBP and DBP were correlated positively and weakly with NLR (r = 0.293, p < 0.001 and r = 0.227, p = 0.001, respectively) (Figure 3 and Figure 4).

Linear regression analysis show that LVMI was independently correlated with NLR (β = 5.440, 95% CI:
obesity, body size, insulin, and the renin-angiotensin system contribute to LVH development [18]. Also, non-hemodynamic factors, including transforming growth factor β1, tumor necrosis factor-alpha (TNF-α), and cytokines plays an important role in LV remodelling [19,20].

The physiological response of leukocytes to systemic inflammatory conditions is an increase in the number of neutrophils and a corresponding decrease in the relative lymphocyte count. WBC counts and its subtypes, such as neutrophil and lymphocyte, have an important role in modulating the inflammatory response in the atherosclerotic process [21], heart failure [22], aortic stenosis [23], HT [24] and LVH [25]. For this reason, the NLR is used as a biomarker of subclinical inflammation. In many studies, a higher NLR is generally correlated with high mortality and poor prognosis in cardiovascular disease [21,22]. Hypertrophic geometric patterns (EH, CH) are associated with poor prognosis in hypertensive patients [26]. In the present study, we found that NLR was higher in eccentric and concentric LV geometric patterns.

9.259 - 21.715; p < 0.001), SBP (β = 0.284, 95% CI: 0.313 - 0.883; p < 0.001), EF (β = -0.201, 95% CI: -168.55 - -0.470; p < 0.001), E/A (β = -2.270, 95% CI: -168.55 - -0.967; p = 0.24), and high-density lipoprotein cholesterol (β = -0.245, 95% CI: -1.376 - 0.553; p < 0.001) (Table 3).

**Discussion**

This is the first study which investigated the relationship between NLR and different LV geometry patterns in newly diagnosed hypertensive patients. In the present study, the major finding is that in patients with newly diagnosed HT, the NLR and PLR that were measured on admission were both significantly higher in patients with EH and CH. There was a positive and significant correlation between NLR and LVMI, SBP, and DBP. Moreover, we found that NLR is an independent predictor of LVH in newly diagnosed hypertensive patients.

In clinical studies, LVH is generally indentified as LVMI and it develops in response to chronic pressure and volume overload, which are responsible for cardiomyocyte hypertrophy and cardiac fibrosis. In addition, non-hemodynamic factors such as age, sex, obesity, body size, insulin, and the renin-angiotensin system contribute to LVH development [18]. Also, non-hemodynamic factors, including transforming growth factor β1, tumor necrosis factor-alpha (TNF-α), and cytokines plays an important role in LV remodelling [19,20].

The physiological response of leukocytes to systemic inflammatory conditions is an increase in the number of neutrophils and a corresponding decrease in the relative lymphocyte count. WBC counts and its subtypes, such as neutrophil and lymphocyte, have an important role in modulating the inflammatory response in the atherosclerotic process [21], heart failure [22], aortic stenosis [23], HT [24] and LVH [25]. For this reason, the NLR is used as a biomarker of subclinical inflammation. In many studies, a higher NLR is generally correlated with high mortality and poor prognosis in cardiovascular disease [21,22]. Hypertrophic geometric patterns (EH, CH) are associated with poor prognosis in hypertensive patients [26]. In the present study, we found that NLR was higher in eccentric and concentric LV geometric patterns.
PLR were associated with LVH in patients with newly
diagnosed HT. Our results support the relationship be-
tween the immune system activation and the presence of
LVH in patients with HT. But, this relationship has
generally been shown in the literature by more specif-
ic inflammation markers such as, cytokines and adhe-
sion molecules in both experimental and clinical studies
[38,39]. Moreover, NLR and PLR from peripheral venous
blood are less accurate than in vitro studies [40] sug-
Suggest that cytokines are associated with cardiac myo-
cyte hypertrophy in predicting LVH. Therefore, we can
say that the power of our study is lower.

As LVH effectively and independently predicts mor-
bidity and mortality in cardiovascular disease, it is im-
portant to diagnose LVH both for clinical practice and
research. In clinical practice, the diagnosis of LVH usu-
ally includes ECG and TTE. ECG is widely used and rou-
tine test in all patients with HT. However, a normal ECG
doesn’t excluded the presence of LVH due to its low sen-
sitivity [41]. M-mode echocardiography is currently the
standard clinical diagnosis method for LVH [26]. Data
from real-time three dimensional (3D) echocardiogra-
phy with regarding to LVM were performed few studies
[42]. Even so, 3D echocardiographic LVM data available
in normal subjects are not sufficient and its use in clini-
cal practice is not recommended [16]. Cardiac magnetic
resonance imaging is a very accurate method for LVH
detection. It is recommended for clinical trials investig-
ating LVM regression [41]. Nonetheless, its use in clinical
practice is quite difficult and expensive. The present
study showed that NLR measured on admission might
be usefull marker to predict the LVM. It is convenient
marker and can be easily measured blood samples.

Study Limitations

The limitations of the present study are as follows. (i)
The current study included a relatively small number of
patients, and didn’t include control group. (ii) Only one
measurement of admission full blood count and calcu-
lation of PLR and NLR were included in the analysis. (iii)
Although, many studies have demonstrated relation-
ships between cytokines and LVH, we didn’t evaluate
cytokines in the present study. (iv) These type of studies
do not establish causality.

Conclusions

Our study suggested that NLR and PLR are related
with different LV geometry patterns in hypertensive
patients, which has not been reported previously.
Also, NLR was an independent predictor of LVH in
hypertensive patients. The present study demonstrated
that NLR might be a useful and cost-effective marker to
evaluate LVH in newly diagnosed hypertensive patients.
However, larger scale studies are needed to support
these results.

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References


