NLR—A Simple Indicator of Inflammation for the Diagnosis of Left Ventricular Hypertrophy in Patients with Hypertension

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Summary

We aimed to investigate the relationship between neutrophil-to-lymphocyte ratio (NLR), C-reactive protein (CRP), brain natriuretic peptide (BNP), and left ventricular hypertrophy (LVH) in hypertension. Methods: This study included 386 patients with hypertension. Mann-Whitney *U* test and multivariate binary logistic regression analysis were used to investigate the relationship between NLR, CRP, BNP, and LVH in patients with hypertension, as well as compare the levels of NLR, CRP, and BNP in the four configurations. Receiver operator characteristic (ROC) curve was used to compare the diagnostic efficacy of NLR, CRP, and BNP on LVH. Results: The NLR and CRP and BNP levels of the LVH group were significantly higher than those of the non-LVH group. In the multivariate logistic regression analysis, NLR as well as age, BMI, and SBP were associated with LVH. In addition, in patients with eccentric and concentric hypertrophy, the NLR and CRP and BNP levels were higher than those of the normal left ventricular geometry and concentric remodeling groups. The cutoff values of NLR, CRP, and BNP obtained by ROC curve were 2.185, 2.205, and 283.45, respectively, for the prediction of LVH. Conclusions: NLR is independently associated with LVH in patients with hypertension, and this is consistent with the diagnostic efficacy of CRP and BNP, which may be a simple and convenient indicator for judging LVH.

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Key words: CRP, BNP

Hypertension may cause severe organ damage and increase the risk of patient death and its incidence has been increasing year by year.^{1,2)} Left ventricular hypertrophy (LVH) is a common pathological change in hypertension, which is a marker for and contributes to coronary events, heart failure, and cardiovascular mortality.^{3,4)} Inflammation, fibrosis, and oxidative stress as well as ischemia play significant roles and are the leading pathways in left ventricular remodeling.⁵⁾ It is well known that C-reactive protein (CRP), brain natriuretic peptide (BNP), and some inflammatory factors play important roles in the progress of myocardial remodeling. Neutrophil-to-lymphocyte ratio (NLR) is a new inflammation marker in recent years, which is widely used in cardiovascular research.⁶⁻⁸⁾ Neutrophils, as phagocytic cells, are involved in the clearance of pathogens in infectious inflammation, and play an important role in tissue repair and immune regulation in aseptic inflammation. Lymphocytes participate in the long-term response of the immune system. NLR combines two different immune pathways and serves as a comprehensive indicator of inflammation, with a strong predictive ability for cardiovascular disease.

Studies have confirmed that elevated NLR is an independent risk factor of coronary atherosclerotic plaque progression, cardiac death after coronary stent implantation, and long-term mortality of acute coronary syndrome.⁹⁻¹⁴⁾ Although some studies have found that inflammatory cytokines (CRP, interleukin [IL]-6, IL-18, etc.) are associated with LVH,^{15,16)} no study clearly showed a relationship between NLR and LVH. We aimed to assess whether NLR is associated with LVH in patients with hypertension and to compare the diagnostic efficacy of NLR, CRP, and BNP for LVH.

Methods

Study population: This study enrolled a total of 386 patients with hypertension admitted to the Department of Cardiology, Tianjin Medical University from July to September 2015. All enrolled patients signed informed consent forms. Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg in the absence of antihypertensive drugs and when the blood pressure was measured

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three times a day. SBP ≥ 140 mmHg and DBP < 90 mmHg was considered simple systolic hypertension. Patients with a history of hypertension and currently using antihypertensive drugs were also diagnosed as hypertensive even if the blood pressure was lower than 140/90 mmHg. From these subjects, the patients who met the following conditions were excluded: (1) acute and chronic infection; (2) congenital heart disease; (3) severe heart failure; (4) myocardial infarction and cerebrovascular disease recently; (5) blood system diseases; (6) renal dysfunction and hepatic insufficiency; (7) rheumatic immune system diseases; (8) taking glucocorticoids, drugs that affect coagulation. In total, we enrolled 386 patients (aged 64.35 ± 12.436 years, including 201 males and 185 females).

Determination of laboratory indicators: The subjects were fasted for 10 hours and blood samples were drawn from the elbow vein. The blood samples were sent to the Laboratory Center of the General Hospital of Tianjin Medical University for the detection of blood routine item, BNP, creatine kinase, creatine kinase isoenzyme MB (CKMB), alanine aminotransferase, aspartate aminotransferase (AST), total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, creatinine, uric acid, fasting plasma glucose, CRP. All the tests were performed by professional laboratorians of the Tianjin Medical University General Hospital in strict accordance with instructions.

Echocardiography: Echocardiographic examination of the patients was performed by experienced cardiologist according to the guidelines of the American Society of Echocardiography.¹⁷⁾ The echocardiograph measurements included left ventricular internal dimension in end-diastole (LVIDd), interventricular septum thickness (IVST), posterior wall thickness (PWT). Left ventricular mass (LVM) (g) = $1.04 \times [(LVIDd + IVST + PWT)^3 - LVDd^3] - 13.6$ g. Left ventricular mass index (LVMI) (g/m²) = LVM (g) / BSA (m²). Relative wall thickness (RWT) = $2 \times PWT / LVIDd$. LVH is defined as LVMI ≥ 125 g/m² in men and LVMI ≥ 120 g/m² in women.¹⁸⁾ A partition value of 0.45 was used for RWT. All subjects were divided into four configuration groups according to Ganau's classification as follows: normal left ventricular geometry (normal LVMI and RWT), concentric remodeling (normal LVMI and increased RWT), eccentric LVH (increased LVMI and normal RWT), and concentric LVH (increased LVMI and RWT).

Statistical analysis: Data analysis was performed with the SPSS 20.0 software. Continuous variables are expressed as mean $\pm$ SD or quartile, and categorical variables are expressed as percentage. Comparisons among groups were made by chi-squared test for categorical variables. The *t*-test, Kruskal-Wallis test, and one-way ANOVA were used for continuous variables according to the number of groups and the distribution of the data. Multiple linear regression analysis was used to establish the relationship between NLR and LVH after the adjustment for gender, age, BMI, SBP, HR, CKMB, and AST. Receiver operator characteristic (ROC) curves were used to assess the diagnostic performance of NLR, CRP, and BNP. All *P*-values were the results of two-tailed tests. A value of *P* < 0.05 indi-

cated a statistically significant difference.

Results

Baseline characteristics, laboratory findings, and echocardiographic data in LVH and non-LVH groups: Patients were divided into two groups according to the 2010 Chinese guidelines for the management of hypertension, the LVH (190 patients) and non-LVH (196 subjects) groups. Table I shows the general characteristics between the two groups. Patients with LVH had higher CRP, AST, SBP, EF, BNP, CKMB, IVST, PWT, LVM, and LVMI compared with those of the non-LVH group (*P* < 0.05 for all). The NLR level of the LVH group (2.635 [1.898-3.590]) was significantly higher than that of the non-LVH group (2.190 [1.720-3.030]). The CRP level of the LVH group (2.555 [0.8-8.705]) was significantly higher than that of the non-LVH group (1.425 [0.6825-3.97]). The level of BNP of the LVH group (368 [88.325-1181]) was significantly higher than that of the non-LVH group (95.97 [52.91-271.775]) (Figure 1).

Multivariate logistic regression: In order to clarify the independent predictors of LVH, gender, age, BMI, SBP, HR, CKMB, and AST were included in the multivariate logistic regression. The result showed that NLR (odd ratio [OR]: 1.188; 95% confidence interval [CI]: 1.018-1.385; *P* = 0.029), age (OR: 1.023; 95% CI: 1.004-1.041; *P* = 0.016), BMI (OR: 1.075; 95% CI: 1.009-1.144; *P* = 0.024), and SBP (OR: 1.020; 95% CI: 1.009-1.031; *P* < 0.001) were independently associated with LVH (Table II).

NLR, CRP, and BNP levels were significantly increased in the eccentric LVH and concentric LVH groups compared with the normal left ventricular geometry and concentric remodeling groups: In order to clarify the relationship between NLR and LVH, we further divided the subjects into four groups according to their left ventricular configuration on the basis of LVMI and RWT as follows: a normal left ventricular geometry group, a concentric remodeling group, an eccentric LVH group and a concentric LVH group. Table III shows the general characteristics of the four groups. The results showed that NLR, and the levels of CRP and BNP were significantly increased in the eccentric LVH and concentric LVH groups compared with those of the normal left ventricular geometry and concentric remodeling groups (Figure 2).

The analysis of the ROC curve: The NLR cutoff value in the analysis of the ROC curve was 2.185 for predicting LVH (sensitivity: 68.9%; specificity: 50%), and the area under the ROC curve was 59.3% (95% CI: 0.536-0.65; *P* = 0.002). The area under the ROC curve of CRP was 59.9% (95% CI: 0.542-0.655; *P* = 0.001). The cutoff value was 2.205, and the sensitivity and specificity were 55.3% and 61.7%, respectively. The area under the ROC curve of BNP was 68.5% (95% CI: 0.632-0.738; *P* < 0.001). The cutoff value was 283.45, and the sensitivity and specificity were 57.9% and 76%, respectively (Figure 3 and Table IV).

Table I. Clinical Characteristics of the Study Subjects in the Control and LVH Groups ($n = 386$)

	Control group	LVH group	<i>P</i>
<i>n</i>	196	190	
Males, <i>n</i> (%)	101 (51.53)	100 (52.63)	0.829
Age, years	64 (58, 71)	66 (58, 75)	0.057
Diabetes, <i>n</i> (%)	59 (30.1)	48 (25.26)	0.288
Smoking, <i>n</i> (%)	72 (36.73)	82 (43.16)	0.198
Atrial fibrillation, <i>n</i> (%)	27 (13.78)	38 (20)	0.102
HR	72 (64, 80.75)	73.5 (63, 83)	0.731
SBP	140 (121.25, 150)	142 (130, 157.25)	0.003
DBP	80 (75, 90)	80 (71.75, 90)	0.753
BMI, kg/m ²	25.7866 ± 3.63106	26.4108 ± 3.58461	0.09
CK	72 (51, 112)	84.5 (52, 165)	0.073
CKMB	10 (7, 16)	12 (8, 18)	0.021
ALT, U/L	16 (12, 28)	19 (12, 31)	0.244
AST, U/L	17 (13, 28)	20.5 (14, 31)	0.049
TC, mmol/L	4.3923 ± 0.9492	4.4146 ± 1.25981	0.845
TG, median, mmol/L	1.335 (0.9625, 1.87)	1.31 (0.99, 1.8125)	0.955
HDL, mmol/L	1.015 (0.85, 1.2475)	0.99 (0.8275, 1.2225)	0.482
LDL, mmol/L	2.6105 ± 0.80069	2.6958 ± 1.03277	0.366
Scr, mmol/L	70 (60, 80)	71 (60.75, 81.25)	0.464
UA, μmol/L	359.5 ± 110.729	366.66 ± 107.395	0.522
FPG, median, mmol/L	5.25 (4.7, 6.0)	5.25 (4.8, 6.125)	0.506
CRP	1.425 (0.6825, 3.97)	2.555 (0.8, 8.705)	0.001
BNP	95.97 (52.91, 271.775)	368 (88.325, 1181)	< 0.001
NLR	2.19 (1.72, 3.03)	2.635 (1.8975, 3.59)	0.002
EF	63 (60, 64)	60 (51.75, 64)	< 0.001
IVST, mm	10 (9, 11)	11 (10, 12)	< 0.001
PWT, mm	10 (9, 10)	11 (10, 12)	< 0.001
RWT	0.4167 (0.3913, 0.4634)	0.4314 (0.3846, 0.4804)	0.214
LVM, g	187.425 (157.8, 216.19)	253.02 (220.52, 290.72)	< 0.001
LVMI, g/m ²	105.85 (96.0875, 114.4975)	140.735 (131.6225, 157.4175)	< 0.001

Comparisons between groups were analyzed by chi-squared test for enumeration data (males, diabetes, smoking, and atrial fibrillation) and t-test for measurement data (remaining others). Data are mean ± SD or percentage. LVH indicates left ventricular hypertrophy; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; Scr, serum creatinine; BNP, brain natriuretic peptide; CK, creatine kinase; CKMB, creatine kinase isoenzyme MB; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Scr, creatinine; UA, uric acid; FPG, fasting plasma glucose; CRP, C-reactive protein; EF, ejection fraction; LVDD, left ventricular internal dimension in end-diastole; IVST, interventricular septum thickness; PWT, posterior wall thickness; RWT, relative wall thickness; LVM, left ventricular mass; and LVMI, left ventricular mass index.

Figure 1. NLR levels between the non-LVH and LVH groups. * $P < 0.05$ versus non-LVH group.

Table II. Multivariate Logistic Regression of Left Ventricular Hypertrophy in Hypertensive Patients

Variables	B	<i>P</i>	OR	95% CI (lower-upper)	
Gender (1)	-0.047	0.831	0.954	0.618	1.473
Age	0.022	0.016	1.023	1.004	1.041
BMI	0.072	0.024	1.075	1.009	1.144
SBP	0.020	0.000	1.020	1.009	1.031
HR	0.003	0.646	1.003	0.989	1.017
CKMB	0.01	0.055	1.010	1.000	1.019
AST	0.002	0.700	1.002	0.990	1.014
NLR	0.172	0.029	1.188	1.018	1.385

OR indicates odds ratio; CI, confidence interval; BMI, body mass index; SBP, systolic blood pressure; HR, heart rate; CKMB, creatine kinase isoenzyme MB; AST, aspartate aminotransferase; and NLR, neutrophil-to-lymphocyte ratio.

Table III. Clinical Characteristics of the Study Subjects in Left Ventricular Geometry Pattern

	Normal Lvgeometry	Concentric remodeling	Concentric LVH	Eccentric LVH	P
<i>n</i>	133	63	77	113	
Males, <i>n</i> (%)	69 (51.8)	32 (50.79)	41 (53.25)	59 (52.21)	0.993
Age, years	62.27 ± 11.270	64.17 ± 14.995	63.58 ± 13.402	66.83 ± 11.140	0.06
Diabetes, <i>n</i> (%)	40 (30.08)	19 (30.16)	17 (22.08)	31 (27.43)	0.619
Smoking, <i>n</i> (%)	52 (39.10)	20 (31.75)	31 (40.26)	51 (45.13)	0.38
Atrial fibrillation, <i>n</i> (%)	16 (12.03)	11 (17.46)	14 (18.18)	24 (21.24)	0.748
HR	72.73 ± 14.72	76.62 ± 15.572	73.44 ± 11.904	75.02 ± 17.822	0.35
SBP	138.95 ± 20.028	137.02 ± 17.214	149.49 ± 22.088	140.96 ± 18.455	< 0.001
DBP	80 (71.5, 90)	80 (79, 90)	80 (74, 95)	80 (71, 90)	0.393
BMI, kg/m ²	25.6725 ± 3.6389	26.0275 ± 3.63158	26.5073 ± 3.33238	26.3450 ± 3.7651	0.338
CK	68 (52.5, 102)	94 (49, 123)	70 (47.5, 116)	98 (58.5, 226)	0.001
CKMB	10 (7, 16)	10 (8, 16)	9 (7, 14)	14 (9, 22)	< 0.001
ALT, U/L	17 (12.5, 29.5)	15 (11, 24)	16 (12, 25)	19 (13, 35.5)	0.125
AST, U/L	17 (13, 29.5)	18 (13, 22)	17 (13, 25)	23 (15, 45)	0.003
TC, mmol/L	4.3702 ± 0.93080	4.439 ± 0.99292	4.2071 ± 0.94418	4.556 ± 1.42253	0.194
TG, median, mmol/L	1.33 (0.97, 1.89)	1.34 (0.94, 1.87)	1.42 (0.96, 1.91)	1.29 (1.01, 1.78)	0.89
HDL, mmol/L	1.01 (0.845, 1.23)	1.03 (0.86, 1.27)	0.99 (0.83, 1.17)	0.98 (0.82, 1.285)	0.756
LDL, mmol/L	2.5786 ± 0.79121	2.6778 ± 0.82267	2.5131 ± 0.82003	2.8203 ± 1.14238	0.093
Scr, mmol/L	68 (58, 79.5)	70 (60, 80)	71 (58, 85.5)	70 (61, 79)	0.642
UA, μmol/L	359.06 ± 103.923	360.44 ± 124.796	364.03 ± 114.819	368.49 ± 102.423	0.921
FPG, median, mmol/L	5.3 (4.8, 6.0)	5.1 (4.6, 6.3)	5.3 (4.75, 5.85)	5.2 (4.8, 6.4)	0.715
CRP	1.53 (0.705, 4.705)	1.21 (0.60, 3.5)	2.1 (0.67, 4.955)	3.08 (1.125, 10.455)	0.001
BNP	95.94 (54.76, 277.55)	96 (46.06, 257.3)	220.9 (70.155, 616.8)	612.1 (112.95, 1945.5)	< 0.001
NLR	2.23 (1.73, 3.02)	2.04 (1.67, 3.64)	2.60 (1.875, 3.375)	2.66 (1.96, 3.67)	0.014
EF	62 (60, 64)	63 (60, 65)	64 (60.5, 65)	57 (42, 63)	< 0.001
IVST, mm	10 (9, 10)	11 (10, 11)	12 (12, 13)	10 (10, 11)	< 0.001
PWT, mm	10 (9, 10)	11 (10, 11)	12 (12, 13)	10 (10, 11)	< 0.001
RWT	0.4 (0.3774, 0.4176)	0.4783 (0.4651, 0.5106)	0.4898 (0.4783, 0.5217)	0.4 (0.3589, 0.4211)	< 0.001
LVM, g	187.093 ± 38.182	189.624 ± 38.684	261.425 ± 46.922	264.118 ± 60.748	< 0.001
LVMI, g/m ²	105.26 (95.355, 113.89)	106.92 (97.26, 115.33)	145.03 (132.595, 157.985)	139.21 (130.01, 157.31)	< 0.001

Comparisons between groups were analyzed by ANOVA or Kruskal-Wallis test for measurement data and chi-squared test for enumeration data. Data are mean ± SD or percentage. LV indicates left ventricle; LVH, left ventricular hypertrophy; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index; Scr, serum creatinine; BNP, brain natriuretic peptide; CK, creatine kinase; CKMB, creatine kinase isoenzyme MB; ALT, alanine aminotransferase; AST, aspartate aminotransferase; TC, total cholesterol; TG, triglycerides; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; Scr, creatinine; UA, uric acid; FPG, fasting plasma glucose; CRP, C-reactive protein; EF, ejection fraction; LVIDd, left ventricular internal dimension in end-diastole; IVST, interventricular septum thickness; PWT, posterior wall thickness; RWT, relative wall thickness; BSA, body surface area; LVM, left ventricular mass; and LVMI, left ventricular mass index.

Discussion

The findings of the present study demonstrate that a simple ratio (NLR) obtained from blood routine examination provides relevant information regarding LVH in patients with hypertension. The level of NLR of the patients with LVH is higher than that of the normal configuration, and its diagnostic efficacy is similar to those of CRP and BNP; it can be used as a marker to judge the LVH.

In the past several years, it has become increasingly evident that hypertension is an inflammatory process that involves the transmigration and accumulation of both innate and adaptive immune cells into the interstitium of affected tissues, where they release cytokines and promote oxidative stress.¹⁹⁾ The immune system has two major components, the innate and the adaptive systems, which closely interact with each other. The innate immune response includes epithelial cells, which prevent the entrance of pathogens, professional phagocytes (neutrophils and macrophages), the complement system, and pattern recognition receptors. In contrast to the innate immune re-

sponse, the adaptive immune response is designed to respond specifically to foreign antigens. In the case of T-cell activation, antigen-presenting cells degrade foreign proteins to small peptides that are presented by major histocompatibility complex proteins, produce cytokines, and alter the expression of surface receptors that lead to their transferring from secondary lymphoid organs to sites of peripheral inflammation. Studies have shown that angiotensin II (Ang II) and excessive catecholamines lead to the formation of effector-like T cells that infiltrate the perivascular regions of both large arteries and arterioles. There is also accumulation of monocyte/macrophages in these regions. Cytokines released from these cells promote vascular dysfunction and damage, leading to increased systemic vascular resistance. Reactive oxygen species are components of the innate immunity response, which activates proinflammatory transcription factors such as Nrf2 and NF-κB. These, in turn, modulate the expression of genes encoding adhesion molecules and chemokines, and further promote inflammation.²⁰⁻²²⁾ Cardiac stress can induce morphological, structural, and functional changes of

Figure 2. NLR and CRP and BNP levels in the four groups. * $P < 0.05$.

the heart, which is referred to as cardiac remodeling. Sustained overload, as a result of pathological causes by hypertension, results in progressive heart remodeling and finally leads to heart failure.²³⁾ Damage to the vascular endothelium occurs under long-time high impact of blood pressure, leading to the activation of the inflammatory response system, prompting the organizations and organs to produce a large amount of inflammatory cytokines. Inflammatory cells and cytokines participate in the pathological process of cardiovascular remodeling, and they can clear necrotic cells and foreign antigens as well as pro-

mote angiogenesis and scar repair. Inflammatory factors are typically derived from white blood cells, particularly neutrophils.²⁴⁻²⁶⁾

Recent studies have found that the reciprocal interaction between macrophages and T cells in the heart stimulates IFN- γ expression, leading to increased MCP-1 expression in macrophages, which results in a forward-feed recruitment of macrophages, thus contributing to Ang II-induced cardiac inflammation and fibrosis.²⁷⁾ Wu, *et al.* reported that neutrophil-generated S100a8/S100a9 proteins are the key molecules that initiate Ang II-induced cardiac

Figure 3. The receiver operator characteristic (ROC) curve analysis of NLR, CRP, and BNP for LVH.

Table IV. ROC Curve Parameters of NLR, CRP, and BNP

	NLR	CRP	BNP
AUC	59.30%	59.90%	68.50%
95% CI	0.536–0.65	0.542–0.655	0.632–0.738
<i>P</i>	0.002	0.001	0.001
Cut-off	2.185	2.205	283.45

inflammation and fibrosis independently from the high blood pressure response.²⁸⁾ Jiang, *et al.* reported that after infusion of Ang II to mice, elevated blood pressure, neutrophil accumulation, proinflammatory cytokine expression, reactive oxygen species production, and cardiac fibrosis occurred.²⁹⁾ Wei, *et al.* reported that the development of hypertension and LVH in patients with hypertension may be associated with the apoptosis in lymphocytes. The levels of apoptosis of lymphocytes in the hypertensive group (65%) was significantly higher than that of the control group (12.5%). The lymphocyte apoptosis rate of the LVH group (33.1%) was significantly higher than that of the non-LVH group (12.43%; $P < 0.05$).³⁰⁾ Özzbıçer, *et al.* reported that the level of LVMI could be predicted by IL-18 level independently in both the general population and the newly diagnosed patients with hypertension.¹⁵⁾ Salles, *et al.* found that LVH was associated with microalbuminuria and CRP levels.³¹⁾

BNP, a cardiac neurohormone, is released in response

to increased left ventricular wall stretch.¹⁴⁾ BNP is associated with cardiac remodeling in patients with hypertension.³²⁾ CRP, the most investigated cytokine in hypertension, has been repeatedly implicated in both the initiation and progression of the disorder.³³⁾ NLR, an indicator of systemic inflammation, has been proposed as a useful biomarker to predict cardiovascular risk and events.⁶⁾ Karagöz, *et al.* reported that NLR was a predictor of diastolic dysfunction in patients with hypertension.³⁴⁾ Shi, *et al.* reported that white blood cell count may be associated with LVH in patients with hypertension currently taking antihypertensive drugs. NLR may be useful for predicting left VR in patients with ST elevation myocardial infarction after primary percutaneous coronary intervention.¹⁴⁾ Suliman, *et al.* reported that NLR is clearly an independent predictor of all-cause mortality in patients with ACS.³⁵⁾

In conclusion, NLRs were significantly increased in the LVH patients. NLR, as a simple, relatively inexpensive, and universally available test may become the indicator of LVH, which can facilitate the monitoring of cardiac remodeling and taking early treatment measures to reduce the incidence of heart failure.

There were some limitations to our study. The number of samples was relatively small and the conclusions drawn from our study should be tested with a larger sample size. Furthermore, our study is a cross-sectional study that would be more valuable for assessing the relationship between NLR and hypertension if we could have

followed-up the prognosis of these patients. Since previous reports already suggested the importance of inflammatory cytokines, we will make efforts to measure cytokines in the future study. We should also select healthy subjects without hypertension in the future for control.

Disclosure

Conflicts of interest: The authors declare that they have no competing interests.

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