# Systemic immune-inflammation index (SII) may be an effective indicator in predicting the left ventricular hypertrophy for patients diagnosed with hypertension

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# Abstract

The development of left ventricular hypertrophy (LVH) induced by hypertension is considered as a poor prognosis for patients. Similarly, high values of the systemic immune-inflammation index (SII) can be a leading cause for the increase of mortality and morbidity in cardiovascular events. Within this context, our study aimed to detect the association of SII with LVH caused by hypertension. The study encompassed 150 clients diagnosed with hypertensive in total, and evaluated them as two separate groups with regard to left ventricular mass index (LVMI), including 56 patients (37.3%) with LVH and 94 patients (62.6%) with non-LVH. The SII values regarding the group with LVH was detected remarkably higher than those of the non-LVH group ($p<0.001$). Additionally, the SII level of clients with eccentric and concentric hypertrophy was detected higher than those of the normal ventricular geometry and concentric remodeling groups. With regard to curve analysis of the receiver-operating characteristic (ROC), SII values above 869.5 predicted LVH with a sensitivity of 82.1% and specificity of 86.2% (AUC: 0.861; 95%CI: 0.792-0.930; $p < 0.001$). LVH can be predicted independently through the use of SII in clients diagnosed with hypertension, which may be a simple and easily calculable marker for judging LVH. Moreover, SII can serve as an accurate determinant for the prediction of LVH, in comparison to NLR and PLR.


# Summary

### What is known about this topic

- LVH is a hypertensive target organ damage that independently and clearly predicts the risk of cardiovascular disease that leads to mortality and morbidity, thus making the diagnose of LVH significant in terms of studies and clinical practise.

### What this study adds

- The SII values regarding the group with LVH was detected remarkably higher than those of the non-LVH group.
- High SII as well as high sensitivity C-reaktive protein and systolic blood pressure were determined to predict the presence of LVH as independent variables.
- The SII level of patients with eccentric and concentric hypertrophy was detected higher than those of the normal ventricular geometry and concentric remodeling groups.
- SII was found to be stronger predictor when compared to NLR and PLR.

# Introduction

Hypertension (HT) is an important public health problem worldwide. Recent data show that approximately 1.4 billion people across the world show the symptoms of high blood pressure, of which only 14% can be controlled [1]. The concept of cardiac remodeling can be defined clinically as caused by both dimensional and functional changes in the heart after injury, and cardiac remodeling directly related

to ventricular dysfunction caused by hypertension is indicative of poor prognosis. It is well known that inflammation, fibrosis and oxidative stress as well as ischemia play significant roles and are the leading pathways in the progress of myocardial remodeling [2]. Since left ventricular hypertrophy (LVH) occurs as an adaptive reaction to increased pressure and volume overload, it can independently lead to worse cardiovascular outcomes, such as stroke, a blockage in coronary arteries, or sudden cardiac death [3, 4]. Thus, it can be argued that hypertensive patients with LVH have a higher cardiovascular risk compared to those without LVH [5].

Low-grade inflammation, predominantly governed by different inflammatory cascades (C-reactive protein [CRP], interleukin [IL]-6, IL-18, etc.) has proven to exert a fundamental influence on the emergence of LVH [6, 7]. All white blood cells and their subtypes with platelets are the essential cells of systemic inflammatory status. As a novel marker of inflammation that includes three types of inflammatory cells (SII; platelet count x neutrophil count/lymphocyte count), the systemic immune-inflammation index (SII) can be used as a marker to predict the clinical course in cancer patients and inflammatory diseases [8]. In studies on cardiovascular diseases, high SII values were detected to pose a negative independent threat for coronary atherosclerotic plaque progression, adverse developments such as congestive heart failure, and in-hospital and long-term disease course in severe coronary syndromes [9–11].

Although SII serves as a comprehensive indicator of inflammation, with a strong predictive ability in revealing diseases that involve the heart and vessels, no research has clearly indicated a relationship between SII and LVH. In the light of the aforementioned data, we tried to determine the relationship between SII and LVH, especially in clients showing hypertensive symptoms, by evaluating the role of SII in diagnosing LVH through the use of platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR).

## Methods

# Study population

Our study encompassed 150 consecutive hypertensive patients who applied to the Cardiology Department of Yozgat City Hospital from June 2021 to January 2022. We conducted a single-center and cross-sectional study, in which those who have systolic blood pressure (SBP) ≥ 140 mmHg and/or a diastolic blood pressure (DBP) ≥ 90 mmHg after three seperate measurements in the sitting position in the office or; those with an average 24-hour ambulatory blood pressure SBP ≥ 130 mmHg and/or DBP ≥ 80 mmHg were considered hypertensive [12].

The patients were recruited taking into consideration the rules mentioned in the Declaration of Helsinki, subsequent to receiving an approval from the Ethics Committee of Yozgat Bozok University, and obtaining a written informed consent from each client.

We excluded the clients who got through any disease related to coronary artery, idiopathic cardiomyopathy (that could cause hypertrophy), those having a body mass index (BMI) over 30 kg/$m^2$, those suffering from active infection or any disease-causing systemic inflammation, congenital heart disease, left ventricular (LV) systolic dysfunction (ejection fraction < 50%), chronic renal failure or chronic liver disease, hematological disorders, drugs that affect coagulation, atrial fibrillation, valvular heart disease, secondary HT.

# Laboratory Measurements

We recorded clinical features of all patients and collected the required demographic data, along with risk factors related to cardiovascular diseases. Routine biochemical tests were conveyed so as to detect the levels of pro-brain natriuretic peptide (pro-BNP). Peripheral venous blood samples were taken from the median cubital vein by atraumatic puncture subsequent to a twelve-hour intermittent fasting.

We used AU 5800 autoanalyzer by Beckman Coulter for measuring kidney function tests, electrolytes, lipid panel, and high sensitivity-CRP (hs-CRP), besides calculating low-density lipoprotein (LDL) with Friedewald's formula. Complete blood count variables were assessed by an automated blood cell counter (Beckman Coulter LH 750; Beckman Coulter Inc., USA) and expressed as x1000 cells/$mm^3$. NLR was calculated by dividing the neutrophil count by the lymphocyte count, PLR was calculated by dividing the platelet count by the lymphocyte count, and SII was calculated by multiplying the NLR ratio by the platelet count, both of which were obtained from the same blood samples.

# Echocardiographic Evaluation

Echocardiographic examination of parasternal and apical views was performed by the same experienced cardiologist on all patients, who were kept in the left lateral decubitus position, considering the guidelines advised by the American Society of Echocardiography [13]. Data were acquired by means of a GE Vivid 7 Ultrasound Machine (Horten, Norway, GE) with a S5-1 (3.5-MHz) transducer, including standart echocardiographic analysis, two-dimensional, M-mode, Doppler flow, and tissue Doppler echocardiography, and three consecutive measurements were made for each parameter. Interventricular septum thickness (IVS), LV chamber dimensions, left atrial (LA) diameter, and LV posterior wall thickness (PWT) were measured on the edges of mitral leaflets by cutting the parasternal long axis vertically with M-mode.

The value of mitral early to late diastolic filling velocity (E/A ratio) was assessed through the apical converter position of the sample volume located between the mitral leaflet tips, in addition to calculating the ratio of E to A (E/A ratio). The early diastolic myocardial (Em) wave velocity was measured from the mitral lateral and septal annulus by using tissue Doppler on sample volume. We followed the guidelines declared by the American Society of Echocardiography so as to calculate the LV mass from M-mode echocardiograpy, and the LV mass index (LVMI, g/$m^2$), which cut-off values of 115 g/$m^2$ for men and 95

g/$m^2$ for women, was obtained through calulating the body surface area. We also measured the LA volume by tracing the LA border from the apical four- and two- chamber views by means of the biplane Simpson's method and used the body surface index to obtain the LA volume index (LAVI, ml/$m^2$).

Relative wall thickness (RWT) was measured as: RWT = 2 × end-diastolic PWT / end-diastolic LV diastolic diameter. ≤ 0.42 values are normal; values > 0.42 were considered as increased RWT [13]. Those with both increased RWT and LVMI values were evaluated to have the risk of concentric hypertrophy; whereas eccentric hypertrophy was observed with those having increased ratios of LVMI together with normal RWT. Values with normal LVMI and increased or normal ratios of RWT were considered to have concentric remodeling or normal geometry, respectively.

# Statistical analysis

We performed the analysis of the study through the SPSS software version 22.0 for Windows (SPSS Inc., Chicago, IL, USA), and determined the distribution patterns of the variables by using Kolmogorov–Smirnov test. We presented categorical variables in numbers and percentage, whereas defining continuous variables as mean ± standard deviation or median, depending on the distribution pattern. We used the Mann–Whitney U-test for the comparison of non-parametric continuous variables, while Fisher's exact tests or Pearson's chi-square were used for categorical variables, which were expressed in percentages. Comparison of multiple means was done through Kruskal-Wallis test or analysis of variance. Probable agents detected in univariate analysis were assessed by logistic regression analysis so as to identify independent indicators of LVH. We used receiver operating characteristic (ROC) curve analysis in order to determine the optimal cut-off value of SII in diagnosing LVH and to evaluate its specificity and sensitivity. We used the DeLong test on MedCalc program in order to evaluate the results of ROC curve analysis relating NLR, PLR and SII. The two-sided p value of < 0.05 was assessed to be statistically significant.

# Results

The research encompassed 150 clients in total, as the targety population was separated into two groups regarding the LVMI, including 56 patients (female:36; mean age: 56.4 ± 10.3 years) with LVH and 94 patients (female:44; mean age: 53.3 ± 8.6 years) with non-LVH. Evaluation of the clinical characteristics and laboratory parameters among patients with LVH and non-LVH are presented in Table 1. The systolic pressure and female patient ratio were determined as significantly high in the group with LVH. The frequency of diabetes mellitus, smoking rate, heart rate, DBP, BMI, and pre-hospital medications demonstrated similar features for all groups (Table 1). No significance could be found among the groups in terms of levels of fasting blood glucose, sodium, urea, creatinine, total cholesterol, potassium, HDL, hemoglobin, and triglyceride. However, levels of white blood cell (WBC), platelet count, neutrophil count, hs-CRP, and pro-BNP were found notably high in patients with LVH, while lymphocyte count and LDL were significantly lower (Table 1).

Comparison of the echocardiographic parameters with the left ventricular hypertrophy is demonstrated in Table 2. Left atrial volume index, RWT, and E/Em ratios were significantly high for the group with LVH. The frequencies of isovolumetric relaxation time (IVRT), deceleration time and E/A ratios were detected to be identical for all groups (Table 2).

The new inflammatory parameters were evaluated for patients with LVH and non-LVH. The NLR [4.66 (3.16-8.2) vs. 2.23 (1.87-3); p<0.001], PLR [165.2 (136.4-245.2) vs. 113.4 (93-149.2); p<0.001], and SII [1288 (969-1935) vs. 543 (424-712); p<0.001] were found significantly high in the group with LVH (Figure 1).

The possible factors that were suggested to be important by the univariate analyses were evaluated through the multivariate logistic regression analysis in order to identify independent predictors of LVH. SII (OR: 1.003, 95%CI: 1.001-1.005; p=0.007) was observed to have an independent relationship with the presence of LVH. Moreover, hs-CRP (OR: 3.588, 95%CI: 1.590-8.098; p=0.002) and SBP (OR: 1.040, 95%CI: 1.012-1.069; p=0.005) were also independent predictors of LVH (Table 3).

As a result of the ROC curve analysis; the cut-off value of 869.5 for SII estimated LVH with a sensitivity of 82.1% and a specificity of 86.2% (AUC: 0.861; 95%CI: 0.792-0.930; p<0.001), the cut-off value of 3.13 for NLR indicated LVH with a sensitivity of 76.8% and a specificity of 79.8% (AUC: 0.825; 95%CI: 0.753-0.897; p<0.001), and the cut-off value of 63.4 for PLR predicted LVH with a sensitivity of 71.4% and a specificity of 73.4% (AUC: 0.753; 95%CI: 0.672-0.834; p<0.001). ROC curve analysis results of NLR, PLR and SII were compared using the DeLong test. The assessment of the ROC curves revealed that AUC of SII was larger than that of NLR (0.861 vs. 0.825, p=0.02) and PLR (0.861 vs. 0.753, p=0.001) (Figure 2).

In order to identify the relationship between SII and LVH, we further divided the patients into 4 different groups on the basis of their left ventricular configurations considering LVMI and RWT as follows: normal geometry, concentric remodeling, eccentric LVH and concentric LVH. The findings revealed that SII was remarkably excessive for the groups with the eccentric LVH and concentric LVH, in comparison to those of groups with the normal left ventricular geometry and concentric remodeling (Figure 3).

# Discussion

As indicated by the findings of our study, SII obtained from complete blood cell subtypes provides relevant information according to LVH in patients with hypertension. The level of SII of the patients with LVH is higher than that of the normal ventricular geometry, and its predictive efficacy is more than those of PLR and NLR; which indicates the presence of the LVH. Furthermore, we detected that SII can be an independent and prominent sign of LVH. Thus, we can sugest that this research is the first attempt to reveal the relationship of SII with LVH in patients with hypertension.

Hypertension is admitted as the widely seen risk factor for cardiovascular disease. Despite extensive studies, the exact mechanism underlying hypertension remains to be elucidated. Although low-grade

inflammation is considered to have connection with the emergence of hypertension, it has not been clarified whether inflammation is a cause or a consequence of hypertension [14–16].

Many studies indicate that immune cell infiltration of the endothelial, renal and central nervous systems, as well as their counterparts of oxidative stress, activated renin-angiotensin mechanism and increased sympathetic tone can considerably cause the emergence of hypertension [17, 18]. Studies have demonstrated that immune-modulatory cells infiltrating the heart, perivascular adipose tissue or kidneys contribute to the dysfunction of these organs and may cause hypertension [19, 20]. Therefore, inflammation participates in many conditions that contribute to the development of high blood pressure. Several studies have shown a positive relationship between hypertension and high WBC count, CRP, and IL-6 levels [21, 22]. In another experimental study, it was performed that mice or rats without functional T and B lymphocytes were protected from experimental hypertension [23, 24]. Many studies targeting blood cells such as lymphocytes, monocytes, and neutrophils as well as targeting inflammation indicate their contribution to vascular remodeling and hypertension [25–27].

Being another sign of chronic low-grade systemic inflammation, C-reactive protein has been used in different populations as an indicator of poor cardiovascular outcomes, beyond traditional risk factors. CRP levels are also usually high in patients with hypertension, and high CRP can be detected before the development of arterial hypertension and may be a predictive factor for the disease [28, 29]. Platelet-to-lymphocyte ratio (PLR) and neutrophil-to-lymphocyte ratio (NLR), which also lead to inflammation, have been clearly hypothesized by recent researches that they can act as strong predictive and prognostic markers for the presence of hypertension. Several cross-sectional studies revealed that PLR and NLR were positively linked by hypertension [30, 31]. The results that we obtained through this study are compatible on a large scale with the findings pointed out by those researches. Likewise, we determined that PLR, NLR and SII were remarkably high in the group with LVH in comparison to the group without LVH.

The presence of inflammatory markers is often associated with other HT-mediated organ damage, particularly LVH, and disseminated atherosclerotic vascular disease. Therefore, understanding the relationships between LVH and low-grade markers of systemic inflammation and endothelial disfunction is clinically and prognostically important [32, 33]. LVH is a hypertensive target organ damage that independently and clearly predicts the risk of cardiovascular disease that leads to mortality and morbidity, thus making the diagnose of LVH significant in terms of studies and clinical practise. Although the pathophysiological mechanisms underlying the progression from LVH to the development of cardiovascular events are still unclear, accelerated atherosclerosis due to systemic inflammation and endothelial dysfunction may be cited as the cause. When examined in this context, studies have shown that the cells of inflammation and immune system are heavily effective in the development of coronary atherosclerosis. While the evaluation of the above-mentioned cells alone gives important results about the development of atherosclerosis, these results have become even more important with the use of some ratios such as PLR, NLR and SII. All these rates are presented as the markers for atherosclerosis and are associated with the prevalence of coronary artery disease (CAD) and poor cardiovascular outcomes.

Recently, it has been suggested that SII, developed by Hu et al, may provide more valuable information about inflammation because it includes 3 cell types [34]. There are studies in the literature demonstrating that PLR, NLR, and SII can be used as new predictors of prognosis and mortality in patients with CAD [11, 35], but studies on the superiority of these indices over each other are limited. In another research conducted by Erdoğan et al, it has been revealed that SII can be a strong predictor of coronary obstruction, considered hemodynamically significant, in comparison to PLR and NLR, and can serve as an independent predictor of coronary artery occlusion, which may cause a heart attack [36]. In another study, we showed that SII can estimate the grade of high thrombus burden in patients more accurately than PLR and NLR [37]. The findings of our study clearly revealed that the predictability of SII in terms of LVH was stronger than NLR and PLR.

LVH is usually identified as the left ventricular mass index, and it occurs subsequent to the increase of chronic blood pressure and volume overload, which results in cardiac fibrosis and cardiomyocyte hypertrophy. Hypertrophic geometric patterns (eccentric hypertrophy, concentric hypertrophy) have close relationship with poor prognosis for patients with hypertension [38]. In a recent research conveyed by Afşin et al, NLR was found to be higher in eccentric and concentric LV geometric patterns [31]. The outcomes of our study are compatible with the findings revealed by the above-mentioned research. Similarly, we determined that SII was higher in the groups with eccentric and concentric LVH, in comparison to those having normal configuration.

The outcomes of our research should be evaluated with some considerations based on the following limitations. Initially, the study was conducted on relatively a small sample, and we did not record any follow-up data because of the cross-sectional design of the study. Secondly, we included only one measurement of admission complete blood count and calculation of SII in the analysis. As with any new prognostic indicator, the normal reference range for SII was not systematically investigated and established in this study. Finally, despite of numerous researches that revealed association of LVH with cytokines, we couldn’t handle cytokines in our research at all.

In conclusion, our study results suggested that SII can independently predict the presence of LVH, which has not been reported previously. This research revealed that SII could be a strong and cost-effective marker which is easily taken from complete blood cell subtypes for judging LVH. The result of our study showed possible links between SII, NLR, PLR, and LVH in clients having hypertension. Additionally, this predictor of SII is stronger than PLR and NLR, which serve as prominent signs of inflammation. SII can assist researchers predict which clients can have high-risk of LVH and provide a new preventive strategy for LVH by modulating hypertension.

# Declarations

**Author contributions** Concept: OK, SGN; design: OK, MCÇ; supervision: OK, SGN, MCÇ; materials: OK, SGN; data collection and/or processing: OK, SGN; analysis and/or interpretation: MCÇ, OK; literature search: OK, MCÇ, SGN; writing: OK, MCÇ; critical review: MCÇ, OK

### Compliance with ethical standards

**Conflict of interest** The authors declare no competing interests.

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# Tables

**Table 1**. Comparison of baseline characteristics and laboratory parameters of the study population

Abbreviations: ACE, angiotensin converting enzyme; ARB, angiotensin receptor blocker; BNP, brain natriuretic peptide; CRP, C-reactive protein; DBP, diastolic blood pressure; HDL, high-density lipoprotein; IQR, interquartile range; LDL, low-density lipoprotein; NLR, neutrophil to lymphocyte ratio; PLR, platelet to

| Variables | LVH group (n:56) | non-LVH group (n:94) | p value |
|---|---|---|---|
| **Baseline characteristics** | | | |
| Age, years, mean ± SD | 56.4 ± 10.3 | 53.3 ± 8.6 | 0.054 |
| Gender (female), n (%) | 36 (64.3) | 44 (46.8) | **0.038** |
| Diabetes Mellitus, n (%) | 13 (23.2) | 17 (18.1) | 0.447 |
| Smoking status, n (%) | 15 (26.8) | 26 (27.7) | 0.951 |
| Heart rate, beats/minute, median (IQR) | 73.5 (70-83.2) | 77.5 (71-83.2) | 0.152 |
| SBP, mmHg, median (IQR) | 148.5 (130-163.7) | 139 (125-150) | **0.006** |
| DBP, mmHg, median (IQR) | 83 (80-91.5) | 80 (74.7-93.5) | 0.188 |
| Body mass index, kg/$m^2$, median (IQR) | 28.3 (26.2-29.6) | 27.7 (25.2-29.5) | 0.228 |
| Prehospital medications, n (%) | | | |
| ACE inhibitor | 25 (44.6) | 32 (34) | 0.196 |
| ARB | 20 (35.7) | 31 (33) | 0.732 |
| Beta blocker | 9 (16.1) | 15 (16) | 0.985 |
| Calcium channel blocker | 23 (41.1) | 29 (30.9) | 0.203 |
| Hydrochlorthiazide | 31(55.4) | 38 (40.4) | 0.076 |
| **Laboratory parameters** | | | |
| Glucose, mg/dl, median (IQR) | 97.5 (90.2-105.7) | 100.1 (87.7-109) | 0.637 |
| Urea, mg/dl, median (IQR) | 31 (25-36.7) | 31 (26-36.2) | 0.853 |
| Creatinine, mg/dl, median (IQR) | 0.77 (0.65-0.93) | 0.78 (0.70-0.92) | 0.829 |
| Sodium, mmol/L, median (IQR) | 140.5 (139-142) | 140 (139-142) | 0.477 |
| Potassium, mmol/L, median (IQR) | 4.4 (4.1-4.6) | 4.4 (4.1-4.7) | 0.977 |
| HDL-C, mg/dl, median (IQR) | 47.9 (38.6-59) | 46 (40.8-54) | 0.504 |
| LDL-C, mg/dl, mean ± SD | 111.8 ± 29.5 | 122 ± 30.7 | **0.049** |
| Triglyceride, mg/dl, median (IQR) | 137 (104-205) | 141 (97-201) | 0.627 |
| Total cholesterol, mg/dl, mean ± SD | 193.4 ± 36 | 200.4 ± 38 | 0.271 |
| WBC count, x$10^3$/μL, median (IQR) | 10 (8.4-12) | 8 (6.9-9.3) | **<0.001** |
| Neutrophil count, x$10^3$/μL, median (IQR) | 7.6 (6-9.4) | 4.8 (3.8-6.2) | **<0.001** |

| | | | |
|---|---|---|---|
| Lymphocyte count, x$10^3$/μL, median (IQR) | 1.55 (1.12-2.37) | 2.1 (1.5-2.62) | **0.004** |
| Hemoglobin, g/dL, median (IQR) | 14 (13.4-15.3) | 14.6 (13.5-15.6) | 0.267 |
| Platelet count, x$10^3$/μL, mean ± SD | 283.6 ± 90.7 | 250.1 ± 71.9 | **0.014** |
| High-sensitivity CRP, mg/L, median (IQR) | 1.05 (0.57-1.54) | 0.6 (0.35-1.19) | **<0.001** |
| Pro-BNP, ng/ml, median (IQR) | 68.7 (37.2-119) | 40 (24-81.2) | **0.006** |
| **Inflammatory Indexes** | | | |
| NLR, median (IQR) | 4.66 (3.16-8.2) | 2.23 (1.87-3) | **<0.001** |
| SII, median (IQR) | 1288 (969-1935) | 543 (424-712) | **<0.001** |
| PLR, median (IQR) | 165.2 (136.4-245.2) | 113.4 (93-149.2) | **<0.001** |

lymphocyte ratio; SBP, systolic blood pressure; SD, standard deviation; SII, systemic inflammatory index; WBC, white blood cell. Bold values means statistically significant.

**Table 2.** Comparison of the echocardiographic parameters of patients in regard to left ventricular hypertrophy

| Variables | LVH group (n:56) | non-LVH group (n:94) | p value |
|---|---|---|---|
| Left atrium volume index, ml/$m^2$, median (IQR) | 19.3 (15.8-22.7) | 14.5 (12.8-17.2) | **<0.001** |
| RWT, g/$m^2$, median (IQR) | 0.47 (0.43-0.51) | 0.42 (0.39-0.46) | **<0.001** |
| Deceleration time, ms, median (IQR) | 232 (204.5-264) | 235.5 (189.2-279.2) | 0.711 |
| IVRT, ms, mean ± SD | 81.6 ± 15.1 | 81.7 ± 13.9 | 0.967 |
| E, cm/s, median (IQR) | 67.2 (59.5-87.4) | 71.7 (58.1-86.6) | 0.947 |
| E/A | 0.85 (0.73-0.98) | 0.92 (0.72-1.14) | 0.175 |
| E/Em | 8.4 (7.2-11) | 7.7 (6.5-9.2) | **0.027** |
| Left ventricular geometry, n (%) | | | |
| Normal geometry | - | 42 (44.7) | |
| Concentric remodelling | - | 52 (55.3) | |
| Eccentric hypertrophy | 9 (16.1) | - | |
| Concentric hypertrophy | 47 (83.9) | - | |

Abbreviations: E, mitral early diastolic wave; 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

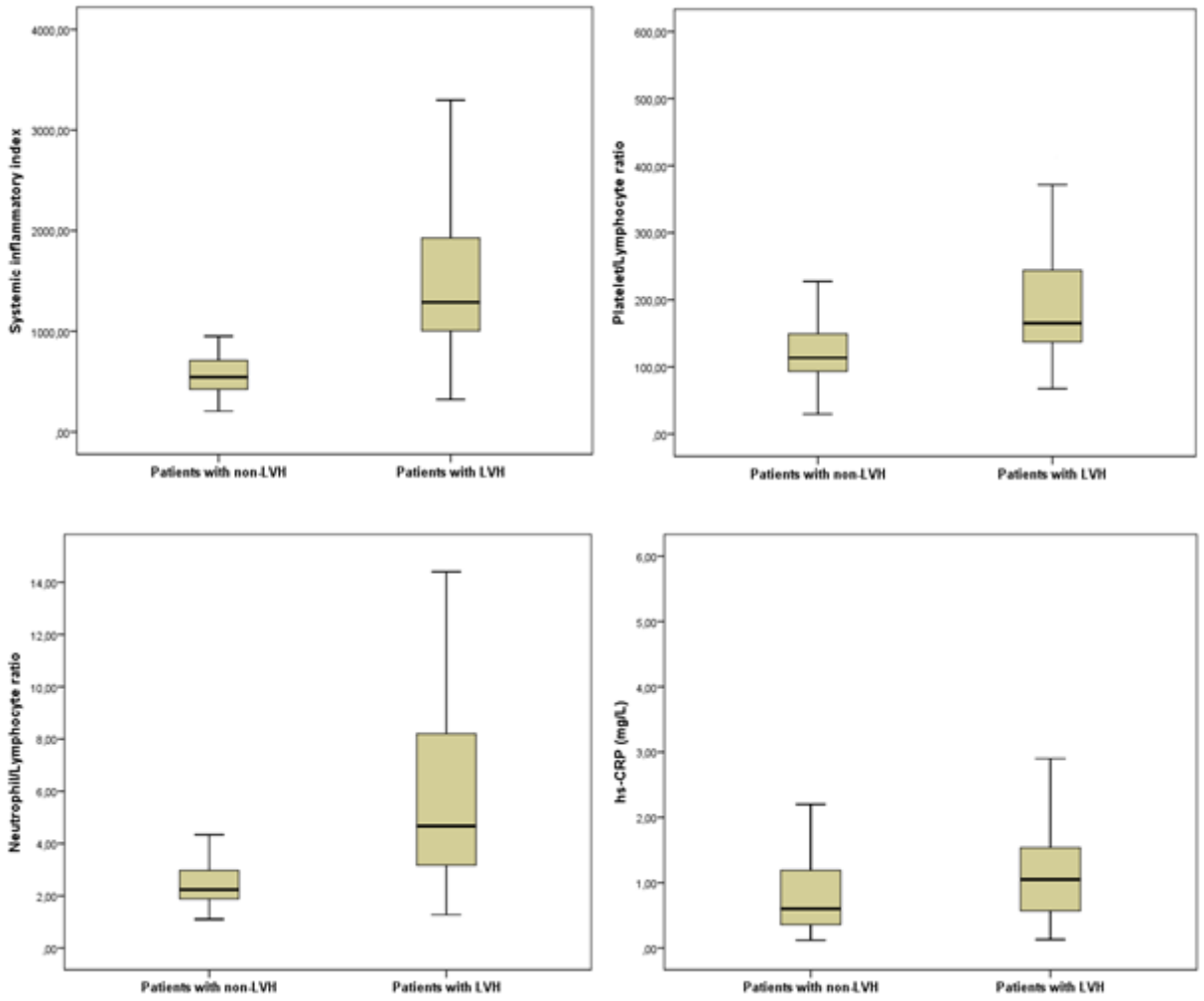
Velocity of Early Diastole; IQR, interquartile range; IVRT, isovolumetric relaxation time; RWT, relative wall thickness; SD, standard deviation. Bold values means statistically significant.

**Table 3.** Univariate and Multivariate Logistic Regression Analysis Showing the Independent Predictors for the Presence of Left Ventricular Hypertrophy

| Variables | Univariate analysis | | Multivariate analysis | |
|---|---|---|---|---|
| | OR ( 95% CI) | p value | OR ( 95% CI) | p value |
| **Age** | 1.036 (0.999-1.074) | 0.056 | 1.009 (0.950-1.072) | 0.764 |
| **SBP** | 1.026 (1.008-1.044) | 0.004 | 1.040 (1.012-1.069) | 0.005 |
| **PLR** | 1.010 (1.005-1.015) | <0.001 | 0.994 (0.983-1.006) | 0.333 |
| **NLR** | 1.902 (1.487-2.433) | <0.001 | 1.168 (0.748-1.824) | 0.494 |
| **SII** | 1.003 (1.002-1.004) | <0.001 | 1.003 (1.001-1.005) | 0.007 |
| **E/Em** | 1.145 (1.017-1.289) | 0.025 | 1.143 (0.944-1.383) | 0.170 |
| **hs-CRP** | 3.160 (1.687-5.918) | <0.001 | 3.588 (1.590-8.098) | 0.002 |
| **Pro-BNP** | 1.005 (1.001-1.009) | 0.016 | 1.006 (0.999-1.013) | 0.073 |

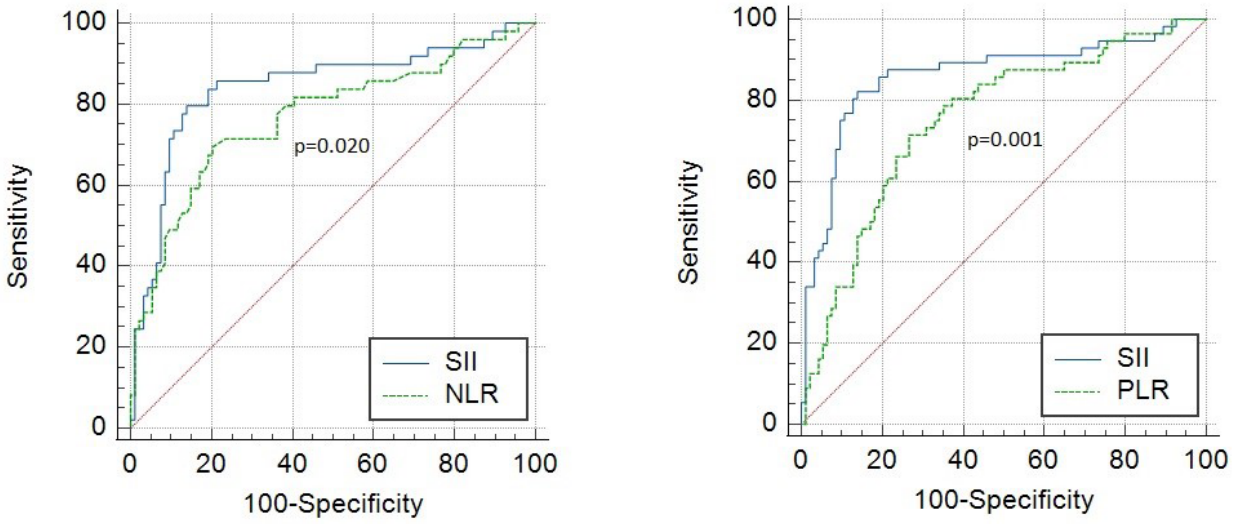
Abbreviations: BNP, brain natriuretic peptide; CI, confidence interval; CRP, c-reactive protein; E/Em, peak velocity of early diastolic flow across mitral valve/myocardial peak velocity of early diastole; NLR, neutrophil to lymphocyte ratio; OR, odds ratio; PLR, platelet to lymphocyte ratio; SII, systemic inflammatory index.

# Figures



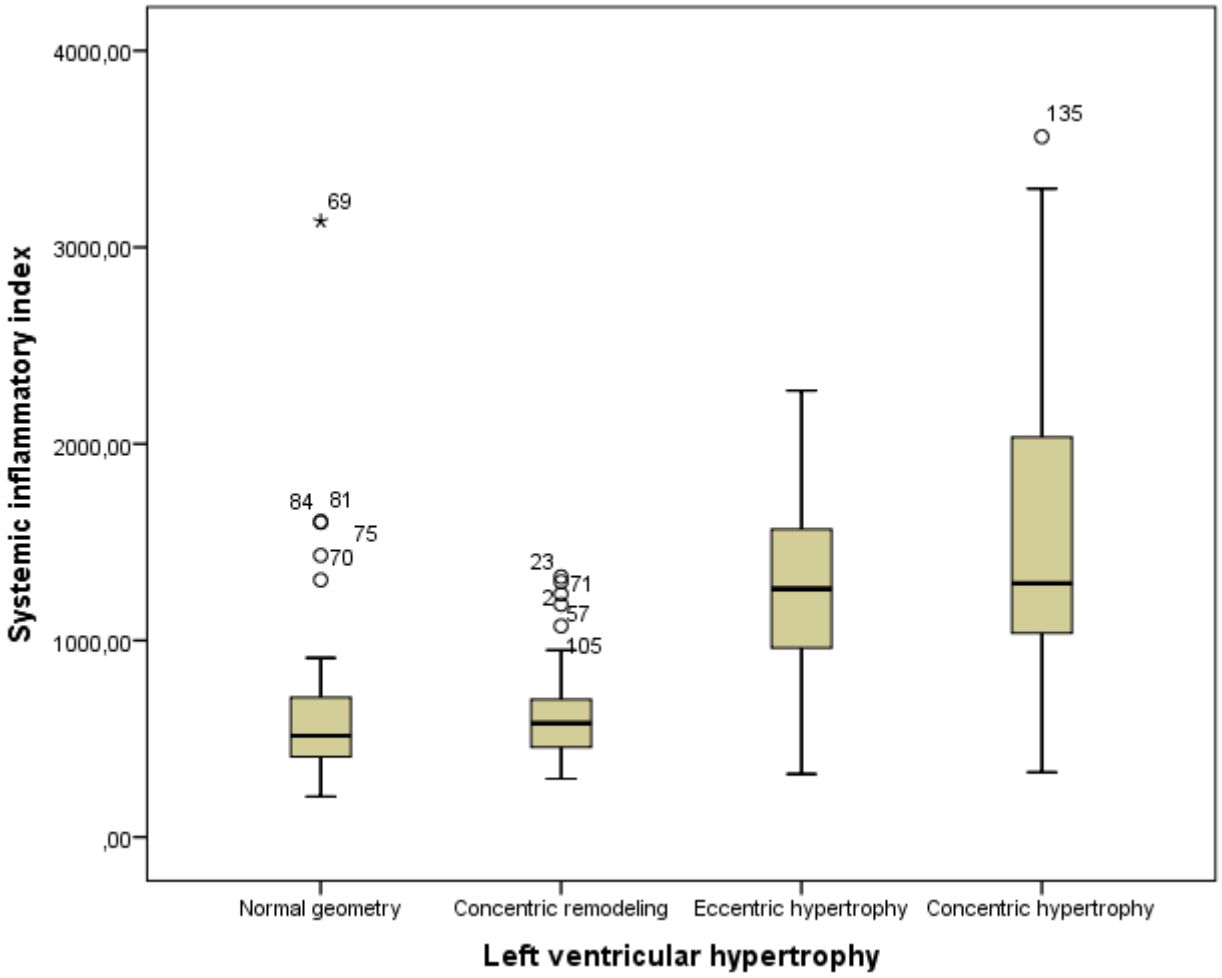

## Figure 1

Comparison of Systemic Immune Inflammation Index, NLR and PLR of the Study Groups




## Figure 2

The Receiver-Operating Characteristics (ROC) Curve Analyses of SII, NLR and PLR for the Identification of Left Ventricular Hypertrophy




## Figure 3

The Relationship Between SII and Different Left Ventricle Geometry Patterns