

Investigating the role of age, admission systolic blood pressure, and neutrophil-to-lymphocyte ratio in predicting left ventricular hypertrophy among patients with primary hypertension

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Abstract

Objective: In patients with primary hypertension (PH), left ventricular hypertrophy (LVH) is a critical predictor of cardiovascular events. We aimed to identify clinical and laboratory predictors of LVH in patients with PH.

Methods: This retrospective cohort study included 2321 patients with PH at the Fifth Affiliated Hospital of Xinjiang Medical University from December 2022 to January 2024. Patients were classified into LVH and non-LVH groups; LVH was defined as left ventricular mass index (LVMI) $>115 \text{ g/m}^2$ for men and $>95 \text{ g/m}^2$ for women. Univariate and multivariate logistic analysis were used to identify risk factors for LVH.

Results: Univariate analysis revealed significant differences between the LVH and non-LVH groups in age, sex, smoking, systolic blood pressure (SBP), neutrophil-to-lymphocyte ratio (NLR), body mass index, serum creatinine (SCr), lymphocyte count, and hypertension duration. Multivariate analysis identified age, sex, SBP, SCr, and NLR as independent risk factors for LVH.

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A combined receiver operating characteristic (ROC) model had an area under the ROC curve of 0.711 (95% CI: 0.68–0.74), with 75.3% sensitivity and 59.2% specificity.

Conclusion: Age, sex, SBP, SCr, and NLR were independent predictors for LVH in patients with PH. Our combined diagnostic model provides valuable insight for early LVH screening, aiding timely clinical intervention.

Keywords

Neutrophil-to-lymphocyte ratio, age, sex, serum creatinine, admission systolic blood pressure, primary hypertension, left ventricular hypertrophy

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Introduction

Hypertension is one of the leading risk factors for morbidity and mortality worldwide. With the aging population, the incidence of hypertension in China is gradually increasing.^{1,2} Hypertension is also a common cause of left ventricular hypertrophy (LVH), which is an independent predictor of cardiovascular events. The left ventricular mass index (LVMI) is a crucial indicator for detecting and diagnosing LVH and predicting cardiovascular events.^{3,4}

Many hypertensive patients may initially be asymptomatic but can later develop hypertension-mediated organ damage. Once organ dysfunction occurs, it is difficult to control.⁵ Elevated systolic blood pressure increases myocardial oxygen demand and the likelihood of hypertensive target organ damage, leading to LVH. Early and proactive blood pressure control can reduce or even reverse the occurrence of LVH, thereby improving left ventricular function.^{6,7} Therefore, early detection of LVH is vital for reducing the rate of cardiovascular events.

Many electrocardiographic, clinical, and laboratory variables have been investigated to predict the presence of LVH. For example, electrocardiographic changes, such as the Sokolow–Lyon criteria and Cornell criteria, have been widely used to assess LVH in hypertensive patients. Laboratory markers

like serum creatinine (SCr), B-type natriuretic peptide, and the neutrophil-to-lymphocyte ratio (NLR) have also shown promise in predicting LVH. The use of these variables in conjunction with clinical measures could improve the accuracy of LVH detection and prognosis.^{8–10}

As shown in Figure 1, many primary health care facilities lack echocardiography equipment, and most physical examinations do not include echocardiography. Thus, exploring new indicators for screening and predicting LVH in patients with primary hypertension (PH) is particularly important. Hypertension and advanced age are well-recognized causes of LVH. However, recent research indicates that inflammation leading to endothelial damage plays a crucial role in hypertension-mediated organ damage. Studies have shown that the NLR reflects chronic vascular inflammation and is an independent predictor of acute myocardial infarction, stroke, chronic kidney disease, and other conditions.^{11–14}

The aim of this study was to identify clinical and laboratory predictors of LVH in patients with PH. We investigated the relationship with LVH of age, sex, SCr, admission systolic blood pressure, and NLR in patients with PH. By exploring these factors, we sought to identify convenient new indicators for screening LVH in

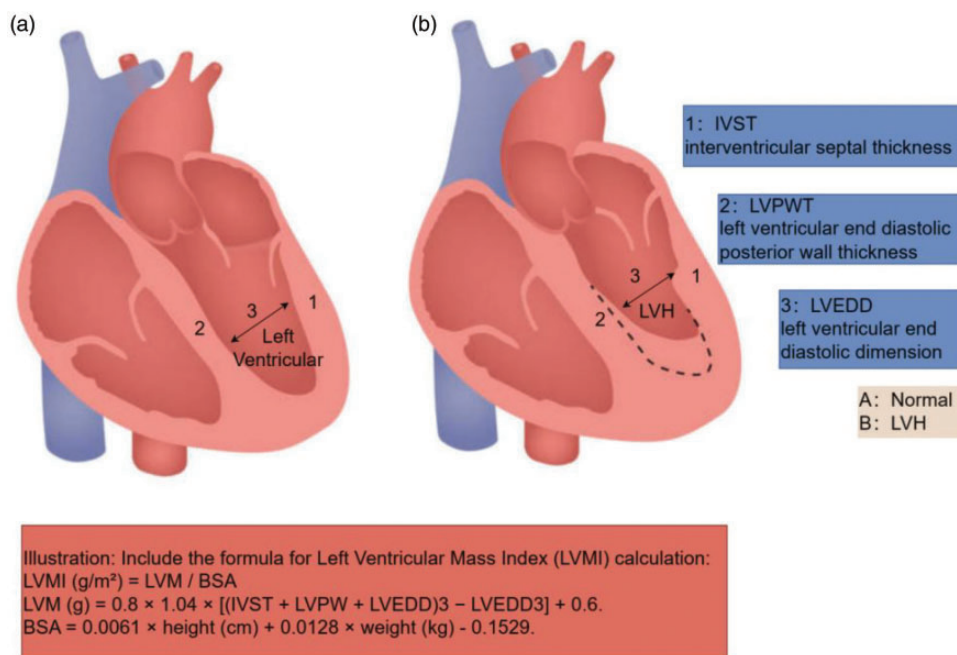


Figure 1. Pathophysiology and diagnostic indicators of left ventricular hypertrophy (LVH) in hypertension. The figure illustrates progression from hypertension to LVH, showing how elevated systolic blood pressure increases myocardial oxygen demand, leading to hypertrophy. The figure also highlights the role of a high neutrophil-to-lymphocyte ratio (NLR) as an indicator of chronic inflammation contributing to LVH. The schematic drawings show normal and hypertrophic left ventricles, highlighting diagnostic indicators.

this population. Our novel approach confirms the relevance of the NLR and enhances predictive accuracy by incorporating additional clinical parameters. By doing so, our research offers a more comprehensive and accessible screening tool, which is particularly valuable in primary health care settings lacking advanced diagnostic equipment.

Methods

Study design

In this retrospective study, we analyzed the clinical data of patients with PH admitted to the Fifth Affiliated Hospital of Xinjiang Medical University between December 2022 and January 2024. The aim of the study was to investigate the relationship with LVH of age, sex, SCr, admission

systolic blood pressure, and the NLR. We also sought to evaluate the screening value for LVH of these parameters in this patient population.

Participants

Patients were consecutively selected based on the inclusion and exclusion criteria outlined below.

Inclusion criteria. The inclusion criteria were patients aged between 30 and 89 years, diagnosed with PH according to the 2018 Chinese Guidelines for the Management of Hypertension,⁴ and currently on at least one antihypertensive medication. Additionally, participants had to be willing to undergo necessary examinations, including echocardiography and complete blood count.

Exclusion criteria. Participants were excluded if they had white coat hypertension or secondary hypertension. Individuals with conditions affecting the NLR, such as hematological diseases, autoimmune diseases, acute and chronic infections, and malignancies, were also excluded. Those with acute cardiovascular and cerebrovascular diseases, including acute myocardial infarction, severe heart failure (New York Heart Association class III or above), cerebral hemorrhage, or cerebral infarction, were not included. Other exclusion criteria were congenital heart disease, cardiomyopathy, valvular heart disease, atrial fibrillation, and an inability to tolerate study-related examinations or refusal to cooperate with study protocols.

Data collection

At admission, we collected patient data including age, sex, duration of hypertension, systolic and diastolic blood pressure, medication history, smoking history (defined as smoking at least one cigarette per day for ≥ 6 months)¹⁵ height, and weight. Body mass index (BMI) was calculated using the formula $BMI = \text{weight (kg)} / \text{height (m)}^2$.

Blood pressure was measured in accordance with the 2018 Chinese Guidelines for the Management of Hypertension.⁴ After resting for at least 5 minutes, seated upper arm blood pressure was measured three times at 5-minute intervals, with the arm positioned at heart level. The average of these three measurements was recorded. All measurements were performed by the same experienced nurse using an electronic blood pressure monitor (Omron (Dalian) Co., Ltd., Dalian City, Liaoning Province, China).

Fasting antecubital venous blood samples were collected the morning after admission. An automated biochemical analyzer was used to measure levels of white blood

cells, lymphocytes, neutrophils, platelets, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, lipoprotein(a), fasting blood glucose (FBG), SCr, blood urea nitrogen, serum uric acid (SUA), and homocysteine (Hcy).

Echocardiography

A professional color Doppler ultrasound diagnostic apparatus (LOGIQ E9; GE, Milwaukee, WI, USA) was used to measure the left ventricular end-diastolic dimension (LVEDD), left ventricular end-diastolic posterior wall thickness (LVPWT), and inter-ventricular septal thickness (IVST). The left ventricular mass (LVM) was calculated using the Devereux correction formula:

$$LVM(g) = 0.8 \times 1.04 \times [(IVST + LVPWT + LVEDD)^3 - LVEDD^3] + 0.6$$

The body surface area (BSA) was calculated as follows: $BSA = 0.0061 \times \text{height (cm)} + 0.0128 \times \text{weight (kg)} - 0.1529$. The LVMI was then calculated as: $LVMI (g/m^2) = LVM / BSA$.¹⁶ These calculations provided standardized measures of LVH, allowing for accurate comparisons across patients with varying body sizes.

Ethical considerations

This study was conducted in strict accordance with the ethical standards outlined in the Declaration of Helsinki (1975), as revised in 2013. Ethical approval was obtained from the Ethics Committee of the Fifth Affiliated Hospital of Xinjiang Medical University (approval number: XYDWFYLSH-2022-06, approval date: January 14, 2022). A copy of the Institutional Review Board approval document has been uploaded as supplementary material.

All patient data were fully anonymized to ensure confidentiality, in compliance with both institutional and national guidelines. Written and verbal Informed consent was obtained from all participants. Institutional protocols for data use and confidentiality were strictly followed throughout the study.

Statistical analysis

Statistical analysis was conducted using IBM SPSS 29.0 (IBM Corp., Armonk, NY, USA). Figures 1 and 2 were generated using Figdraw (Home for Researchers, Hangzhou, China), a paid software platform (Export ID: SOTAW14221 for Figure 1 and WTUYS686b6 for Figure 2), ensuring compliance with licensing and the highest standards of reproducibility.

The data distribution was first assessed using the Shapiro–Wilk test. For normally distributed continuous variables, the mean $\pm$ standard deviation was calculated, and differences between two groups were assessed using independent samples *t*-tests. When comparing more than two groups, one-way analysis of variance was performed, followed by post-hoc Tukey tests to control for multiple comparisons. For non-normally distributed data, the median and interquartile range [P25, P75] were determined, and the Mann–Whitney U test was used for two-group comparisons. For comparisons among more than two groups, the Kruskal–Wallis test was used, followed by Dunn’s post-hoc test to adjust for multiple comparisons. Categorical data were analyzed using the chi-square test or Fisher’s exact test, as appropriate based on

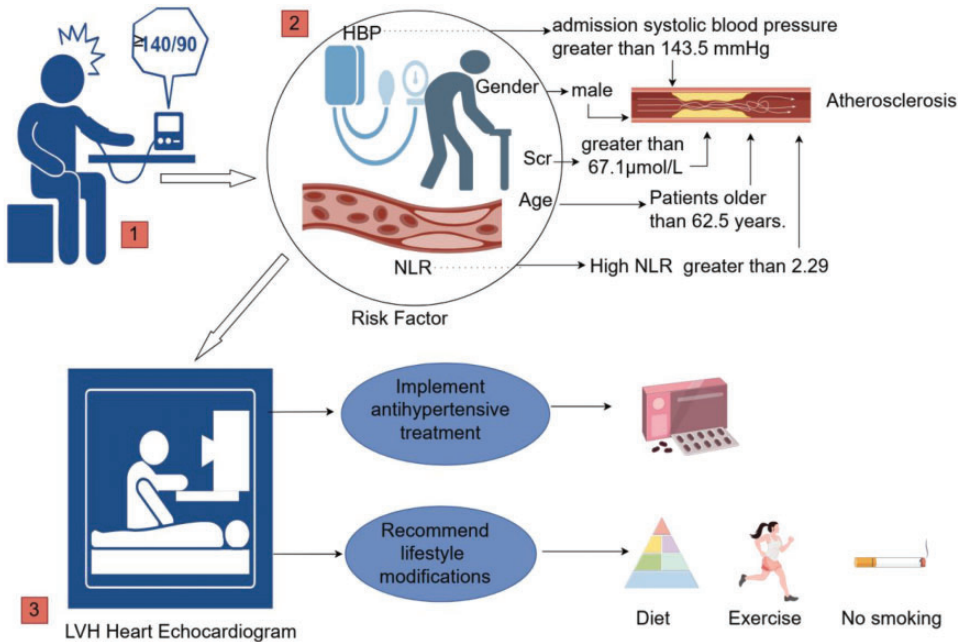


Figure 2. Clinical pathway for hypertension management and left ventricular hypertrophy (LVH) screening. The flowchart outlines the clinical steps for managing hypertension and screening for LVH, including the initial diagnosis of hypertension, risk factor assessment (age, sex, serum creatinine, neutrophil-to-lymphocyte ratio, and systolic blood pressure), echocardiography for LVH detection, and subsequent interventions such as antihypertensive treatment and lifestyle modification.

the expected frequencies in the contingency tables. Correlation analyses were performed to explore the relationships between variables; Pearson's correlation was used for normally distributed variables and Spearman's rank correlation for variables that were not normally distributed.

Multivariable logistic regression analysis was carried out to evaluate the independent effects of age, sex, systolic blood pressure, BMI, SUA, FBG, SCr, lymphocyte count, NLR, hypertension duration, Hcy, and smoking on the risk of developing LVH, adjusting for potential confounders identified in the univariate analysis with a p-value <0.10 . The final model selection was based on the Hosmer–Lemeshow goodness-of-fit test to ensure model adequacy.

The receiver operating characteristic (ROC) curve was used to evaluate the screening value of predictors for LVH in patients with PH. The area under the ROC curve (AUC) was compared using the DeLong test to determine if one model significantly outperformed another. All statistical tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Results

This study included 2321 patients, with an average age of 58.04 ± 11.86 years. Based on their LVMI, patients were categorized into two groups: the LVH group (270 patients, 32% men) and the non-LVH (NLVH) group (2051 patients, 59% men). LVH was defined as LVMI $>115 \text{ g/m}^2$ for men and $>95 \text{ g/m}^2$ for women.

Clinical data of patients with primary hypertension (PH)

The LVH group showed significant differences in age, sex, duration of hypertension, BMI, smoking status, admission systolic blood pressure, SUA, SCr, FBG,

lymphocytes, and NLR, as compared with the NLVH group (all $p < 0.05$), as detailed in Table 1.

Correlation analysis of LVMI and NLR in patients with PH

In the analysis of the LVMI with categorical variables like smoking status and sex, logistic regression was used to ascertain their impact on LVH. This approach was substantiated by classifying the LVMI into clinically relevant categories. The associations were robust, with the findings detailed in Table 2.

Further analysis refined our understanding of the NLR. The revised rho value for the NLR was 0.115, which provided a more accurate reflection of its correlation with the LVMI, maintaining statistical significance ($p < 0.001$). This correlation is depicted in Table 3, highlighting the predictive relevance of the NLR in the context of LVH.

Independent risk factors for LVH in patients with PH

Using LVH as the dependent variable (NLVH = 0, LVH = 1), multivariable binary logistic regression analysis was performed after adjusting for all significant covariates in the univariate analysis ($p < 0.10$), including age, sex, smoking status, SUA, SCr, lymphocyte count, BMI, duration of hypertension, admission systolic blood pressure, FBG, NLR, Hcy, and smoking. A multicollinearity assessment was conducted between SUA and SCr to determine the most suitable variable for inclusion. The results indicated that age, sex, SCr, NLR, and admission systolic blood pressure were independent risk factors for LVH in patients with PH (all $p < 0.05$), as shown in Table 2.

Table 1. Comparison of general data and laboratory test indicators between patient groups.

Variable	NLVH group (n = 2051)	LVH group (n = 270)	$t/Z/\chi^2$	p
Age (years)	57.38 ± 11.60	63.04 ± 12.37	7.13	<0.001
Sex (n, %)			8.46	<0.001
Male	1215 (59.24)	87 (32.22)		
Female	836 (40.76)	183 (67.78)		
BMI (kg/m ²)	26.29 ± 3.74	25.69 ± 3.79	-2.45	0.01
Smoking (n, %)	726 (35.40)	56 (20.66)	-4.81	<0.001
Admission systolic BP (mmHg)	145.09 ± 19.70	151.68 ± 24.26	4.29	<0.001
Admission diastolic BP (mmHg)	86.88 ± 13.801	85.3 ± 16.34	-1.52	0.13
TC (mmol/L)	4.67 ± 1.14	4.78 ± 3.53	0.50	0.62
TG (mmol/L)	1.64 (1.20,2.31)	1.54 (1.12,2.07)	-2.16	0.31
HDL-C (mmol/L)	1.38 ± 0.34	1.41 ± 0.37	1.22	0.22
LDL-C (mmol/L)	2.81 ± 0.85	2.75 ± 0.94	-1.10	0.31
BUN (μmol/L)	5.57 ± 1.64	5.67 ± 1.79	1.00	0.32
SUA (mmol/L)	354.86 ± 89.93	333.49 ± 86.29	-3.69	<0.001
SCr (μmol/L)	70.44 (60.43,80.17)	66.04 (55.12,78.72)	-3.29	<0.001
Lipoprotein(a) (mmol/L)	123.40 (56.71,241.26)	156.57 (55.6,271.47)	-1.44	0.15
Hcy (mmol/L)	16.22 ± 10.38	15.36 ± 6.48	-1.33	0.09
FBG (mmol/L)	5.79 ± 2.55	6.22 ± 4.52	2.36	0.02
WBC (10 ⁹ /L)	6.45 ± 1.73	6.35 ± 1.93	-0.82	0.41
Platelet count (10 ⁹ /L)	231.35 ± 58.07	232.78 ± 61.82	0.38	0.71
Lymphocyte count (10 ⁹ /L)	1.98 (1.59,2.38)	1.80 (1.38,2.37)	-4.02	<0.001
Neutrophil count (10 ⁹ /L)	3.73 ± 1.35	3.86 ± 1.62	1.45	0.21
NLR	2.02 ± 1.09	2.5 ± 2.48	5.57	<0.001
Hypertension duration (years)	4 (1,10)	6 (1,12)	-2.856	0.004

Normally distributed data are expressed as mean ± standard deviation. Non-normally distributed data are expressed as quartiles, with P50 (P25, P75).

BMI, body mass index; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC, total cholesterol; TG, triglycerides; NLR, neutrophil-to-lymphocyte ratio; FBG, fasting blood glucose; SCr, serum creatinine; BUN, blood urea nitrogen; SUA, serum uric acid; WBC, white blood cell; Hcy, homocysteine; BP, blood pressure.

Predictive value for LVH of age, sex, SCr, NLR, and admission systolic blood pressure in patients with PH

The ROC curve analysis of the combined predictors (age, sex, admission systolic blood pressure, NLR, and SCr concentration) demonstrated an AUC of 0.711 (95% CI: 0.68–0.74), with sensitivity of 75.3% and specificity of 59.2%. To rigorously assess whether the combined model provides superior diagnostic performance compared with the individual indicators, we applied the DeLong test for comparing ROC curves. This analysis confirmed that the combined predictive model significantly

outperformed the models based on individual indicators alone in terms of diagnostic accuracy, with a p-value <0.05. These findings are detailed in Table 4, comparing the AUC values, sensitivities, and specificities of each model, thereby demonstrating the statistical and clinical relevance of using the combined indicators for predicting LVH in patients with PH. The detailed clinical pathway for hypertension management and LVH screening is illustrated in Figure 2. The enhanced understanding of individual diagnosis and the effectiveness of the combined model in diagnosing LVH is visually represented in Figure 3 and Table 4.

Table 2. Multivariate binary analysis of LVH in patients with primary hypertension.

Variable	OR	95% CI	p-value
Age	1.024	1.011–1.037	<0.001
Sex	0.293	0.195–0.441	<0.001
BMI	1.004	0.966–1.042	0.856
Admission systolic BP	1.014	1.008–1.021	<0.001
SUA	1.000	0.998–1.002	0.881
FBG	1.021	0.979–1.066	0.335
SCr	1.006	1.001–1.011	0.031
Lymphocyte count	0.959	0.763–1.204	0.733
NLR	1.177	1.086–1.276	0.001
Hypertension duration	1.005	0.989–1.021	0.545
Hcy	0.993	0.977–1.009	0.402
Smoking	1.239	0.803–1.911	0.333

LVH, left ventricular hypertrophy; OR, odds ratio; CI, confidence interval; BMI, body mass index; NLR, neutrophil-to-lymphocyte ratio; FBG, fasting blood glucose; SCr, serum creatinine; SUA, serum uric acid; Hcy, homocysteine; BP, blood pressure.

Table 3. Results of Pearson correlation analysis for LVMI in patients with primary hypertension.

Variable	r	p
Age	0.227	<0.001
BMI	0.430	0.038
Admission systolic BP	0.135	<0.001
SUA	−0.034	0.980
FBG	0.049	0.190
NLR	0.115	<0.001
SCr	−0.003	0.878
Duration of hypertension	0.870	<0.001
Lymphocyte count	−0.104	<0.001

LVMI, left ventricular mass index; BMI, body mass index; NLR, neutrophil-to-lymphocyte ratio; FBG, fasting blood glucose; SCr, serum creatinine; SUA, serum uric acid; BP, blood pressure.

Discussion

In this study, we retrospectively analyzed the clinical data of patients with PH to explore the relationships with LVH of age, sex, SCr, admission systolic blood pressure, and the NLR. We also assessed the

potential screening value of these indicators for LVH in this population. Our findings revealed that age, sex, SCr, admission systolic blood pressure, and NLR were positively correlated with LVH, positioning them as independent risk factors for this condition. These results are particularly relevant in the context of early detection and timely intervention for LVH, which may significantly reduce the cardiovascular risk in hypertensive individuals.

In terms of clinical presentation, patients in the LVH group were older and exhibited significantly higher levels of SCr compared with those in the non-LVH group. Both age and SCr were found to be positively correlated with the LVMI, which is consistent with existing literature on the pathophysiology of LVH in hypertension. Age-related decline in kidney function increases systemic vascular resistance, contributing to a rise in arterial stiffness and elevated cardiac workload. These changes, in turn, exacerbate left ventricular remodeling, leading to the development of LVH. This finding underscores the importance of closely monitoring older patients with hypertension, particularly those with renal dysfunction, who may have a heightened risk for LVH and its associated complications, including heart failure and stroke.^{17,18}

In this study, sex emerged as a significant factor influencing LVH risk. In line with previous studies, our results suggest that older male patients with hypertension, especially those with impaired renal function, are particularly susceptible to LVH. This observation reinforces the need for tailored clinical approaches in managing hypertension, where sex-specific considerations may help refine risk stratification and intervention strategies.

Our study findings also highlighted admission systolic blood pressure as another independent risk factor for LVH. The LVH group showed significantly higher systolic blood pressure upon admission

Table 4. ROC curve analysis of different factors predicting LVH in middle-aged and young patients with primary hypertension.

Variable	AUC	Cut-off value	Sensitivity (%)	Specificity (%)	95% CI
Age	0.628*	62.50	50.90	68.10	0.59–0.66
Sex	0.636*	1.00	32.10	40.80	0.60–0.67
Admission systolic BP	0.578*	143.50	63.80	49.00	0.54–0.62
SCr	0.561*	67.10	46.90	40.70	0.52–0.60
NLR	0.560*	2.29	40.00	73.00	0.52–0.60
Combined diagnosis	0.711	0.10	75.30	59.20	0.68–0.74

* $p < 0.05$ for the difference in AUC (DeLong test) for comparison with combined diagnosis group.

AUC, area under the receiver operating characteristic curve; CI, confidence interval; NLR, neutrophil-to-lymphocyte ratio; SCr, serum creatinine; BP, blood pressure.

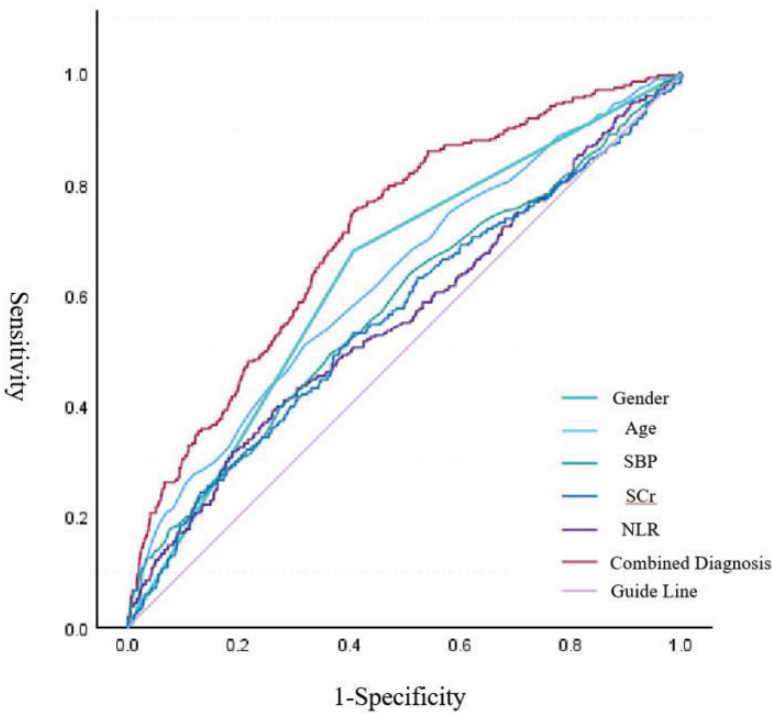


Figure 3. Receiver operating characteristic curves for the diagnosis of left ventricular hypertrophy in patients with primary hypertension, both individually and in combination.

compared with the non-LVH group, with systolic blood pressure being positively correlated with the LVMI. This aligns with the mechanism whereby elevated systolic blood pressure increases myocardial oxygen demand and afterload, driving myocardial hypertrophy and ventricular remodeling.^{19,20}

Our study further supports the notion that early and aggressive blood pressure control can mitigate or even reverse the progression of LVH,^{19,21} thereby reducing the risk of cardiovascular events. This finding emphasizes the critical role of prompt intervention in hypertensive patients with markedly

elevated systolic blood pressure, advocating for tighter blood pressure management strategies to prevent the onset of LVH.

The NLR emerged as a particularly notable predictor of LVH in this study. The NLR, which reflects the systemic inflammatory state, has previously been associated with the occurrence and progression of cardiovascular conditions, including acute myocardial infarction, stroke, chronic kidney disease, and LVH.^{18,22–25} Our analysis revealed that the NLR was significantly higher in the LVH group and positively correlated with the LVMI. Additionally, we found that the LVMI was negatively correlated with lymphocyte count, suggesting that patients with lower lymphocyte counts may have a higher risk of developing LVH. This finding supports the hypothesis that a reduced lymphocyte count may reflect immune system suppression owing to chronic inflammation, which is implicated in the development of LVH. Chronic inflammation plays a crucial role in the pathogenesis of hypertension-induced cardiovascular organ damage, further validating the importance of NLR as a biomarker for LVH. The ability to assess the NLR, as a simple and cost-effective tool in clinical practice, may facilitate early identification of hypertensive patients at high risk for LVH, aiding in the implementation of timely therapeutic interventions.

Several studies have explored the role of inflammatory markers and hematological indices, such as the NLR, in predicting LVH in hypertensive patients. Liu Ya-Nan et al. (2023) demonstrated that the NLR had superior predictive value for LVH compared with other markers, such as the platelet-to-lymphocyte ratio (PLR) and mean platelet volume (MPV), in patients with essential hypertension.²⁶ This finding is consistent with our study results where NLR, along with age and admission systolic blood pressure, emerged as independent predictors of LVH. Similarly, Pavlova et al. (2024)

emphasized the important role of systemic inflammation in the development of LVH, highlighting the contribution of inflammatory indices such as the NLR to the pathogenesis of LVH.²⁵ Our findings align with the results of these studies, further supporting the clinical relevance of the NLR in the context of PH and LVH.

Our study used a novel approach by integrating age, systolic blood pressure, and NLR into a unified predictive model for LVH in hypertensive patients. This model enhances diagnostic accuracy and provides a more comprehensive and practical tool for early detection in primary health care settings where access to advanced diagnostic equipment, such as echocardiography, may be limited. The integration of these readily available clinical parameters allows for an efficient, cost-effective, and scalable screening method for LVH, which could ultimately lead to earlier intervention and improved patient outcomes.

Abdulmecit et al. (2019) confirmed the association between the NLR and LVH, with higher NLR levels found in hypertensive patients with LVH compared with those who did not have LVH.⁸ Furthermore, Yu et al. (2020) identified NLR as an independent predictor of LVH, further validating our findings. However, our study is unique in that we combined the NLR with other critical clinical parameters, such as age and systolic blood pressure, to develop a predictive model that may be more clinically useful than relying on a single biomarker. This comprehensive approach can provide clinicians with a robust tool to better assess LVH risk and guide clinical decision-making.²⁷

Clinical implications

Our study provides a practical and accessible screening tool for LVH in patients with PH, based on a combination of age, sex, SCr, NLR, and admission systolic blood

pressure. This tool is particularly valuable in primary health care settings, where advanced diagnostic tests may not be readily available. By incorporating these easily obtainable clinical indicators, clinicians can more efficiently identify high-risk patients and implement appropriate interventions in a timely manner. The novel approach of combining multiple clinical markers into a unified predictive model further emphasizes the utility of this model in guiding clinical practice, improving diagnostic efficiency, and facilitating early intervention.

After adjusting for confounding factors such as lymphocyte count, BMI, and the duration of hypertension, the factors age, sex, SCr, admission systolic blood pressure, and NLR remained significant independent predictors of LVH. This suggests that these parameters are highly reliable in assessing LVH risk among hypertensive patients. Although other factors such as BMI and the duration of hypertension are important contributors to LVH risk, our findings highlight the particularly strong associations with LVH of age, systolic blood pressure, and NLR, a result that warrants focused clinical attention to these factors in the management of hypertension.

Study limitations and future directions

Although our study provides valuable insights into the prediction of LVH in hypertensive patients, several limitations must be acknowledged. As a retrospective, single-center study, the potential for selection bias and incomplete data may affect the generalizability of our findings. Furthermore, the study population was limited to individuals with PH; thus, the applicability of these findings to other hypertensive subtypes or populations with comorbidities remains unclear. Future multicenter, large-scale prospective studies are needed to validate our findings

and expand their applicability across different patient cohorts and health care settings. Additionally, further research into the predictive performance of NLR in different ethnic and regional populations, as well as its combined use with other inflammatory markers, could refine the present LVH prediction model to provide a more precise risk stratification tool for clinicians.

Conclusion

In this study, we systematically analyzed the relationships of LVH with age, sex, SCr, admission systolic blood pressure, and NLR, proposing a simple and effective screening model with important clinical implications. Our results highlight the importance of incorporating these clinical parameters into routine practice for the early identification and management of LVH in hypertensive patients. The model developed in this study offers a cost-effective alternative to advanced diagnostic tools, which is particularly important in resource-limited settings. Future research should focus on validating this predictive model in larger and more diverse populations, as well as investigating the underlying mechanisms that link these factors to LVH. This will ultimately enhance our ability to better predict, prevent, and treat LVH in patients with hypertension, improving patient outcomes and reducing the burden of cardiovascular disease.

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Author contributions

Conceptualization and Design: Hongjian Li, Xiaoyong Hu; Data Collection: Zhaoying Yang, Rui Tang, Ting Zou; Statistical Analysis

and Interpretation: Xiaoyong Hu; Drafting the Manuscript: Xiaoyong Hu, Djandan Tadum Arthur Vithran; Critical Review and Final Approval: Hongjian Li. All authors have read and approved the final version of the manuscript.

Data availability statement

The datasets generated and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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