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Original Article

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Association of the Left Ventricular Hypertrophy and Red Cell Distribution Width in Newly Diagnosed Hypertensive Patients

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ABSTRACT

Objective: Red cell distribution width is a component of the complete blood count analysis and has a rather important place in the diagnosis of anemia. High red cell distribution width has also been defined as an independent risk factor in patients diagnosed with a stroke, hypertension, and coronary artery disease. Another identified independent risk factor for cardiovascular events is left ventricular hypertrophy. In this study, we aimed to investigate the association between red cell distribution width and left ventricular hypertrophy in newly diagnosed hypertensive patients.
Methods: Our study included 393 patients who were newly diagnosed with hypertension by ambulatory blood pressure monitoring and had echocardiography. Patients were divided into 2 groups as patients with left ventricular hypertrophy (Group 1) and patients without left ventricular hypertrophy (Group 2). Red cell distribution width levels were compared between groups. A *P* value less than .05 was considered significant.
Results: The median age of the study group was 54 years (interquartile range = 42-64 years). Mean red cell distribution width was 13.01 ± 0.9 in Group 1 and 12.38 ± 0.6 in Group 2. Red cell distribution width was found to be significantly higher in Group 1 than in Group 2 ($P < .001$). Logistic regression analysis revealed that red cell distribution width is an independent risk factor for left ventricular hypertrophy.
Conclusion: The present study showed that a higher red cell distribution width value is an independent risk factor for left ventricular hypertrophy. Patients having higher red cell distribution width values should be examined for left ventricular hypertrophy regularly.
Keywords: Ambulatory blood pressure, hypertension, left ventricular hypertrophy, non-dipping, red cell distribution width

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INTRODUCTION

Red cell distribution width (RDW) is a component of complete blood count analysis. It is widely used in the diagnosis of many hematological diseases, especially anemia.[1-3] Except for routine use, it has been stated that high RDW may be an indicator of morbidity and mortality in terms of cardiovascular events.[1] High RDW has also been defined as an independent risk factor in patients diagnosed with a stroke, hypertension (HT), coronary artery disease (CAD), and myocardial infarction.[2-7]

Hypertension is among the modifiable risk factors for cardiovascular diseases.[8] Many authors have reported that resting blood pressure (BP) measurements do not always provide accurate information.[9] Therefore, ambulatory blood pressure monitoring (ABPM) is still the gold standard for diagnosis.[9] According to ABPM results, patients are divided into 2 groups as dipping HT and non-dipping HT pattern.[10] As it is known, blood pressure has a circadian rhythm and decreased more than 10% at night.[7] The non-dipping HT pattern describes a situation where nighttime BP does not drop to the required

extent.[7] Considering the studies investigating the relationship between circadian BP and RDW, it was stated that especially in patients with non-dipping HT pattern, high RDW poses a risk in terms of stroke, cardiovascular diseases, and subclinical target organ damage.[7]

Another identified independent risk factor for cardiovascular events is left ventricular hypertrophy (LVH).[11] Left ventricular hypertrophy is unfortunately a very common complication in hypertensive individuals and is called target organ damage[12] and the non-dipping HT pattern has an important role in its development.[13] In addition, the presence of high BP increases left ventricular wall thickness and causes cardiac functional changes.[14] Complications of LVH include congestive heart failure, arrhythmias, and sudden death.[15] Measurements made with ABPM, the gold standard in the diagnosis of HT, have an extremely important prognostic value for LVH,[11] and this value is more prominent when compared to office BP measurements.[16] Studies examining the relationship between left ventricular mass index (LVMI) and ABPM have demonstrated the importance of ABPM and BP monitoring in predicting the increase in left ventricular mass.[17] Finally, there are studies suggesting that untreated newly diagnosed hypertensive individuals with LVH may have an increased RDW compared to individuals without LVH.[18]

In this study, we aimed to investigate the association between RDW and LVH in newly diagnosed hypertensive patients.

METHODS

Patients

Electronic files of all patients who underwent ABPM between April 2019 and April 2021 were retrospectively analyzed. Patients newly diagnosed with HT were included in the study. Laboratory tests, echocardiographic measurements, demographic data, and comorbidities were recorded. All laboratory tests were analyzed with an automatic integrated analyzer (Cobas 6000, Roche, Switzerland). Patients with anemia, receiving antihypertensive medication, hematological or oncological malignancy, and no echocardiographic examination were excluded from the study. Patients were divided into 2 groups as patients with LVH and patients without LVH. A total of 456 patients were screened for the study; 63 were excluded because of comorbidities, having no hypertension, and having no echocardiography (Figure 1).

Stable CAD was defined as a history of myocardial infarction and coronary plaque documented by catheterization.[19] Hyperlipidemia (HL) was diagnosed with National Cholesterol Education Program-Adult Treatment Panel III (NCEP-ATP III criteria).[20]

The study was approved by the Afyonkarahisar Health Sciences University ethics committee (Date: December 3, 2021 meeting number: 2021-13, decision no: 535). Informed consent was not taken from patients because of the retrospective design.

Blood Pressure Data

A 24-hour ambulatory BP measurement was performed with a verified device (Cardioline monitor [Walk200b model] and Cardioline blood pressure cuff). Ambulatory BP was recorded every 30-min period in the daytime (from 08:00 AM to 11:59 PM) and every 60-min period at nighttime (from noon to 7:59 AM). If the first measurement failed, the measurement was repeated once again and measurements were accepted only when at least 75% of the measurements were successful. Values outside of normal physiological ranges and spurious readings due to motion artifacts were automatically edited by the analysis software. Dipping status was evaluated based on the amount of decrease in mean systolic BP at nighttime. Patients with a decrease of ≥10% in mean systolic BP at night compared to daytime were defined as dippers, whereas those who have a decrease of less than 10% in mean systolic BP at nighttime were defined as non-dippers.[21]

Echocardiography

A Vivid 7 ultrasound machine (Vingmed-General Electric, Norway) with a 3.5 MHz transducer was used for echocardiographic evaluation. Standard echocardiographic windows were performed using parasternal long-axis and apical

MAIN POINTS

- It was stated that high red cell distribution width (RDW) especially in patients with non-dipping hypertension (HT) pattern poses a risk for stroke, cardiovascular diseases, and subclinical target organ damage.
- Untreated newly diagnosed hypertensive patients with left ventricular hypertrophy (LVH) may have higher RDW levels than patients without LVH.
- A higher RDW value is an independent risk factor for LVH.
- Patients having higher RDW values should be examined for LVH regularly.

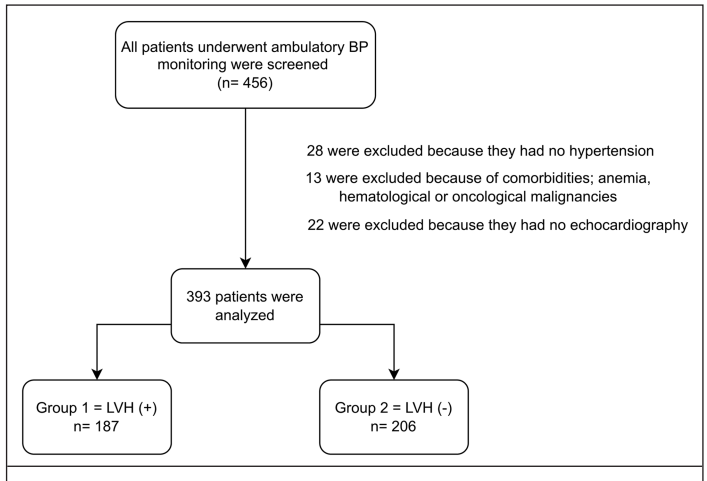

Figure 1. Study design.

views in the left lateral decubitus position. Standard M-mode images in end diastole were used for measurements of left ventricular end-systolic diameter, left ventricular end-diastolic diameter, posterior wall thickness, and interventricular septal thickness.[22] When determining LV mass, 2D echocardiographic measurements were calculated using the M-mode formula and the result was normalized to body surface area.[23] Left ventricular hypertrophy was defined as LVMI > 125 g m^{-2} for males and LVMI > 110 g m^{-2} for females. Examinations were performed by a single cardiologist who is an expert in echocardiography.

Statistical Analysis

Categorical variables were presented as frequencies and percentages. Chi-square test was employed for the comparison of categorical variables. The compliance of continuous variables to normal distribution was checked by visual histograms and Shapiro–Wilk test. Normally distributed continuous variables were presented as mean ± standard deviation, non-normally distributed continuous variables were presented as median and interquartile ranges. Normally distributed continuous variables were compared with the independent-samples *t*-test, and continuous variables not normally distributed were compared with the Mann–Whitney *U*-test. Univariate and multivariate logistic regression analyses were done for determining the risk factors of LVH presence. Parameters having a *P* value less than .2 were taken for univariate regression analysis. Parameters found as risk factors in the univariate analysis were included in the multivariate analysis. All *P* values presented in the study are bidirectional, and $P < .05$ was considered as statistically significant. International Business Machines' Statistical Package for Social Sciences 26.0 program was used for data analysis.

RESULTS

The median age of the study group was 54 years (interquartile range (IQR) = 42-64 years); 56% of the patients (n = 220) were female and 44% were (n = 173) male. Ambulatory blood pressure monitoring revealed that 46.8% of patients (n = 184) were dippers and 53.2% of patients (n = 209) were non-dippers. Dipping status was observed at a rate of 35.3% (n = 66) in patients with LVH, while it was detected at a rate of 57.3% (n = 57.3) in patients without LVH. Table 1 shows the characteristic features of the groups.

Red cell distribution width was found to be significantly higher in Group 1 than in Group 2 ($P < .001$). Figure 2 shows the comparison of RDW between groups.

Univariate analysis showed that dipping status, nighttime diastolic BP, and RDW are risk factors for LVH ($P < .001$, $P = .001$, and $P < .001$, respectively). Multivariate analysis showed that nighttime diastolic BP and RDW are independent risk factors for LVH (hazard ratio (HR) = 1.025, 95% CI = 1.004-1.046, $P = .018$, and HR = 2.305, 95% CI = 1.710-3.107, $P < .001$, respectively). Table 2 shows univariate and multivariate regression analyses for LVH presence.

Table 1. Characteristic Features of Patients According to LVH Groups

Parameter	LVH (+) (n = 187)	LVH (–) (n = 206)	*P*
Mean age (years)	54.33 ± 16.9	52.77 ± 15.5	.192
Female, n (%)	100 (53.5)	120 (58.3)	.361
Dipper status, n (%)			
Dipper	66 (35.3)	118 (57.3)	**<.001**
Non-dipper	121 (64.7)	88 (42.3)	
Presence of DM, n (%)	50 (26.7)	39 (18.8)	.053
Presence of CAD, n (%)	27 (14.5)	26 (12.6)	.658
Presence of HL, n (%)	41 (21.9)	10 (4.9)	**<.001**
Weight (kg)	77.41 ± 11.4	75.96 ± 11.7	.162
Height (mt)	1.67 ± 0.08	1.66 ± 0.08	.425
Daytime SBP (mmHg)	141.24 ± 8.8	142.38 ± 9.4	.287
Daytime DBP (mmHg)	76.63 ± 10.9	77.8 ± 11.1	.457
Nighttime SBP (mmHg)	126.9 ± 19.5	124.7 ± 13.7	.383
Nighttime DBP (mmHg)	72.81 ± 11.9	69.13 ± 10.2	**.001**
LVM index (g/m^2)	121.10 ± 8.9	107.9 ± 11.7	**<.001**
RDW (%)	13.01 ± 0.9	12.38 ± 0.6	**<.001**

CAD, coronary artery disease; DBP, diastolic blood pressure; DM, diabetes mellitus; HL, hyperlipidemia; LVM, left ventricular mass; RDW, red cell distribution width; SBP, systolic blood pressure.
P-values less than accepted as statistically significant.

DISCUSSION

Our study included 393 patients with ABPM and echocardiographic examinations. The parameters that examined the presence of LVH were evaluated. Considering dipper status, the

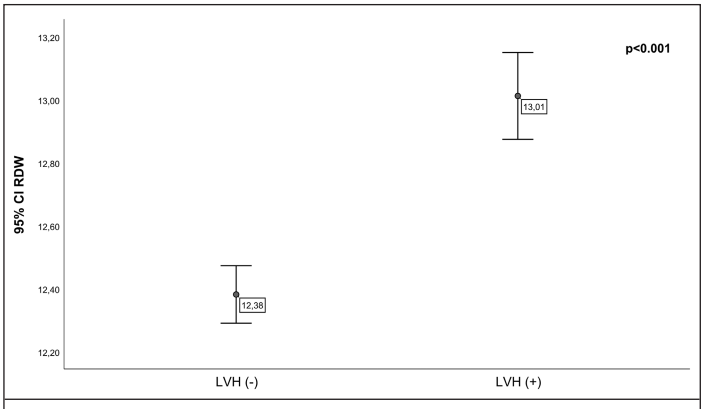

Figure 2. Comparison of red cell distribution width between left ventricular hypertrophy groups.

Table 2. Logistic Regression Analysis for LVH Presence

	Univariate		Multivariate	
Parameters	**OR (95% CI)**	***P***	**OR (95% CI)**	***P***
Age	1.006 (0.994-1.019)	.328		
Non-dipper status	2.458 (1.635-3.695)	**<.001**	1.314 (0.825-2.091)	.250
DM	1.401 (0.862-2.278)	.174		
Hyperlipidemia	1.381 (0.721-2.648)	.331		
Weight	1.002 (0.985-1.019)	.825		
Nighttime DBP	1.032 (1.013-1.052)	**.001**	1.025 (1.004-1.046)	**.018**
RDW	2.545 (1.935-3.346)	**<.001**	2.305 (1.710-3.107)	**<.001**

DBP, diastolic blood pressure; DM, diabetes mellitus; LVH, left ventricular hypertrophy; OR, odds ratio; RDW, red cell distribution width.
P-values less than accepted as statistically significant.

most striking finding was the relationship between the LVH and RDW. The distribution of non-dipping cases in the LVH group was consistent with the literature.

As it is known, HT is a very common disease and is an extremely important risk factor for target organ damage such as myocardial infarction, stroke, and kidney failure.[24] Many clinical studies over the last 25 years have highlighted the importance of the relationship between ABPM and the prevalence of cardiovascular events.[9] Many researchers have shown the correlation between HT[6,25,26] and especially non-dipping HT[7,27,28] and RDW. In another study evaluating non-dipping HT cases, it was stated that high RDW is an independent risk factor in coronary artery plaque formation and myocardial infarction pathology.[29]

In our study, RDW was higher in the group with LVH than in the other group, and our results were consistent with the literature. This situation can be explained by studies pointing out the relationship between LVH and non-dipper status. The researchers found that patients with non-dipping HT pattern had an increased RDW compared to those with dipping HT pattern.[11,28,29] Some hypotheses state that increased oxidative stress, endothelial damage, and inflammation may cause an increase in RDW levels.[29,30] In addition, with the effect of inflammatory cytokines, there may be suppression in the bone marrow and entry into the blood circulation before the erythroid cells mature.[29] Although the underlying mechanisms for the non-dipping HT pattern have not been clearly revealed yet, the studies carried out revealed that it is more common in obese, elderly, individuals with autonomic dysfunction, low sleep quality, and high sympathetic activity.[7] However, we did not detect a significant feature in the demographic data. Two explanations may bring clarity to this situation. First, patients divided into groups have similar age characteristics; second, the limited number of both the geriatric population and the total number of patients.

Many studies are addressing the relationship between LVH and non-dipper status. Increased pulsatile load on the heart with high BP can lead to structural changes in cardiac tissue.[11] It has been emphasized that an increase in LVMI increases the risk of mortality in terms of cardiovascular diseases.[31] In addition, arterial stiffness as a result of pulsatile load can increase the workload of the heart and cause LVH.[11] Similarly, there are publications stating that BP, which has a high course at night, may increase arterial stiffness, leading to the deterioration of compliance in the great arteries, and therefore the increased load may be reflected on the left ventricular tissue and cause LVH.[11] Finally, in another study by Balci et al.[13] ventricular hypertrophy was found to be significantly higher in the group with non-dipping HT compared to the group with dipping HT (42.9% vs. 6.3%, $P < .03$). On the other hand, there are publications that do not overlap with the studies mentioned above. In the study of Cicconetti et al[32] in which 40 patients with dipping/non-dipping HT pattern were included and evaluated echocardiographically, no significant difference was found between the groups in terms of LVMI, left ventricular wall thickness, and left ventricular mass.

In our study, the relationship between LVH and increased RDW is similar to the literature. There are some hypotheses explaining the possible relationship between LVH and RDW. In a study on cardiomyocytes, it was stated that tumor necrosis factor (TNF), endothelin-1, and pulsatile mechanical stresses may cause cardiac hypertrophy.[33] This study also showed that intracellular reactive oxygen radicals, which have a role in the development of LVH, can be inhibited by antioxidants. The depletion of antioxidants or the release of many reactive oxygen products results in oxidative stress.[34] Moving away from the antioxidant effect in terms of metabolic balance may cause high RDW.[18] Another hypothesis that may cause RDW elevation is that cytokines such as interleukin 1-beta (IL-1β) and IL-6, which have a very important role in inflammation, affect iron metabolism and suppress the bone marrow.[29,35] In addition, when the RDW levels of hypertensive and normotensive individuals were compared, it was observed that the RDW levels of hypertensive individuals were found to be high.[6,25,27] From this point of view, it is highly likely to encounter increased RDW in both individuals with LVH and hypertensive individuals.

Another factor that may support the relationship between LVH and RDW in our study may be dipper status. Because it is known that non-dipping HT pattern can cause high RDW independent of LVH.[27] Studies conducted in recent years indicate that cases with non-dipping HT pattern have higher RDW levels compared to cases with dipping HT pattern.[28,29] On the

other hand, the comparison of these 2 groups allowed us to obtain striking data. In the comparison of the groups, we think that the presence of non-dipper status stands out in terms of increased RDW. Because the role of non-dipper status in the development of LVH has been expressed in many studies.[11,14] In this respect, the relationship between LVH and RDW in non-dipper individuals is quite remarkable in our study. In this context, the observed increases in RDW may be caused by many interrelated factors.

Finally, the relationship between LVH, myocardial dysfunction, CAD, and epicardial adipose tissue is known.[36] There are studies showing that HL is higher in patients with high epicardial fat thickness than in those with low.[36] In our study, the presence of HL in the group with LVH is remarkable in this respect. However, it was not evaluated as an independent risk factor for LVH. This may be due to the small number of patients in the subgroups.

There are some limitations in our study. As it is known, ABPM is not a routinely requested examination in hypertensive cases. Therefore, the first limitation we should mention is the insufficient number of patients newly diagnosed with HT and undergoing ABPM examination. Second, one-time RDW measurements may have increased the likelihood of analytical disadvantages. Third, parameters such as high-sensitivity C-reactive protein (hs-CRP), TNF alpha, and IL-6 could not be evaluated to assess inflammation. Finally, the fact that it was a retrospective study involving small patient groups may have caused bias as well as affected the cause-effect relationship.

Untreated newly diagnosed hypertensive individuals with LVH may have an increased RDW than patients without LVH. RDW is an easily accessible complete blood count parameter. The present study showed that a higher RDW value is an independent risk factor for LVH. Patients having higher RDW values should be examined for LVH regularly. Considering the existing limitations, multicenter studies with larger numbers of patients are needed to confirm our results.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Afyonkarahisar Health Sciences University (Date: December 3, 2021, Decision number: 535).

Informed Consent: Informed consent was not taken from patients because of the retrospective design.

Peer-review: Externally peer-reviewed.

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