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ORIGINAL RESEARCH ARTICLE

Impact of hypertension and hypertensive left ventricular hypertrophy on left ventricular mechanical dyssynchrony in patients with normal gated-SPECT myocardial perfusion imaging

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ABSTRACT

Introduction: Left ventricular hypertrophy (LVH) represents structural changes of the myocardium in hypertensive patients, which can lead to heart failure. The main aim of this study was to investigate the impact of hypertension and hypertensive LVH on left ventricular mechanical dyssynchrony (LVMD).

Methods: This study was conducted on patients without known history of coronary artery disease who were referred by cardiologists for myocardial perfusion imaging (MPI) in 2023 and 2024. The presence or absence of LVH was determined based on the patient's echocardiography. MPI was performed in stress and rest phases by gated-SPECT imaging, and LVMD was determined using QGS software phase analysis.

Results: In this study, 843 patients were evaluated, including 575 women (68.21%) and 268 men (31.79%). Four hundred and fifty patients (53.38%) had a history of hypertension, and 155 patients (18.45%) had LVH. The prevalence of LVMD in hypertensive patients with LVH (23.87%) was significantly higher (P-value=0.007) than in hypertensive patients without LVH (13.36%) and normotensive patients (13.99%). LVMD was significantly more common in men than women (P-value=0.000), but there was no significant association between age, body mass index, smoking, and hyperlipidemia with LVMD (P-value>0.05).

Conclusion: Hypertension without LVH had no significant impact on the prevalence of LVMD, while hypertensive LVH was associated with LVMD therefore hypertensive patients with LVH and LVMD may need more intensive treatment and follow-up.

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INTRODUCTION

Hypertension is a common disorder, affecting one in three adult people in the United States [1]. Hypertensive patients are mostly asymptomatic and up to 40% of patients newly diagnosed with hypertension having already developed cardiovascular complications or hypertensive kidneys, eyes or brain damage [2]. Hypertensive heart disease represents microscopic and macroscopic changes of myocardium leading to structural and functional abnormalities, including cardiac fibrosis and remodeling of the heart chambers and arterial system [3]. In addition to aggravating the process of epicardial coronary atherosclerosis, hypertension can cause microvascular angina and myocardial dysfunction through microvascular dysfunction [4].

Left ventricular hypertrophy (LVH) is a known hypertension-induced complication that is associated with microvascular dysfunction and can ultimately lead to heart failure with preserved ejection fraction (HFpEF) or reduced ejection fraction (HFrEF) [5]. Detection of microvascular dysfunction, even in patients without epicardial coronary artery disease, plays a key role in mitigating cardiovascular complications, especially heart failure [6]. HFpEF is usually seen in hypertensive patients in whom microvascular dysfunction and LVH have caused interstitial tissue fibrosis [7]. Impaired myocardial shortening is an early finding in HFpEF in patients with hypertensive heart disease and it plays an important role in the prognosis of these patients [8]. As the same way, left ventricular mechanical dyssynchrony (LVMD) may represent subclinical myocardial dysfunction in these patients. The LVMD as an early presentation of left ventricular dysfunction has been demonstrated in patients with diabetes [9], chronic kidney disease [10], and post-chemotherapy [11]. In addition, LVMD is linked to poor prognosis in patients with known coronary artery disease and in patients with heart failure [12, 13]. Schindler and Valenta reported that treatment of hypertension, as one of the major risk factors of cardiac microvascular dysfunction, along with proper control of diabetes and hyperlipidemia, exerts a positive effect on the prognosis of these patients [14].

Cardiac interstitial fibrosis secondary to hypertension, as an indicator of HFpEF in hypertensive patients, can be detected with high precision by cardiac MRI [15]. However, it is a costly option that is not widely available, and other causes of microvascular dysfunction, such as obstructive epicardial coronary artery disease, must be ruled out before adopting this method. Echocardiographic methods such as estimation of coronary blood flow reserve by contrast-enhanced echocardiography and left ventricular global circumferential or longitudinal strain in 3D echocardiography are also widely used in this context. Echocardiographic techniques have advantages such as no ionizing radiation, lower cost, and more widespread application. However, they have some limitations such as high artifact and inter-observer variability, especially in obese patients, patients with pulmonary disease, and patients with mild to moderate myocardial ischemia [16, 17]. Obstructive epicardial coronary artery disease must also be ruled out as a major cause of cardiac fibrosis on echocardiography.

Gated-SPECT Myocardial Perfusion Imaging (MPI) is a common procedure to screen patients suspected of coronary artery disease, especially those with cardiovascular disease risk factors such as diabetes or hypertension. Using phase analysis of quantitative gated-SPECT (QGS) software, LVMD can be detected without any change in imaging procedures, injected radiopharmaceutical dose, or scanning cost. At the same time, the presence or absence of coronary artery disease as a cause of LVMD can be confirmed or ruled out. In addition, phase analysis is completely automated and operator-independent, and its variables also bear independent value in estimating the risk of major cardiac events [18]. Hosseinzadeh et al. showed that LVMD diagnosis by phase analysis of gated-SPECT MPI in diabetic patients may prevent progress to heart failure [9]. However, patient's exposure to radiation is one of the drawbacks of this method compared to MRI and echocardiography.

Echocardiography is commonly performed in hypertensive patients, and finding of LVH is consistent with myocardial structural changes secondary to hypertension. However, LVH does not necessarily indicate myocardial dysfunction or interstitial tissue fibrosis. In contrast, LVMD represent myocardial dysfunction, which can be caused by microvascular dysfunction and cardiac interstitial tissue fibrosis. Therefore, the diagnosis of LVMD may be an early finding in HFpEF, and it may have prognostic value [19]. This finding may also affect type or intensity of treatment in hypertensive patients [20].

Relationship between LVMD and hypertension is not established. Actually, in the study by Yaghoubi et al. comparison of LVMD indices derived from gated SPECT phase analysis revealed no significant difference between 99 hypertensive patients and a control group of 100 patients without a history of hypertension [21]. Therefore, further research seems to be warranted in this field. The main goal of this study was to investigate the effect of hypertension

and hypertensive LVH on the prevalence of LVMD using the phase analysis in the gated-SPECT MPI.

METHODS

This was a prospective study. The study population consisted of people clinically suspected of coronary artery disease who were referred by cardiologists to the nuclear medicine department of Kowsar Hospital for gated-SPECT MPI in 2023 and 2024. Among these people, patients with a history of diabetes, renal failure, and history of myocardial or pulmonary diseases (including known coronary artery disease, heart failure, heart valves disorders, any type of arrhythmia and heart block, asthma, and chronic obstructive pulmonary disease) were excluded from the study. The echocardiography had been performed by the cardiologists for the patients in the past month was reviewed, and patients whose left ventricular ejection fraction were below 50% were excluded. Diagnosis of LVH in the patient's echocardiography (end-diastolic interventricular septal wall thickness >11 mm for men and >10 mm for women on parasternal long-axis view) was recorded in the patient's questionnaire. This study was approved by the Research Ethics Committee of Kurdistan University of Medical Sciences (Ethics Code IR.MUK.REC.1403.092). Adequate explanations about MPI were offered to all patients and written informed consent was obtained.

Gated-SPECT MPI was performed 60 to 90 minutes after injection of 20 to 25 mCi of the technetium-99m sestamibi ([^{99m}Tc]Tc-MIBI) in the stress (drug or exercise-induced stress) and rest (non-gated SPECT) phases in all patients. Imaging was conducted by a Siemens SPECT dual-head variable angle scanner (Evo-Excel model, made in 2018, Germany) with a low-energy/high-resolution collimator, 32 projections of 20-second in a 180-degree semicircular orbit in the supine position for women and the prone position for men. Images were taken with a 64*64*8 matrix and a 345 mm detector field. Gated-SPECT MPI images were reconstructed by an experienced nuclear medicine specialist using OSEM reconstruction, and normal MPI was considered as summed stress score of 3 or less. Gated ECG data were collected using 8 frames per cardiac cycle with a ±10% acceptance window according to the routine protocol in our nuclear medicine department. Quantitative parameters of left ventricular function, including end-diastolic volume, end-systolic volume, left ventricular ejection fraction, and summed motion and thickening scores of the left ventricle, were calculated using QGS software. Patients with an ejection fraction of less than 50% or a summed score of 3 or more for left ventricular motion or thickening were considered abnormal and excluded from the study. Phase analysis was conducted using QGS software, and left ventricular contractility indices, including phase histogram width (PHB, 95% of the phase distribution), phase standard deviation (PSD), and entropy (percentage of irregularity/asynchrony of ventricular contraction) were obtained. Given the normal ranges defined for the above indices in QGS software (18), LVMD was diagnosed as follows: PHB>39 and/or PSD>11 and/or Entropy>41.

The study data were analyzed by STATA software version. 14. First, a frequency distribution table was drawn for qualitative variables, and the mean and standard deviation were estimated for quantitative variables. To test the study hypotheses, one-way analysis of variance and Pearson correlation coefficient were used for normal data, and non-parametric Kruskal-Wallis test and Spearman correlation coefficient were utilized for abnormal data. The assumption of normality was tested using the Shapiro-Wilk test. A significance level of 5% was considered. Multiple logistic regression was used for evaluating relationship of studied variables with left ventricular mechanical dyssynchrony and managing the confounders.

RESULTS

A total of 843 patients were included in this study, of which 575 (68.21%) were women and 268 (31.79%) were men. The demographic characteristics of the patients are outlined in Table 1. Seventy-eight patients (9.22%) were smokers and 765 patients (90.78%) were non-smokers. One hundred and thirty-nine patients (16.49%) had a history of hyperlipidemia, and 704 patients (83.51%) had no history of hyperlipidemia.

Table 1. Demographic data of the studied patients

	Mean ± SD	Minimum	Maximum
Age (year)	54.27 ± 11.41	21	89
Body mass index (Kg/m^2)	28.84 ± 4.79	16.02	45.92

Four hundred and fifty patients (53.38%) had a history of hypertension, and 155 patients (18.45%) had LVH based on echocardiography. Three patients had incomplete echocardiographic data. The patients were divided into three groups: patients without a history of hypertension and no LVH (HT-LVH-, 393 patients, 46.79%), patients with a history of hypertension and no LVH (HT+LVH-, 292 patients, 34.76%), and patients with a history of hypertension

and LVH on echocardiography (HT+LVH+, 155 patients, 18.45%). The mean end-systolic volume, end-diastolic volume, and left ventricular ejection fraction did not show significant differences in these three groups (Table 2).

Prevalence of all LV mechanical dyssynchrony parameters were higher in hypertensive patients with LVH than other two groups, and the difference was statistically significant for PHB and entropy indices (Table 3).

Considering at least one abnormal phase analysis index as LVMD, prevalence of LVMD in hypertensive patients with LVH was significantly higher than in the other two groups (Table 4).

LVMD was not significantly related to age, body mass index (BMI), smoking, and hyperlipidemia, but it was significantly more common in men than women. The presence of LVH on echocardiography was also significantly associated with LVMD (Table 5).

Table 2. Left ventricular volumes and ejection fraction in the studied groups

	Groups	Mean ± SD	P-value
End diastolic volume (ml)	HT-LVH-[a]	61.73 ± 16.42	0.174
	HT+LVH-[b]	63.41 ± 16.65	
	HT+LVH+[c]	64.16 ± 17.75	
End systolic volume (ml)	HT-LVH-[a]	21.65 ± 9.16	0.362
	HT+LVH-[b]	21.55 ± 9.50	
	HT+LVH+[c]	22.90 ± 9.83	
Ejection fraction (%)	HT-LVH-[a]	66.19 ± 8.13	0.135
	HT+LVH-[b]	66.98 ± 8.42	
	HT+LVH+[c]	65.59 ± 8.36	

[a] No hypertension, no left ventricular hypertrophy
[b] Hypertension without left ventricular hypertrophy
[c] Hypertension with left ventricular hypertrophy

Table 3. Prevalence of LV mechanical dyssynchrony parameters in the studied groups

Groups	PHB>39	P-value	PSD>11	P-value	Entropy>41	P-value
HT-LVH-[a] (N=393)	37 (9.4%)	0.023	37 (9.4%)	0.128	29 (7.3%)	0.014
HT+LVH-[b] (N=292)	30 (10.2%)		27 (9.2%)		27 (9.2%)	
HT+LVH+[c] (N=155)	27 (17.4%)		23 (14.8%)		24 (15.4%)	
Total (N=840)	94 (11.1%)		87 (10.3%)		80 (9.5%)	

[a] No hypertension, no left ventricular hypertrophy
[b] Hypertension without left ventricular hypertrophy
[c] Hypertension with left ventricular hypertrophy

Table 4. Prevalence of left ventricular mechanical dyssynchrony in the studied groups

	Patients without dyssynchrony (%)	Patients with dyssynchrony (%)
HT-LVH-[a]	338 (86.01)	55 (13.99)
HT+LVH-[b]	253 (86.64)	39 (13.36)
HT+LVH+[c]	118 (76.13)	37 (23.87)
Total	709 (84.40)	131 (15.60)

[a] No hypertension, no left ventricular hypertrophy
[b] Hypertension without left ventricular hypertrophy
[c] Hypertension with left ventricular hypertrophy

Table 5. Relationship of studied variables with left ventricular mechanical dyssynchrony using logistic regression

		Odds ratio	Standard error	Z	P-value	95% Conf. interval
Age		0.987	0.011	-1.23	0.218	(0.966, 1.003)
Sex	Female	1.000	-	-	-	(3.303, 9.415)
	Male	5.577	1.490	6.43	<0.001	
Body mass index		1.030	0.028	1.13	0.261	(0.978, 1.086)
Smoking	No	1.000	-	-	-	(0.536, 2.017)
	Yes	1.039	0.352	0.11	0.909	
Hyperlipidemia	No	1.000	-	-	-	(0.331, 1.330)
	Yes	0.660	0.241	-1.17	0.243	
HT-LVH-[a]		1.000	-	-	-	-
HT+LVH-[b]		0.906	0.258	-0.35	0.729	(0.518, 1.585)
HT+LVH+[c]		2.296	0.687	2.78	<0.001	(1.277, 4.129)
Constant		0.059	0.060	-2.77	0.006	(0.008, 0.437)

[a] No hypertension, no left ventricular hypertrophy
[b] Hypertension without left ventricular hypertrophy
[c] Hypertension with left ventricular hypertrophy

DISCUSSION

In our study, the prevalence of LVMD in hypertensive patients without LVH was not significantly different from normotensive patients. In the same way, Yaghoubi et al. [21], did not find a relationship between hypertension and phase analysis variables. In the other hand, the LVMD was significantly more prevalent (23.87%) in hypertensive patients with LVH. In Parmar's study, the prevalence of LVMD using phase analysis (PHB>39.9 and/or PSD>11.9) in patients with LVH and normal myocardial perfusion scan was reported to be 24% [22], which was in harmony with our study. In contrast, Alvandi et al. reported the prevalence of LVMD in patients with hypertension to be 63.2% [23]. The higher prevalence in their study compared to our study is due to the fact that abnormal cutoff they considered for phase analysis variables (PHB>24 and/or PSD>8.6) were considerably lower than our study (and Parmar's study) which was based on the standard QGS software.

Our findings revealed that the prevalence of LVMD in hypertensive patients with LVH was nearly twice that of hypertensive patients without LVH. These findings are similar to the Tan's study which indicated hypertensive patients with LVH had a greater prevalence of both systolic and diastolic asynchrony than non-LVH patients [24].This suggests that LVMD is significantly aggravated following structural changes in the myocardium secondary to hypertension. However, presence of LVH on echocardiography does not imply this subclinical myocardial dysfunction [25], and in our study, more than three-quarters of hypertensive patients with LVH did not have LVMD.

Hypertension is a major risk factor for HFpEF. HFpEF encompasses a spectrum of abnormalities, the early manifestations of which are diastolic dysfunction and reduced left ventricular Global Longitudinal Strain (GLS) [26]. In the early and reversible stages of hypertensive heart disease, despite rising myocardial function (MW), GLS is normal, but the development of interstitial fibrosis causes a decline in myocardial function indices (MWI) [27, 28]. Passive myocardial stiffness due to interstitial fibrosis is considered as the main mechanism of HFpEF in hypertensive patients; however, in the largest study of HFpEF using endomyocardial biopsy to date, only 27% of patients had moderate or more fibrosis [29]. Structural abnormalities in sacrolemic proteins that lead to stiffening of myocytes represent another proposed mechanism of left ventricular stiffness [30]. LVH is a common feature of HFpEF, characterized by an imbalance between the number of myocytes and the severity of collagen deposit [31]. This may explain the higher prevalence of LVMD in hypertensive patients with LVH than hypertensive patients without LVH.

Although diastolic dysfunction is considered a key criterion for HFpEF, Omote et al. revealed that in about one-third of these patients, the left ventricular filling pressure was normal at rest and only increased during exercise [32]. In this regard, the diagnosis of LVMD on MPI, which is typically performed after exercise or pharmacologic stress may be preferable at least in some of these

patients. Furthermore, in patients in whom diastolic parameters are inconclusive, mild systolic abnormality such as a reduced GLS, may raise the suspicion of HFpEF [33]. LVMD, as a mild systolic abnormality, may play a similar role. A recent study by Biering-Sørensen et al. on 327 hypertensive patients showed that the systolic function of the cardiac apex was maintained in patients with hypertensive heart disease but was impaired in patients with HFpEF. They concluded that this regional mechanical impairment may be a marker of the transition from asymptomatic to symptomatic heart disease [34].

We also found that LVMD was present, with approximately the same prevalence, in normotensive patients and in hypertensive patients without LVH. Karakas et al. studied LVMD using color Doppler echocardiography in 72 hypertensive patients and 70 normotensive controls. In their study, LVMD was found in both normotensive and hypertensive groups in patients without a normal 10% drop in blood pressure at night (non-dipping blood pressure pattern). They concluded that this could be attributed to the effects of hypertension (including the lack of the natural nocturnal blood pressure lowering in patients without a history of hypertension) on the myocardium [35]. Karev et al. reported that LVMD in patients without a history of coronary artery disease improved or completely resolved after optimal treatment with antihypertensive drugs [36].

We did not find significant association between LVMD and BMI. Obesity is linked to chronic metabolic disorders and can prompt coronary microvascular dysfunction through various mechanisms such as sympathetic hyperactivity and mild systemic inflammation [37]. In Zhang's study, LVMD was more common in obese patients (BMI >30) in the gated-SPECT MPI [38]. This relationship was not seen in our study probably because the mean BMI of patients in our study was less than 30. In addition, waist-to-height ratio and central obesity measures seems to be more frequently used than BMI in assessing the risk of cardiovascular diseases and heart failure [39]. In the Caliskan study, a weak positive association was found between BMI and phase histogram width in obese patients [40].

Our findings did not show significant association between LVMD and age, but in Chan's study, LVMD during physical activity aggravated with age [41]. The discrepancy between Chan's study and ours is likely due to the fact that the vast majority of MPI procedures in our study were performed with pharmacologic stress without significant physical activity.

In our study, LVMD was significantly more common in men. Similarly, Tsai et al. showed that, under identical conditions of age, blood pressure, hypertension duration, and left ventricular mass, diastolic asynchrony was higher and myocardial strain was worse in men than in women [42].

Hypertensive heart disease predisposes patients to arrhythmias [43], and the diagnosis of LVMD may help predict ventricular arrhythmias in the future [44]. Fan et al. reported that LVMD can affect local coronary flow by causing regional changes in myocardial pressure, resulting in cardiac ischemia [45]. Sakatani et al. showed that LVMD following physical activity is an independent predictor of major cardiac events in patients with normal myocardial perfusion scan [46]. Elevated PHB and PSD as variables of MPI phase analysis are potential risk factors for sudden cardiac death in patients with hypertrophic cardiomyopathy [47]. As the same way, LVMD in patients with hypertensive LVH represent early myocardial dysfunction and may be associated with unfavorable prognosis.

CONCLUSION

Hypertension alone does not alter the prevalence of LVMD, but LVMD is associated with LVH in hypertensive patients. LVMD as a subtle myocardial dysfunction may be an early finding in HFpEF. In this way, hypertensive patients with LVH and LVMD may need more intensive treatment and follow-up.

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