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RESEARCH ARTICLE

Correlation between the Hemoglobin, Albumin, Lymphocyte, and Platelet (HALP) Score and Left Ventricular Hypertrophy in Older Patients with Hypertension*

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Abstract

Introduction: Hypertension and left ventricular hypertrophy (LVH) have emerged as significant risk factors for cardiovascular events and all-cause mortality. Inflammation and nutrition play critical roles in the development of hypertension and damage to target organs. The HALP Score, which assesses levels of hemoglobin, albumin, lymphocytes, and platelets, is an index closely associated with inflammation and nutrition, and has been demonstrated to be particularly effective in the older population. Hence, the objective of this study was to examine the correlation between the HALP Score and LVH in older patients with hypertension.

Methods: We collected and retrospectively analyzed data from 234 older patients, including clinical data, and routine blood, liver function, kidney function, and cardiac ultrasound parameters. All patients were categorized into a non-left ventricular hypertrophy (NLVH) group (n = 131) or an LVH group (n = 103). The association between the HALP Score and LVH was investigated, and potential influencing factors were considered.

Results: The LVH group had a significantly lower HALP Score than the NLVH group. Logistic regression analysis revealed that a lower HALP Score and female sex were independent factors associated with LVH in older patients with hypertension (OR = 0.944, 9.962, 95% CI: 0.910–0.979, 3.866–24.300, P = 0.002, <0.001). The area under the curve for the HALP Score in diagnosing LVH in older patients with hypertension was 0.708 (95% CI: 0.641–0.776, P = 0.002).

Conclusion: The HALP Score is significantly associated with LVH in older patients with hypertension: lower scores indicate a greater likelihood of LVH. The HALP Score has moderate diagnostic value for LVH in this population.

Keywords: HALP score; Left ventricular hypertrophy; hypertension in older adults

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Introduction

Hypertension is a significant risk factor for morbidity and mortality associated with cardiovascular diseases worldwide. The global burden of hypertension

is estimated to affect 874 million people, and approximately 9.4 million deaths from cardiovascular disease occur each year [1]. As the diagnostic criteria for hypertension have relaxed, the prevalence rate of hypertension has increased from 32% to 45.4% in the United States, and has doubled in China [2]. Chronic hypertension can result in elevated cardiac afterload, which can lead to cardiomyocyte remodeling and hypertrophy, and ultimately to the development of left ventricular hypertrophy (LVH). The presence of LVH in patients with hypertension significantly increases the risk of cardiovascular disease; thus, LVH is a major risk factor for cardiovascular events and all-cause mortality [3]. Although numerous studies have investigated the pathogenesis of hypertension and its associated target organ damage, the precise mechanism remains unclear. Previous research has indicated that both inflammation and nutrition play major roles in the development of hypertension and LVH. Masiha [4] has reported associations among CRP, E-selectin, P-selectin, interleukin, and myocardial hypertrophy in patients with hypertension. Yu et al. [5] and Karayiğit et al. [6] have discovered that novel inflammatory indicators, such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic immune-inflammation index (SII), have substantial implications in the occurrence and progression of myocardial hypertrophy. In the context of chronic persistent inflammation, an elevated lymphocyte apoptosis rate results in a compromised immune system and subsequently contributes to perpetuation of inflammation [7]. Furthermore, sustained inflammation stimulates megakaryocyte proliferation, thereby increasing platelet production. Elevated platelet count is associated with an elevated risk of atherosclerosis and subsequent mortality [8]. Various nutritional parameters, including albumin [9], hemoglobin [10, 11], and the prognostic nutritional index (PNI) [12], have been identified to be closely associated with the incidence of cardiovascular adverse events.

The hemoglobin, albumin, lymphocyte, and platelet (HALP) Score has recently emerged as a convenient and accessible tool for assessing both systemic inflammation and nutritional status [13]. By incorporating measurements of hemoglobin, albumin, lymphocyte, and platelet counts, the HALP Score provides a comprehensive assessment of these factors. Notably, this scoring system has shown significant effectiveness, particularly in older patients. Previous studies have used the HALP Score to evaluate the prognosis of patients with conditions such as gastric cancer, bladder cancer, esophageal cancer, and stroke [14–17]. However, limited research is available on the cardiovascular applications of the HALP Score. Kocaoglu et al. [18] have used the modified HALP Score as an indicator of prognosis in patients with heart failure; however, the HALP Score itself was not found to be associated with heart failure prognosis in that study. To date, studies examining the correlation between the HALP Score and LVH in older patients with hypertension are lacking. Thus, the objective of this study was to investigate the potential association between the HALP Score and LVH in this specific population. Additionally, the diagnostic value of the HALP Score in identifying LVH was assessed in comparison with other indicators such as NLR, PLR, PNI, and SII.

Methods

Study Population

This was a retrospective cross-sectional study conducted at a single center. A total of 234 older patients diagnosed with essential hypertension, who were admitted to Hebei General Hospital between December 2021 and November 2022, were included in the study. The cohort consisted of 110 men and 124 women with a mean age of 72 years. All patients were categorized into an LVH group or an NLVH group on the basis of their left ventricular mass index (LVMI). LVH was defined as LVMI $\geq$115 g/m^2 for men and $\geq$95 g/m^2 for women [3]. The inclusion criteria for this study were patients 65 years of age or older with essential hypertension, who had undergone cardiac ultrasound and relevant laboratory tests. The diagnostic criteria for hypertension were based on the Chinese Guidelines for Hypertension Management in older people, 2019 [19]. Patients were excluded if they had acute hypertension, white coat hypertension, secondary hypertension (ruled out by relevant laboratory tests), diabetes mellitus, heart failure, acute coronary syndrome, prior myocardial infarction, hematological

diseases, tumors, acute or chronic infections, thyroid dysfunction, primary kidney disease, autoimmune disease, or major abnormalities in liver and kidney function.

All experimental procedures were approved by the Ethics Committee of Hebei General Hospital (clinical ethics approval number 2019-27). The study was conducted in accordance with the Declaration of Helsinki and with the Good Clinical Practice guidelines defined by the International Conference on Harmonisation. All patients provided written informed consent before enrollment.

General Clinical Data Collection

We collected and recorded the general characteristics of the participants, including age, sex, body mass index (BMI), admission systolic blood pressure, admission diastolic blood pressure, duration of hypertension, medications, and previous history of cerebrovascular disease, by accessing electronic medical records.

Laboratory Related Examination

After patients had fasted 12 hours, professional nurses collected 6 mL of venous blood from the arms of the patients. The blood samples were then analyzed by professional laboratory technicians using an American Beckman Counter AU5800 automatic biochemical analyzer to measure the levels of albumin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), fasting blood glucose, potassium ion concentration, sodium ion concentration, urea, creatinine, total cholesterol (TC), triglyceride (TG), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C). Additionally, total white blood cell (WBC) counts, neutrophil counts, lymphocyte counts, platelet counts, and hemoglobin levels were measured with a Sysmex xn-3000 hematology analyzer.

NLR = neutrophil counts/lymphocyte counts
PLR = platelet counts/lymphocyte counts
SII = neutrophil counts × platelet counts/lymphocyte counts
HALP Score = hemoglobin × albumin × lymphocyte counts/platelet counts
PNI = albumin + 5 × lymphocyte counts

Echocardiography

The patients were placed in the left supine positions, and a color Doppler ultrasonic diagnostic instrument (American PHILIPS EPIQ7C) with a probe frequency of 2–4 MHz was operated by professional ultrasound physicians. The ultrasound examination was conducted while the patients were in a calm breathing state, to observe the cardiac morphology and structure in various sections. Measurements were taken according to the American Society of Echocardiography guidelines [20], and included left atrial diameter (LAD), aortic root diameter (AO), interventricular septum thickness (IVST), left ventricular posterior wall thickness (LVPWT), right atrial diameter, right ventricular diameter, left ventricular end-systolic diameter (LVESD), left ventricular end-diastolic diameter (LVEDD), e′ peak, E peak, A peak, and other indicators. The left ventricular ejection fraction (LVEF) was calculated with the LVEDD and LVESD, and the left ventricular mass (LVM) and LVMI were calculated with the Devereux formula [21].

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

$$\text{Body surface area } (m^2) = 0.0061 \times \text{height (cm)} + 0.0128 \times \text{body mass (kg)} - 0.1529$$

$$\text{LVMI } (g/m^2) = \text{LVM (g)}/\text{body surface area } (m^2)$$

$$\text{Relative wall thickness (RWT)} = (2 \times \text{LVPWT})/\text{LVEDD}$$

Statistical Methods

IBM SPSS 26.0 statistical software was used for analysis. The measurement data that followed a normal distribution in both groups are presented as mean ± standard deviation (SD), and independent samples t-test was used to compare the groups. For measurement data that did not follow a normal distribution in both groups, the median and interquartile range (IQR) were used, and comparisons between groups were conducted with the Mann–Whitney U test. Categorical data are expressed as percentages, and the chi-square test was used for comparisons between groups. Logistic regression analysis was used to identify independent risk factors for LVH in older patients with hypertension. Receiver operating characteristic (ROC) curve

analysis was performed to assess the predictive value of the HALP Score in older patients with LVH. Delong's test was used to compare the diagnostic performance of the HALP Score and PLR in the diagnosis of LVH in older patients with hypertension. A two-sided $P < 0.05$ was considered statistically significant.

Results

Demographic Characteristics and Laboratory Parameters of LVH and NLVH

The basic data and corresponding laboratory test results of the LVH and NLVH groups are shown in Table 1. The LVH group exhibited a significantly higher systolic blood pressure and greater proportion of female patients than the NLVH group ($P < 0.001$). Both groups showed similar characteristics in terms of cerebrovascular disease, diastolic blood pressure, hypertension duration, BMI, and medication intake ($P > 0.05$). No significant differences were observed between groups in albumin, AST, ALT, fasting blood glucose, potassium ion, urea, TC, TG, LDL-C, and lymphocyte counts ($P > 0.05$). However, the LVH group had higher platelet counts, and lower WBC counts, neutrophil counts, and hemoglobin levels ($P < 0.05$). No significant differences were observed in NLR, SII, and PNI between groups. In contrast, the PLR in the LVH group was higher than that in the NLVH group [139.1 (107.0, 181.3) vs 121.9 (98.8, 144.0), $P < 0.001$], and the HALP Score was significantly lower [34.4 (26.9, 45.1) vs 43.6 (35.5, 54.1), $P < 0.001$]. Figure 1 provides a visual representation of these findings.

The comparison of echocardiographic parameters between groups is shown in Table 2. LVMI, IVST, LVPWT, RWT, and E/e′ ratios were significantly higher in the LVH group, whereas e′ was significantly lower ($P < 0.05$). No significant differences were observed in the other indicators.

On the basis of the HALP Score, the patients were divided into two groups: T1 (HALP Score ≤39.4) and T2 (HALP Score >39.4). The distribution of LVH patients vs NLVH patients is depicted in Figure 2.

Relationship between HALP Score and LVH in Older Patients with Hypertension

Multivariate logistic regression analysis was conducted to assess the potential risk factors for LVH, taking into account the statistically significant factors identified in the univariate analysis. After controlling for confounding factors, the HALP Score remained an independent risk factor for LVH in older patients with essential hypertension (unadjusted: OR = 0.942, 95% CI = 0.920–0.964, $P < 0.001$; adjusted: OR = 0.944, 95% CI = 0.910–0.979, $P = 0.002$). Furthermore, female sex was identified as a risk factor for LVH in this population. These findings are presented in Table 3.

ROC curve analysis indicated that the HALP Score had a cutoff value of 34.83 for estimating LVH, with a sensitivity of 78.6% and specificity of 55.3% (AUC: 0.708; 95% CI: 0.641–0.776; $P < 0.001$). In contrast, the cutoff value for PLR in determining LVH was 165.88, with a sensitivity of 35% and specificity of 91.6% (AUC: 0.646; 95% CI: 0.574–0.718; $P < 0.001$). These results are illustrated in Figure 3.

We conducted a Delong test to assess the diagnostic performance of the HALP Score vs the PLR in detecting LVH. Delong's test indicated that the HALP Score was significantly more effective than the PLR in diagnosing LVH (95% CI: 0.027–0.098, $P < 0.001$).

Discussion

To our knowledge, this study is the first to investigate the relationship between the HALP Score and LVH in older patients with hypertension. A low HALP Score in older patients with primary hypertension was associated with early development of LVH, and was found to be an independent risk factor for early LVH in this population. The HALP Score in the NLVH group was higher than that in the LVH group, and its diagnostic value for LVH was superior to that of PLR.

Prolonged hypertension can result in hypertensive heart disease, characterized primarily by LVH, left ventricular diastolic dysfunction, and other alterations in cardiac structure and function. LVH, the most prevalent cardiac injury associated with target

Table 1 Demographic Characteristics and Laboratory Parameters of LVH and NLVH.

Variables	NLVH group (n = 131)	LVH group (n = 103)	P value
Age, years, (median, IQR)	70 (67, 76)	70 (67, 75)	0.992
Gender (female), n (%)	35 (26.7)	89 (86.4)	<0.001
BMI, kg/m^2, (median, IQR)	25.8 (23.0, 27.8)	25.91 (23.4, 28.0)	0.750
Cerebrovascular disease, n (%)	64 (48.9)	52 (50.5%)	0.804
SBP, mmHg, (mean ± SD)	144.2 ± 19.5	149.7 ± 18.9	0.029
DBP, mmHg, (mean ± SD)	82.7 ± 11.6	81.6 ± 11.6	0.457
Hypertension duration, n (%)			
≤Five years	27 (20.6)	23 (22.3)	
Five years-ten years	31 (23.7)	19 (18.4)	0.734
Ten years-twenty years	36 (27.5)	27 (26.2)	
>Twenty years	37 (28.2)	34 (33.1)	
Antithrombotic, n (%)	28 (21.4)	18 (17.5)	0.456
Statins, n (%)	27 (20.6)	18 (17.5)	0.546
Hypotensor, n (%)			
ACEI or ARB	30 (23.07)	23 (22.3)	0.918
Beta-blocker	15 (11.5)	7 (6.8)	0.226
Calcium channel blocker	78 (59.5)	62 (60.2)	0.920
Diuretic	8 (6.1)	7 (6.8)	0.831
Laboratory parameters			
Albumin, g/L, (mean ± SD)	38.6 ± 3.0	38.7 ± 3.3	0.793
ALT, U/L, (median, IQR)	15.8 (11.6, 22.0)	14.3 (11, 20.6)	0.194
AST, U/L, (median, IQR)	20.1 (17.6, 24.0)	20.9 (17.5, 25.6)	0.455
Glucose, mmol/L, (mean ± SD)	4.9 ± 0.6	5.0 ± 0.6	0.167
Potassium, mmol/L, (mean ± SD)	3.8 ± 0.4	3.8 ± 0.4	0.228
Sodium, mmol/L, (median, IQR)	141 (139, 142)	142 (140, 143)	<0.001
Urea, mmol/L, (median, IQR)	4.9 (4.2, 5.9)	4.8 (4.0, 5.9)	0.347
Creatinine, umol/L, (median, IQR)	73.4 (64.1, 83.0)	61.8 (56.3, 69.3)	<0.001
TC, mmol/L, (median, IQR)	4.4 (3.5, 5.3)	4.7 (3.9, 5.4)	0.124
TG, mmol/L, (median, IQR)	1.1 (0.8, 1.6)	1.2 (0.9, 1.6)	0.497
HDL-C, mmol/L, (median, IQR)	1.1 (1.0, 1.3)	1.2 (1.0, 1.4)	0.005
LDL-C, mmol/L, (mean ± SD)	2.8 ± 0.8	3.0 ± 0.8	0.170
WBC count, ×10^9 /L, (median, IQR)	6.1 (5.4, 7.0)	5.4 (4.7, 6.2)	<0.001
Neutrophil count, ×10^9 /L, (median, IQR)	3.7 (3.1, 4.5)	3.2 (2.7, 4.0)	0.001
Lymphocyte count, ×10^9/L, (median, IQR)	1.8 (1.5, 2.1)	1.7 (1.3, 2.1)	0.142
Hemoglobin, g/L, (median, IQR)	137 (129, 148)	127 (119, 136)	<0.001
Platelet count, ×10^9/L, (mean ± SD)	207 (175, 244)	234 (204, 269)	<0.001
HALP Score, (median, IQR)	43.6 (35.5, 54.1)	34.4 (26.9, 45.1)	<0.001
PLR, (median, IQR)	121.9 (98.8, 144.0)	139.1 (107.0, 181.3)	<0.001
NLR, (median, IQR)	2.1 (1.6, 2.8)	1.9 (1.5, 2.7)	0.217
SII, (median, IQR)	452.9 (328.4, 573.1)	451.2 (323.9, 631.0)	0.399
PNI, (median, IQR)	47.6 (44.8, 50.1)	47.2 (45.0, 49.1)	0.609

LVH: left ventricular hypertrophy; NLVH: non-left ventricular hypertrophy; BMI: body mass index; SBP: systolic blood pressure; DBP: diastolic blood pressure; ACEI: angiotensin-converting enzyme inhibitors; ARB: angiotensin receptor blockers; ALT: alanine aminotransferase; AST: aspartate aminotransferase; HDL-C: high-density lipoprotein cholesterol; LDL-C: low-density lipoprotein cholesterol; TC: total cholesterol; TG: triglyceride; WBC: white blood cell; PLR: platelet-to-lymphocyte ratio; NLR: neutrophil-to-lymphocyte ratio; SII: systemic immune-inflammation index; PNI: prognostic nutritional index.

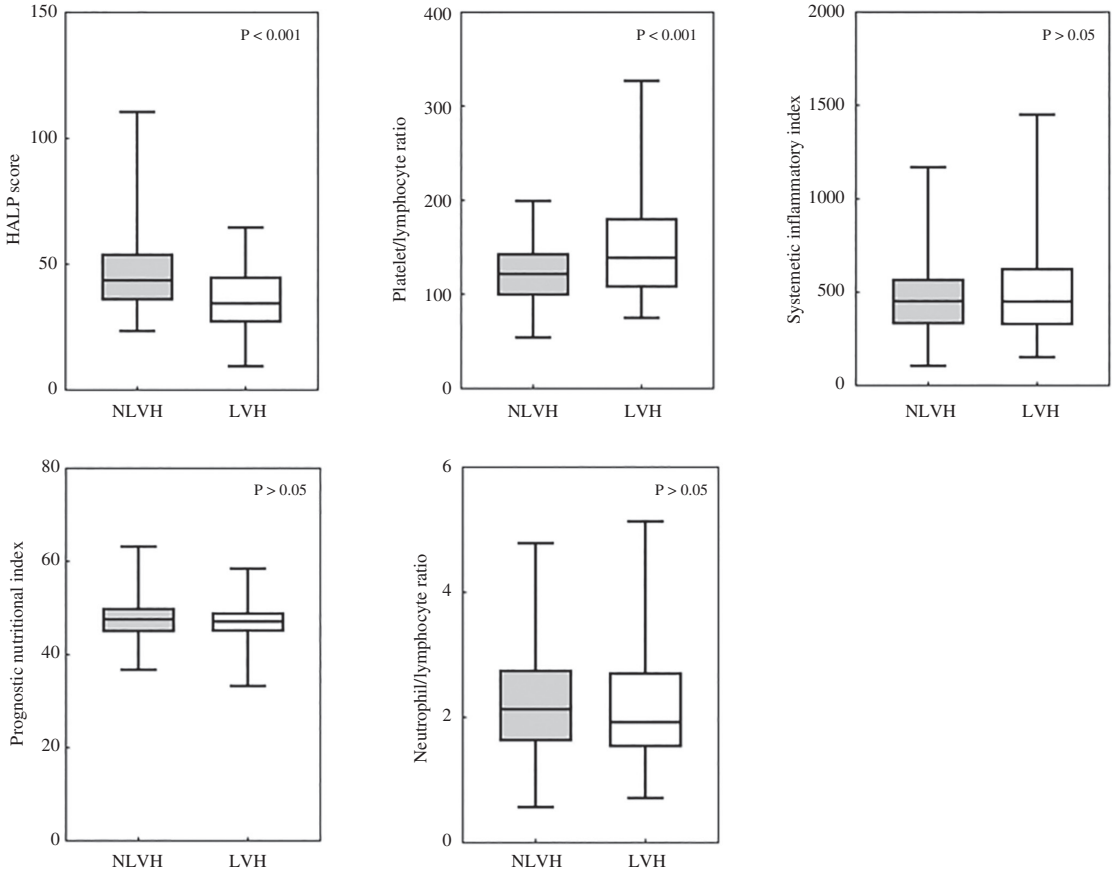

Figure 1 Comparison of the HALP Score, PLR, SII, PNI, and NLR between Groups.
The HALP Scores were higher in patients with NLVH than LVH, and the PLR was lower in patients with NLVH ($P < 0.001$).

Table 2 Comparison of Echocardiography Parameters between the LVH and NLVH Groups.

Variables	NLVH group (n = 131)	LVH group (n = 103)	P value
LVMI, g/m^2, (median, IQR)	82.9 (74.6, 95.2)	99.0 (93.3, 115.5)	<0.001
LAD, mm, (median, IQR)	38 (36, 41)	38 (36, 41)	0.26
IVST, mm, (median, IQR)	9 (9, 10)	10 (10, 11)	<0.001
RWT, mm, (mean ± SD)	0.41 ± 0.04	0.43 ± 0.04	0.001
LVEF, (median, IQR)	66 (63, 68)	64 (62, 68)	0.162
E, m/s, (median, IQR)	0.7 (0.6, 0.8)	0.7 (0.5, 0.8)	0.734
E/A, (median, IQR)	0.7 (0.6, 0.8)	0.7 (0.6, 0.8)	0.181
E/e′, (median, IQR)	11.1 (8.8, 12.8)	12.0 (9.3, 15.3)	0.004
e′, cm/s, (median, IQR)	6.0 (5.2, 7.5)	5.6 (4.5, 7.0)	0.012
LVESD, mm, (median, IQR)	29 (27, 32)	30 (28, 32)	0.071
LVEDD, mm, (median, IQR)	46 (44, 49)	47 (45, 49)	0.236

LVH: left ventricular hypertrophy; NLVH: non-left ventricular hypertrophy; LVMI: left ventricular mass index; LAD: left atrial diameter; IVST: interventricular septum thickness; RWT: relative wall thickness; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-systolic diameter; LVEDD: left ventricular end-diastolic diameter.

organ damage in hypertension, occurs because of remodeling and hypertrophy of myocardial cell [22]. LVH has independent predictive value for cardiovascular disease outcomes, thus highlighting the importance of LVH diagnosis in both research and clinical practice. Currently, the diagnosis of LVH relies primarily on techniques such as electrocardiography, cardiac ultrasound, or cardiac MRI [23].

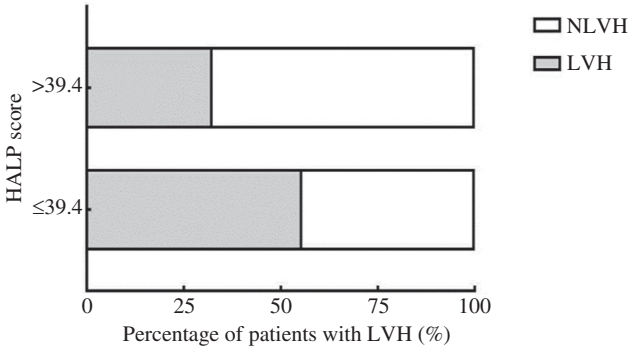

Figure 2 Percentage of Left Ventricular Hypertrophy (LVH) Occurrence According to HALP Score Tertile.

However, owing to the limited accessibility and high cost of these methods, a simple, cost-effective, and readily available indicator for early detection of cardiac hypertrophy is urgently needed.

Although the pathophysiological mechanism underlying the progression from hypertension to LVH remains unclear, a chronic systemic inflammatory response and nutritional status are believed to contribute. Chronic low-level inflammation has been recognized as a significant factor in the occurrence and progression of cardiovascular diseases, including hypertension [24]. Numerous studies

Table 3 Univariate and Multivariate Logistic Regression Analysis Indicating Independent Predictors of LVH.

Variables	Univariate analysis		Multivariate analysis	
	P value	OR (95% CI)	P value	OR (95% CI)
Age	0.507	0.985 (0.943–1.029)	–	–
Female	<0.001	17.437 (8.802–34.542)	<0.001	9.962 (3.866–24.300)
BMI	0.746	1.012 (0.944–1.084)	–	–
HALP Score	<0.001	0.942 (0.920–0.964)	0.002	0.944 (0.910–0.979)
Sodium	<0.001	1.294 (1.133–1.478)	0.091	1.159 (0.977–1.374)
Creatinine	<0.001	0.940 (0.918–0.962)	0.116	0.976 (0.948–1.006)
SBP	0.031	1.015 (1.001–1.029)	0.046	1.021 (1.001–1.041)
HDL-C	0.023	3.210 (1.173–8.785)	0.795	0.824 (0.190–3.572)
WBC count	<0.001	0.643 (0.508–0.815)	0.995	1.003 (0.434–2.318)
Neutrophil count	0.002	0.638 (0.482–0.843)	0.437	0.676 (0.252–1.816)
E/e′	0.004	1.108 (1.034–1.187)	0.600	1.029 (0.924–1.146)
e′	0.011	0.811 (0.691–0.952)	0.103	0.797 (0.607–1.047)

LVH: left ventricular hypertrophy; SBP: systolic blood pressure; BMI: body mass index; HDL-C: high-density lipoprotein cholesterol; WBC: white blood cell.

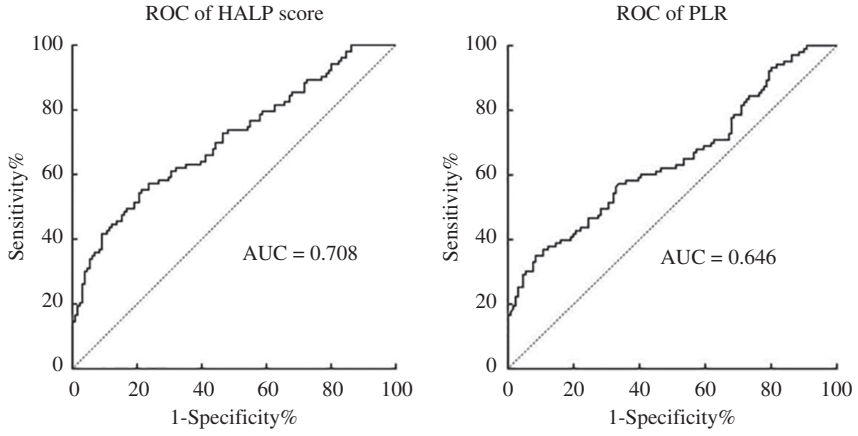

Figure 3 Receiver-Operating Characteristic (ROC) Curve Analyses of the HALP Score and PLR for the Identification of LVH. The area under the curve is larger for the HALP Score than the PLR.

have consistently demonstrated that hypertension is associated with a chronic, persistent inflammatory state. Inflammatory markers such as C-reactive protein (CRP), interleukins, and tumor necrosis factor-α are elevated in individuals with hypertension, and play major roles in fibrosis, a major process in ventricular remodeling. Consequently, patients with hypertension may experience heart damage as a result of these processes [4]. Previous research has indicated that chronic persistent inflammation is associated with elevated lymphocyte apoptosis. A decrease in lymphocyte count has been associated with high susceptibility to complications in patients with hypertension [5]. Furthermore, the infiltration of lymphocyte immune cells in the heart and kidneys can lead to dysfunction in these organs [6]. Platelets are also indispensable in the initiation and progression of inflammation. Sustained inflammation stimulates the proliferation of megakaryocytes, thereby increasing platelet production. Su et al. [25] have revealed that activated platelets release platelet-derived growth factor, platelet factor 4, and P-selectin, which contribute to the proliferation of cardiomyocytes. Another fundamental study [26] has demonstrated that platelet-released serotonin directly regulates cardiac fibroblasts by enhancing the secretion of transforming growth factor-beta (TGF-β) and matrix metalloproteinases, thereby leading to the migration and differentiation of cardiac fibroblasts, and ultimately myocardial remodeling.

Nutritional status is intricately associated with sympathetic activity and the inflammatory response [27]. In a state of poor nutritional status, heightened sympathetic nerve excitability increases norepinephrine levels, thus stimulating α1 adrenergic receptors, and resulting in enhanced protein synthesis in cardiomyocytes and subsequent cardiac hypertrophy [28]. Simultaneously, heightened sympathetic nervous system activity facilitates the infiltration of inflammatory factors into the heart, thereby leading to myocardial fibrosis and further exacerbating myocardial hypertrophy [29]. Furthermore, Kaysen et al. [30] have demonstrated an interdependent relationship between inflammation and malnutrition, which can create a vicious cycle. In the presence of malnutrition, the activation of the inflammatory response promotes the infiltration of numerous inflammatory factors into cardiomyocytes and surrounding tissues. This infiltration subsequently diminishes the bioavailability of nitric oxide and cyclic guanosine acid, alters troponin phosphorylation, and disrupts the balance of Ca^{2+}. These mechanisms ultimately contribute to the development of cardiomyocyte hypertrophy [31].

The novel HALP Score, designed to assess patients' nutritional and inflammatory status, incorporates inflammatory and nutritional markers such as hemoglobin, albumin, lymphocyte, and platelet counts. Previous studies have explored the prognostic value of this index primarily in patients with cancer, whereas limited research has focused on cardiovascular disease. Our findings indicated that a low HALP Score at admission may indicate an underlying state of malnutrition and systemic inflammation in older patients with hypertension. Moreover, a significant association was observed between a lower HALP Score and the presence of LVH.

In contrast to previous studies, our research did not observe any significant differences in albumin, lymphocyte counts, NLR, and SII between groups. We believe that this difference might be attributable to the inclusion of a different population in our study. Specifically, our study focused on older patients with hypertension, who might exhibit a slightly more delayed response to inflammation and nutritional status than younger individuals. Consequently, the corresponding indicators might not have been fully reflected in the early stages (left ventricular hypertrophy due to hypertension), thus making the HALP Score more suitable for the older than the younger population. Consequently, although a significant difference in HALP Score was observed between groups in our study, this hypothesis necessitates further verification through large-scale and multi-center research.

Beyond the HALP Score, our study identified female sex as a risk factor for LVH in older patients with hypertension. The findings align with those from a prospective study in 1419 patients with hypertension without initial LVH, which has highlighted that women have a greater risk of developing LVH than men [32]. Hoshida et al. [33] have shown a more pronounced increase in LVH and myocardial stiffness in older female patients with hypertension than male patients. One possible explanation for this finding is that estrogen regulates the activity of the renin-angiotensin-aldosterone system (RAAS),

the sympathetic nervous system, and oxidative stress, all of which are key factors in the development of structural abnormalities in the heart [34]. Additionally, postmenopausal women tend to have higher levels of inflammatory factors, such as TNF α, interleukin, and plasma protein activator inhibitor-1, as well as the fibrosis marker galectin-3, than men [35]. Therefore, in clinical practice, close attention must be paid to the incidence of LVH in older female patients with hypertension.

Although our study indicated that the HALP Score is more valuable than the PLR in diagnosing LVH in older patients with hypertension, its sensitivity and particularly its specificity were not high. Consequently, on the basis of the results of this study, the HALP Score may not be effective in accurately diagnosing the occurrence of LVH. To further validate its predictive value, large-scale, multi-center prospective studies are required in the future.

This study has several limitations. First, this was a small sample, single-center retrospective study, thereby limiting our ability to establish a causal relationship between a low HALP Score and LVH in older patients with hypertension. Additionally, the relatively small sample size might have introduced bias into the results. Second, previous studies have demonstrated a significant association between LVH and inflammatory markers such as CRP, interleukin-6, and tumor necrosis factor-α. However, we did not collect complete data on these markers, thus potentially affecting the overall analysis and interpretation of the results. Third, the smoking and drinking history of the patients was not comprehensively collected in our study. Consequently, the potential influence of smoking and drinking habits on the outcomes could not be determined. To address these limitations, future research should consider using larger sample sizes, multi-center designs, and collection of comprehensive data on inflammatory markers and lifestyle factors, to provide a more robust understanding of the relationship between the HALP Score and LVH, and potential contributing factors.

Conclusion

Together, our results suggest that the HALP Score is independently associated with the presence of LVH in older patients with hypertension – an association not previously reported. Therefore, the HALP Score may serve as a powerful and cost-effective marker, derived from routine blood analysis and liver function tests, for preliminary assessment of the presence of LVH. The HALP Score may enable healthcare professionals to identify patients at high risk of LVH and implement timely interventions, thus offering a new approach to LVH prevention or reduction in older patients with hypertension.

Data Availability Statement

Clinical data are available through the corresponding author.

Ethics Statement

All experimental procedures were approved by the Ethics Committee of Hebei General Hospital (clinical ethics approval number 2019-27).

Author Contributions

Yingfang Liu , Yan Wang and Yifang Guo conceived the study. Yingfang Liu and Ye Meng acquired the data. Yingfang Liu, Ye Meng and Qiuli Wang analyzed the data. Yingfang Liu reviewed the literature and prepared the first draft of this manuscript. Yan Wang and Yifang Guo critically reviewed and edited the manuscript, and approved the final version. All authors have read and approved the final manuscript.

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Conflict of Interest

All authors declare no relationships of competing interests.

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