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LEFT VENTRICULAR HYPERTROPHY REGRESSION AFTER OPTIMAL BLOOD PRESSURE CONTROL IN HYPERTENSIVE PATIENTS

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ABSTRACT

Background: Hypertension (HT) is a critical risk factor for the development of cardiovascular disease and a major public health concern. There are still a number of unclear issues about antihypertensive medication and LVH regression; one of the most important clinical questions is whether LVH is reversible or not. **Objectives:** To assess the factors associated with left ventricular hypertrophy among patients with optimum controlled hypertension. **Methods:** This is a cross-sectional study. Included adult patients aged more than 18 years old who had hypertension based on a history of prior diagnosis and therapy, blood pressure ≥ 140/90 mmHg on two to three office visits two weeks apart, home blood pressure readings >130/80 mmHg, or a single blood pressure reading of ≥180/110 mmHg. The study patients were assessed at the medical consultation clinic of Al-Shirqat General Hospital from August 2022 to October 2025. The questionnaire consisted from four sections. Section one for sociodemographic and anthropometric measurements, section two for baseline and follow up systolic, diastolic and heart rate measurements, section three for different clinical and biochemical variables and section four for Echocardiographic findings of the patients. **Results:** The study included 400 patients; 50 (12.5%) patients had left ventricular regression and 350 (87.5%) patients with no left ventricular regression. The mean age ± standard deviation of the study participants was 59.25 ± 10.72 years. Statistically significant difference between the two groups regarding their gender, smoking state, presence of obesity, mean of ages and mean of BMI (P value <0.05). In addition to that, statistically significant difference between the two groups with regard to their baseline and follow up means of systolic, diastolic blood pressure (P value <0.05) for all of them. Moreover, statistically significant difference between the two groups with regard to the presence of diabetes (P value = 0.019), and mean of estimated glomerular filtration rate (P value = 0.046). **Conclusion:** Certain factors significantly associated with decrease the possibility of LVH regression. These factors are female gender, elderly, smoking, obesity, higher systolic and diastolic blood pressure with prolonged duration, presence of diabetes and lower estimated glomerular filtration rate. Clinician should treat hypertensive patients as soon as possible with special attention to the presence of these factors.

KEYWORDS: Blood pressure, Heart, Left, Mass, Regression, Ventricle.

1- INTRODUCTION

Hypertension (HT) is a critical risk factor for the development of cardiovascular disease and a major public health concern.[1] Individuals with blood pressure levels between 115/75 and 185/115 mm Hg had a twofold increased risk of cardiovascular events for every 20 mm Hg rise in systolic pressure and a 10-mm Hg increase in diastolic pressure.[2]

Left ventricular hypertrophy (LVH) is often caused by pressure and volume overload and is linked to hypertension and aortic stenosis.[3] Catecholamines,

natriuretic peptides, and peptide hormones including angiotensin II and endothelin 1 all contribute to the development of LVH, which is characterized by myocyte hypertrophy, interstitial and perivascular fibrosis.[4] LVH is linked to increased cardiovascular morbidity and death, making it a crucial target organ for hypertension treatment.[5]

Recently, angiotensin receptor-neprilysin inhibitors have been shown to effectively reduce LVH hypertrophy.[6] SGLT2 inhibitors have been shown to lower LV mass and ambulatory blood pressure, improving the prognosis of heart failure patients. However, their relationship with better LVH requires additional exploration.[7] There are still a number of unclear issues about antihypertensive medication and LVH regression; one of the most important clinical questions is whether LVH is reversible or not.[8] Studies indicates that age, gender, chronic renal disease, obesity, and metabolic syndrome can all impact LVH regression.[9-11] LVH regression may not always occur, especially in women and obese individuals.[12]

As with the kidneys, brain, and eyes, LVH has been identified as one of the organ damages caused by hypertension.[11] Understanding LVH regression can provide insight into the pathophysiology of the condition, despite other variables being linked to it.[13] Both electrocardiography and echocardiography may measure LVH regression, which indicates an improvement in the prognosis.[14]

The aim of this study is to assess the factors associated with left ventricular hypertrophy among patients with optimum controlled hypertension.

2- PATIENT AND METHODS

This is a cross-sectional study. Included adult patients aged more than 18 years old who had hypertension based on a history of prior diagnosis and therapy, blood pressure ≥ 140/90 mmHg on two to three office visits two weeks apart, home blood pressure readings >130/80 mmHg, or a single blood pressure reading of ≥180/110 mmHg. The study patients were assessed at the medical consultation clinic of Al-Shirqat General Hospital from August 2022 to October 2025.

After excluding patients with prevalent cardiovascular disease, those with less than 24 months of follow-up, and those without LVH at baseline, the study population consisted of 400 hypertensive patients with ascertained LVH at the first echocardiogram at enrollment into the registry.

Systolic (SBP) and diastolic (DBP) pressures were measured by sphygmomanometer device in sitting position after five minutes of relaxation. Consistent with current guidelines, blood pressure was monitored three times at two-minute intervals. The office blood pressure was calculated by averaging the last two readings. Isolated systolic hypertension (ISH) is defined as baseline SBP ≥140 mm Hg and baseline DBP <90 mm Hg. Optimal blood pressure management was achieved when office SBP averaged less than 140 mm Hg and DBP averaged less than 90 mm Hg during follow-up visits.

The fasting lipid and glucose profiles were assessed using standard methods. Obesity was defined as having a BMI of 30 kg/m^2 or more, measured by dividing body weight by height in square meters. The GFR was determined using the simplified Modification of Diet in Renal Disease algorithm.[15]

All individuals' prescription antihypertensive medications were documented during first and follow up visits. Medication classes included anti-renin-angiotensin system inhibitors (ACE/ARB), calcium channel blockers (CCBs), beta blockers, and diuretics. Echocardiograms were conducted at the initial and follow-up visits using commercially available devices following a standard technique. The measurements followed the combined recommendations of the American Society of Echocardiography and the European Association of Echocardiography.[16] Carotid ultrasonography was done in a supine posture, with the neck extended in slight rotation. The scanning methodology utilized a 7.5-MHz high-resolution transducer with an axial resolution of 0.1mm. LVM was calculated using a necropsy-validated formula and indexed for height in meters to the power of 2.7 (LVMi). Relative wall thickness was calculated as posterior wall thickness/LV end-diastolic radius, and concentric LV geometry was defined as relative wall thickness ≥0.43. Regression of LVH was determined when LVMi was <50 g/m$^{2.7}$ in men and <47 g/m$^{2.7}$ in women at the time of follow-up visit.[17]

The Kolmogorov-Smirnoff test was used to assess parameter normality, while the. Quantitative data was presented as mean ± standard deviation or median (interquartile range). Qualitative data were presented as frequencies and percentages. The student's t-test was used to determine the statistical significance between the two groups in the quantitative data. The chi-square (X2) test was employed to evaluate two qualitative factors. For the nonparametric variables Mann-Whitney U test was used. A two-sided P value greater than 0.05 was considered statistically significant.

3-RESULTS

The study included 400 patients; 50 (12.5%) patients had left ventricular regression and 350 (87.5%) patients with no left ventricular regression. The mean age ± standard deviation of the study participants was 59.25 ± 10.72 years. Statistically significant difference between the two groups regarding their gender, smoking state, presence of obesity, mean of ages and mean of BMI (P value <0.05). As shown in table 1.

Table 1: Comparison between patients with no LVH regression and those with LVH regression regarding their sociodemographic and anthropometric parameters (number = 400).

Variable	No LVH regression = 350		LVH regression =50		P value
	Number	Percent	Number	Percent	
Gender: -Male -Female	 170 180	 48.5% 51.5%	 32 18	 64% 36%	**<0.001**
Smoking: - Yes - No	 136 214	 38.8% 61.2%	 11 39	 22% 78%	**<0.001**
Presence of obesity: -Yes -No	 201 149	 57.4% 42.6%	 22 28	 44% 46%	**0.012**
Mean age ± standard deviation	62.58 ± 11.72		55.26 ± 8.99		**<0.001**
Body mass index, mean ± standard deviation	34.79 ± 4.73		29.38 ± 4.69		**<0.001**

Table 2 shows comparison between the study groups regarding their blood pressure and pulse rate. Statistically significant difference between the two groups with regard to their baseline systolic, diastolic blood pressure (P value <0.05) for all of them. Furthermore, statistically significant difference between the two groups concerning their follow up systolic and diastolic blood pressure (P value <0.05) for all of them. Lastly, no statistically significant difference between the two groups regarding their baseline and follow up heart rate.

Table 2: Comparison between patients with no LVH regression and those with LVH regression regarding their blood pressure and pulse rate (number = 400).

Variable	No LVH regression = 350	LVH regression =50	P value
Duration of hypertension (years), mean ± standard deviation	8.32 ± 7.38	5.39 ± 7.23	<0.001
Systolic blood pressure baseline (mm Hg), mean ± standard deviation	148.34 ± 21.54	131.24 ± 18.32	<0.001
Diastolic blood pressure baseline (mm Hg), mean ± standard deviation	92.31 ± 11.28	81.69 ± 12.05	0.033
Mean of systolic blood pressure follow up (mm Hg), mean ± standard deviation	142.20 ± 13.29	129.39 ± 12.77	<0.001
Mean of diastolic blood pressure follow up (mm Hg), mean ± standard deviation	83.29 ± 7.39	79.27 ± 6.73	0.038
Heart rate (beat per minute) at baseline, mean ± standard deviation	75.78 ± 8.28	75.89 ± 7.39	0.649
Mean of heart rate (beat per minute) at follow up, mean ± standard deviation	77.81 ± 7.48	76.97 ± 7.44	0.891

Table 3 shows comparison between the study groups regarding their different clinical and biochemical variables. Statistically significant difference between the two groups with regard to the presence of diabetes (P value = 0.019), and mean of estimated glomerular filtration rate (P value = 0.046), while no statistically significant difference between the two groups regarding their total cholesterol, triglyceride, LDL and HDL values (P value >0.05).

Table 3: Comparison between patients with no LVH regression and those with LVH regression regarding their different clinical and biochemical variables (number = 400).

Variable	No LVH regression = 350	LVH regression =50	P value
Presence of diabetes:	49 (14%)	4 (8%)	0.019
Total cholesterol (mg/dl), mean ± standard deviation	204.12 ± 36.27	202.01 ± 35.91	0.739
Triglycerides (mg/dl), mean ± standard deviation	144.39 ± 76.81	142.49 ± 75.14	0.492
LDL (mg/dl), mean ± standard deviation	127.22 ± 36.35	128.33 ± 34.29	0.345
HDL (mg/dl), mean ± standard deviation	41.02 ± 3.32	45.63 ± 4.23	0.395
e-GFR (mL/min/1.73 m^2), mean ± standard deviation	73.68 ± 16.79	75.77 ± 15.03	0.046

Table 4 shows comparison between the study groups regarding their Echocardiographic findings. Statistically significant difference between them regarding median decrease of concentric LV geometry (P value < 0.001), mean of Carotid IMT (P value <0.001).

Table 4: Comparison between patients with no LVH regression and those with LVH regression regarding their Echocardiographic findings (number = 400).

Variable	No LVH regression = 350	LVH regression =50	P value
Decrease in concentric LV geometry (%), median (Interquartile range)	2 (1-4)	13 (3-19)	<0.001
Carotid IMT (mm) mean ± standard deviation	1.83 ± 0.74	1.55 ± 0.77	<0.001

4-DISCUSSION

The present study found that males had significant more left ventricular regression than females, which is similar to Lønnebakken et al study findings.[17] While other studies, after adjusting for baseline differences, suggest women have more favorable LV regression.[18-19] Factors such as baseline LV mass, the method used for measurement (echocardiography vs. electrocardiography), and the indexation method (for example, body height or surface area) can influence the reported outcomes. Smoking is another significant factor found in this study, this indicates that smoking hinders the heart's ability to return to a normal state, even when blood pressure is being managed with medication. Which agrees Journath et al study findings.[20] In the same way, the study explores that obese patients generally have less left ventricular hypertrophy regression than non-obese patients after a similar course of antihypertensive treatment, even when blood pressure is controlled. As obesity is an independent risk factor for LVH, and its presence can hinder the heart's ability to fully remodel and return to a normal structure in response to hypertension treatment. Lakhani et al showed similar results.[21] The study patients who had left ventricular regression are significant younger than those with no regression, indicating antihypertension treatment is more effective in younger patients consistently to Kawasoe et al study findings.[8]

On the other hand, the study found; patients with LVH regression had significantly less duration of hypertension. This finding suggests that early detection and treatment of high blood pressure are crucial, as prolonged hypertension can lead to more irreversible structural changes, such as myocardial fibrosis, which makes the heart less likely to return to a normal state. Kwiecinski et al showed parallel findings.[22] Moreover, the study found patients with LVH regression had less systolic and diastolic baseline and after treatment measures, which in agreement with Kim et al study's findings.[23]

The present study illustrates that the presence of diabetes is significantly associated with significant less LVH regression, this blunted regression is a key factor in the higher risk of cardiovascular events and mortality observed in diabetic patients with LVH. Consistent findings obtained from Mohan et al study findings.[24] Similarly, the study found that patients with lower estimated glomerular filtration rate had significant less LVH regression. Reduced renal function, as indicated by a lower eGFR, is strongly associated with the presence and severity of LVH, which is a key risk factor for cardiovascular events, which aligns with Dervisoglu et al study findings.[25]

Patients with LVH regression shown in the present study to have significantly more decrease in median concentric LV geometry and mean of Carotid IMT. Indicating a reduction in both cardiac and vascular end-organ damage associated with hypertension. Which goes with Lønnebakken et al study findings.[17]

The study limitations are; the study observational with short period of patients follow up and it had small sample size which might affect the result validity.

4- CONCLUSION AND RECOMMENDATION

Certain factors significantly associated with decrease the possibility of LVH regression. These factors are female gender, elderly, smoking, obesity, higher systolic and diastolic blood pressure with prolonged duration,

presence of diabetes and lower estimated glomerular filtration rate. Clinician should treat hypertensive patients as soon as possible with special attention to the presence of these factors.

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Conflict of intertest

About this study, the authors disclose no conflicts of interest.

REFERENCES

1. Poznyak AV, Sadykhov NK, Kartuesov AG, Borisov EE, Melnichenko AA, Grechko AV, Orekhov AN. Hypertension as a risk factor for atherosclerosis: Cardiovascular risk assessment. Frontiers in cardiovascular medicine, 2022 Aug 22; 9: 959285.
2. Rosendorff C, Lackland DT, Allison M, Aronow WS, Black HR, Blumenthal RS, Cannon CP, de Lemos JA, Elliott WJ, Findeiss L, Gersh BJ, Gore JM, Levy D, Long JB, O'Connor CM, O'Gara PT, Ogedegbe G, Oparil S, White WB; American Heart Association, American College of Cardiology, and American Society of Hypertension. Treatment of hypertension in patients with coronary artery disease: a scientific statement from the American Heart Association, American College of Cardiology, and American Society of Hypertension. Circulation, 2015 May 12; 131(19): e435-70.
3. Carabello BA. The pathophysiology of afterload mismatch and ventricular hypertrophy. Structural Heart, 2021 Sep 1; 5(5): 446-56.
4. Đorđević DB, Koračević GP, Đorđević AD, Lović DB. Hypertension and left ventricular hypertrophy. Journal of Hypertension. 2024 Sep 1; 42(9): 1505-15.
5. Nadar SK, Lip GY. The heart in hypertension. Journal of Human Hypertension, 2021 May; 35(5): 383-6.
6. Yamamoto K, Rakugi H. Angiotensin receptor-neprilysin inhibitors: comprehensive review and implications in hypertension treatment. Hypertension research, 2021 Oct; 44(10): 1239-50.
7. Cardoso R, Graffunder FP, Ternes CM, Fernandes A, Rocha AV, Fernandes G, Bhatt DL. SGLT2 inhibitors decrease cardiovascular death and heart failure hospitalizations in patients with heart failure: a systematic review and meta-analysis. EClinical Medicine, 2021 Jun 1; 36.
8. Kawasoe S, Ohishi M. Regression of left ventricular hypertrophy. Hypertension Research, 2024 May; 47(5): 1225-6.
9. Sági B, Késői I, Vas T, Csiky B, Nagy J, Kovács TJ. Left ventricular myocardial mass index associated with cardiovascular and renal prognosis in IgA nephropathy. BMC nephrology, 2022 Aug 16; 23(1): 285.
10. Drożdż M, Moczulska A, Rudziński A, Drożdż D. Metabolic syndrome as risk factor for left ventricular hypertrophy in children with chronic kidney disease. Frontiers in Endocrinology, 2023 May 31; 14: 1215527.
11. Tang L, Li S, Guo X, Lai J, Liu P, Fang J, Liu X. Combinative predictive effect of left ventricular mass index, ratio of HDL and CRP for progression of chronic kidney disease in non-dialysis patient. International Urology and Nephrology, 2024 Jan; 56(1): 205-15.
12. Talib A, Roebroek YG, Paulus GF, van Loo K, Winkens B, Bouvy ND, van Heurn EL. Left ventricular geometrical changes in severely obese adolescents: prevalence, determinants, and clinical implications. Pediatric cardiology, 2021 Feb; 42(2): 331-9.
13. Martin TG, Juarros MA, Leinwand LA. Regression of cardiac hypertrophy in health and disease: mechanisms and therapeutic potential. Nature Reviews Cardiology, 2023 May; 20(5): 347-63.
14. Du Z, Xing L, Ye N, Lin M, Sun Y. Complementary value of ECG and echocardiographic left ventricular hypertrophy for prediction of adverse outcomes in the general population. Journal of Hypertension, 2021 Mar 1; 39(3): 548-55.
15. Swart MJ, Bekker AM, Malan JJ, Meiring A, Swart Z, Joubert G. The simplified modification of diet in renal disease equation as a predictor of renal function after coronary artery bypass graft surgery. Cardiovasc J Afr, 2010 Jan-Feb; 21(1): 9-12.
16. Edvardsen, T., Asch, F.M., Davidson, B., Delgado, V., DeMaria, A., Dilsizian, V., Gaemperli, O., Garcia, M.J., Kamp, O., Lee, D.C. and Neglia, D., 2022. Non-invasive imaging in coronary syndromes: recommendations of the European Association of Cardiovascular Imaging and the American Society of Echocardiography, in collaboration with the American Society of Nuclear Cardiology, Society of Cardiovascular Computed Tomography, and Society for Cardiovascular Magnetic Resonance. *European Heart Journal-Cardiovascular Imaging*, *23*(2): pp.e6-e33.
17. Lønnebakken MT, Izzo R, Mancusi C, Gerdts E, Losi MA, Canciello G, Giugliano G, De Luca N, Trimarco B, de Simone G. Left ventricular hypertrophy regression during antihypertensive treatment in an outpatient clinic (the Campania Salute.
18. Network). Journal of the American Heart Association, 2017 Mar 8; 6(3): e004152.
19. Bella JN, Palmieri V, Wachtell K, Liu JE, Gerdts E, Nieminen MS, Koren MJ, Zabalgoitia M, Wright JT, Dahlöf B, Devereux RB. Sex-related difference in regression of left ventricular hypertrophy with antihypertensive treatment: the LIFE study. J Hum Hypertens, 2004 Jun; 18(6): 411-6.

20. Okin PM, Gerdts E, Kjeldsen SE, Julius S, Edelman JM, Dahlöf B, Devereux RB; Losartan Intervention for Endpoint Reduction in Hypertension Study Investigators. Gender differences in regression of electrocardiographic left ventricular hypertrophy during antihypertensive therapy. Hypertension, 2008 Jul; 52(1): 100-6.
21. Journath G, Nilsson PM, Petersson U, Paradis BA, Theobald H, Erhardt L. Hypertensive smokers have a worse cardiovascular risk profile than non-smokers in spite of treatment–A national study in Sweden. Blood pressure, 2005 Jul 1; 14(3): 144-50.
22. Lakhani M, Fein S. Effects of obesity and subsequent weight reduction on left ventricular function. Cardiol Rev, 2011 Jan-Feb; 19(1): 1-4.
23. Kwiecinski J, Lennen RJ, Gray GA, Borthwick G, Boswell L, Baker AH, Newby DE, Dweck MR, Jansen MA. Progression and regression of left ventricular hypertrophy and myocardial fibrosis in a mouse model of hypertension and concomitant cardiomyopathy. Journal of Cardiovascular Magnetic Resonance, 2020 Jan 20; 22(1): 57.
24. Kim HM, Hwang IC, Choi HM, Yoon YE, Cho GY. Prognostic implication of left ventricular hypertrophy regression after antihypertensive therapy in patients with hypertension. Frontiers in Cardiovascular Medicine, 2022 Dec 20; 9: 1082008.
25. Mohan M, Dihoum A, Mordi IR, Choy AM, Rena G, Lang CC. Left ventricular hypertrophy in diabetic cardiomyopathy: a target for intervention. Frontiers in cardiovascular medicine, 2021 Sep 29; 8: 746382.
26. Dervisoglu E, Kozdag G, Etiler N, Kalender B. Association of glomerular filtration rate and inflammation with left ventricular hypertrophy in chronic kidney disease patients. Hippokratia, 2012 Apr; 16(2): 137-42.