Echocardiographic Findings in Essential Hypertensive Patients Influence of Period on Left Ventricular Hypertrophy Development

نتائج تخطيط صدى القلب لمرضى ارتفاع ضغط الدم ومدى تأثير فترة المرض على تضخم البطين الأيسر

Balqis Al-sayeed Abdulla Ebrahim [1]

Khadika Shafiq Aman [2]

Osam Saeed Abdo Gabli [3]

Walid Nasar Abdulla [4]

(1) (2) (3) Ass. Professor, Internal medicine

(4) Assoc. Professor, Community medicine


2003
١٤٢٤
جامعة الأندلس
للعلوم والتقنية
Alandalus University For Science & Technology

(AUST)

ISSN : 2410-7727

Echocardiographic Findings in Essential Hypertensive Patients Influence of Period on Left Ventricular Hypertrophy Development

Abstract :

Aim of study: This work was done to assess the effect of the duration of arterial hypertension (AHT) on the progression of left ventricular hypertrophy (LVH). An echocardiographic study on left ventricular dysfunctions in a group of hypertensive patients in 22 May Hospital in Aden Governorate.

Method:Echocardiographic study for sixty-one hypertensive patients was done in a cross sectional manner which included 61 hypertensive patients. left ventricular structure and function were studied with B - mode, M-mode and Doppler echocardiography. **Results** :47.6% of the study group were with history of AHT of more than 5 years' duration, while 39.3% were having AHT of 1-4 years' duration. however only 13.1 of them were hypertensive for less than 1-year duration. Mild (grade I) left ventricular impaired relaxation occurred in more half of the cases (57.4). However only 2 (3.3) had sever (grade III) left ventricular impaired relaxation. There was statistically significant correlation between duration of AHT and left ventricular dimension in diastole (LVEDD) (-0.22/0.049), and less significant with left ventricular posterior wall (PLVWD), (P value 0.17/0.09) while there were no statistically significant correlations with inter ventricular septum in diastole (IVSD), (P value 0.048/0.36) as well as left ventricular ejection fraction (EF), (P value 0.18/0.08) and short fraction (SF), (P value 0.114/ 0.193). **conclusion:** Duration of AHT alone has no significant role in progression of left ventricular hypertrophy. Other factor like: age, sex, severity of AHT and adherence to treatment should be take into consideration of further studied need to follow these parameters and another consistent echo cardiac – evaluation of LVH like left ventricular mass indexed to body surface area is mandatory

Keywords: Hypertensive patients, echocardiography, left ventricular hypertrophy.

ISSN : 2410-7727

Introduction:

Left ventricular hypertrophy is an important risk factor in cardiovascular disease and a common consequence of chronic hypertension. Although the hypertrophic response can be considered an adaptive mechanism in the initial stages, its progression is associated with increased cardiovascular morbidity and mortality rates. (1)

The studies have shown the health benefits of lowering of blood pressure (BP) and the efficacy of specific lifestyle and pharmacological interventions, but hypertension is still a major health problem world-wide. (2)

Hypertension is a mass non-infectious disease with a prevalence of over 20% of adult population. (3) At some point in the natural history of hypertension, the compensatory increase in left ventricular mass (LVM) ceases to be beneficial. It becomes a preclinical disease and an independent risk factor for congestive heart failure, ischemic heart disease, arrhythmia, sudden death, and stroke. (4)

One of the earliest adaptation mechanisms to increase after load is LVH. However, the hypertrophy is an adaptation very quickly changes into a pathological condition. Nowadays there is a common agreement that LVH is an independent risk factor for cardiovascular morbidity and mortality. High BP is only one of the factors in hypertension which lead to LVH. A significant role in the development of LVH can be played by many other un hemodynamic factors such as: age, sex, sympathetic nervous system, renin-angiotensin system, genetic factors and overweight. (5) Long-lasting, essential hypertension is usually associated with LVH and impairs left ventricular diastolic function. (6)

Echocardiography is by far the most sensitive and accurate method for the diagnosis of LVH and more particularly, M-mode and two-dimensional echocardiography(7),which provide comprehensive wall, chamber, and LVM measures, together with systolic and diastolic performance indices, while remaining cheap, widely available, and wholly sophisticated and more accurate techniques, such as magnetic resonance imaging or computerized tomography, are inevitably more expensive and time-consuming, and of limited availability. (8)

ISSN : 2410-7727

Doppler trans mitral flow velocities expressed as the early (E) to a trial (A) wave ratio reveal LVH as a state of potential or actual myocardial ischemia. All antihypertensive drugs regress LVH, notably the angiotensin-converting enzyme inhibitors, which may also target the detrimental tissue changes. (9)

Demographically Age, gender, race, and body size can all influence LVM, possibly mediated via cardiac load. Thus, LVH prevalence increases with age, in both hypertensive and normotensives, perhaps due to the combination of age-related BP elevation and declining aortic compliance. Similarly, there is a gender difference in LVM, which becomes evident in adolescence and remains constant during adult life; although the age-related increased in LVM is greater in postmenopausal women than in men, gender is not a significant determinant of cardiovascular complications or of the prognostic impact of LVH. Hypertensive LVH is more evident in blacks than in whites at similar increases in blood pressure; certain cardiovascular complications, such as heart failure and sudden death, are also more common in blacks. Body size, notably obesity, which compounds hemodynamic load independently of a clear-cut increase in BP, is a major determinant of LVM, with dietary sodium, it is associated with increased plasma volume and cardiac output, and may be responsible for hypertensive LVH. (10)

Uncontrolled and prolonged elevation of BP can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of LVH ,coronary artery disease, various conduction system diseases, and systolic and diastolic dysfunction of the myocardium, complications that manifest clinically as angina or myocardial infarction, cardiac arrhythmias especially atrial fibrillation and congestive heart failure. (11)

Guide lines on the assessment of cardiovascular risk, released in late 2013 by the American Heart Association/American College of Cardiology , recommend use of a revised calculator for estimating the 10-year risk of developing a first atherosclerotic cardiovascular disease event, which was defined as nonfatal myocardial infarction, death from coronary heart disease, or stroke (fatal or nonfatal) in a person who was initially free from atherosclerotic cardiovascular

ISSN : 2410-7727

disease. The calculator uses clinical and laboratory risk factors, including systolic BP and treatment for hypertension. (12)

The increased risk of cardiovascular events with LVH depends on its type. Concentric LVH poses the greatest risk of such events, as much as a 30% risk over a 10-year period in one study, compared with a 15% risk with eccentric remodeling and a 9% risk without any LVH. (13) The degree of LVH, as assessed by LVM index was also related to the cardiovascular mortality rate, with a relative risk of 1.73 for men and 2.12 for women for each $50g/m^2$ increase in the Left ventricular mass index over a 4-year period. With LVH, the relative risk of mortality is increased 2-fold in patients with coronary artery disease and 4-fold in patients without coronary artery disease. (14)

Cardiac hypertrophy is associated with a decrease in coronary reserve. However, factors which may modulate the interaction between myocardial growth and vascular proliferation, such as duration and severity of hypertrophy, have not been evaluated. (15) Although any type of LVH increases the incidence of cardiovascular disease, the concentric type of LVH has been identified as the cardiac structural parameter that was most strongly related with cardiovascular risk. (16)

LVH is a major maladaptive response to chronic pressure overload and an important risk factor in patients with hypertension. The development of LVH is highly correlated with systolic hypertension. In the Framingham Heart Study, even borderline isolated systolic hypertension at an elderly age was associated with increased left ventricular wall thickness and impaired diastolic filling. (17)

While elevated systemic arterial pressure plays a role in the pathogenesis of LVH, the extent of cardiac growth and response to increased pressure loading is not uniform among patients suggesting genetic mechanisms in cardiac hypertrophy. (18)

Since not all patients with hypertension develop LVH, there were clinical findings that should be kept in mind that may alert the physician to the presence of LVH so a more definitive evaluation can be performed. The detection of LVH is important because these patients' risk of cardiovascular morbidity and mortality is two-to-four-fold increase compared to patients with normal LVM. (19) Changes in LVM can indicate disease progression or regression in hypertensive

ISSN : 2410-7727

heart disease. Cardiovascular magnetic resonance was superior to echocardiograph because of excellent reproducibility and because echocardiographic methods were dependent on symmetry on left ventricular shape, (20) of cost echocardiograph was more commonly used in clinical assessment of hypertensive patients. However, measuring changes in LVM using cardiovascular magnetic resonance has become a precise useful research tool when testing the benefit of pharmacologic treatment of LVH hypertrophy. (21)

It was appreciated that LVH is mediated not only by the mechanical stress of pressure overload, but also by various neuro hormonal substances that independently exert trophic effects on myocytes and non-myocytes in the heart. (22)

Methods:

This study was comprised 61 hypertensive patients (37 men, 24 women; mean age,55.3 **(**SD±11.8 years**)** years; range, 31 to 77) with mild to moderate essential hypertension evaluated in 22 May Hospital. They were recruited for participation in a clinical research and informed consent was obtained from each participant.

All patients underwent two-dimensional echocardiography,M-mode and Doppler ultrasound. BP measurements was documented.

Echocardiographic study :

Echocardiographic examination was performed with a Hewlett-Packard imaging system (Sons 1000). Complete M-mode, two-dimensional, and pulsed-wave Doppler echocardiographic studies were obtained. The tracings were recorded and tape at 50 mm/sec. At the end of the study, echocardiograms were numerically coded and read in a random sequence by two physicians according to the recommendation of the American Society of Echocardiography. Quantitative analysis of M-mode echocardiograms provided the following parameters, thus allowing the assessment of left ventricular anatomy and function: end-diastolic and end-systolic diameters, inter ventricular septum and posterior wall thickness in diastole at three sequential levels: the mitral valve, papillary muscles, and apex. Fractional shortening was used as an index of systolic function. Diastolic function was assessed by calculating the early diastolic

ISSN : 2410-7727

filling velocity (E wave), late diastolic filling velocity (A wave), and E-to-A ratio (<1 being indicative of diastolic dysfunction).

Results :

In this study the distribution of the studied sample regarding their type of sex it was found that more than half (60.7%) of patients were male and the rest (39.3 %) of patients were female. (Table 1)

Regarding their age, it was noticed that the mean age of the patients was 55.3years (SD±11.8 years) the maximum age was 77 years and the minimum was 31 years, the majority of the patients (65.6%) aged 50 years and more. (Table 2)

Distributions of studied samples as regards hypertension duration it was observed that Approximately 27.9% of the patients were suffering of blood hypertension disease since 5 years and more, while 39.3% of the patients suffering of hypertension for 1-4 years, only 13.1% of the patients were suffering less than one year. (Table 3)

The studied samples according to E/A ratio revealed that more than half (57.4%) of patients were E/A ratio mild, followed by normal E/A ratio (29.5%) and moderate E/A ratio (9.8%) while sever cases represented only (3.3%) of total. (Table 4)

Left ventricle was studied by echocardiographic, there was statistically significant increased in correlation between LVEDD and period of blood hypertension in patients (P -value= -0.22/0.049) when compared with PLVWD and IVSD parameter (correlation were statistically less significant (P-value=0.17/0.09) and no significant P-value=0,048/0.36) respectively. (Table 5)

There was statistically less significant correlation as measured by echocardiograph between parameters (EF and SF) and AHT duration in which the P-value were (0.18/0.08 and 0.114/0.193) respectively. (Table 5)

Table No 1. Distribution of patients according to sex:

Sex	Number (n=61)	Percent %
Male	37	60.7
Female	24	39.3

ISSN : 2410-7727

Table No 2. Distribution of patients according to age:

Age (years)	Number (n=61)	Percent %
31-39	5	8.2
40-50	16	26.2
>50	40	65.6

Table No 3. Distribution of patients according to duration of hypertension:

Period (years)	Number (n=61)	Percent %
< 1	8	13.1
1-4	24	39.3
5-14	17	27.9
≥ 15	12	19.7

Table No 4. Distribution of patients according to E/A ratio:

E/A ratio	Number (n=61)	Percent %
Normal	18	29.5
Mild (Grade I)	35	57.4
Moderate (Grade II)	6	9.8
Sever (Grade III)	2	3.3

Table No 5. Cardiac parameters according to echocardiographic measurement:

variable	Mean	SD	Period (r/P value)
IVSD	11.7	3.007	(0,048/0.36)
LVEDD	47.9	7.85	(-0.22/0.049)
PLVWD	9.64	4.62	(0.17/0.09)
EF	56,0	10.7	(0.18/0.08)
SF	0.29	0.05	(0.114/0.193)
IVSD = interventricular septal diameter, LVEDD = left ventricle end diastolic diameter, PLVWD = posterior left ventricle wall diameter, EF = ejection fraction, SF=short fraction, r=partial correlation, df=58, one tail significance.			

ISSN : 2410-7727

Discussion :

In these samples of patients of all age with AHT 60.7% of men and 39.3% of women were found to have LVH as defined by tow – dimension and M- mode echocardiography. The main predictors of LVH in this study of 61 subjects were age, gender, period of hypertension and echocardiography parameters of LVH.

The study we found that LVH is a more prevalent among men (60.7) than in women (39.3%).

levy et al, noted, however, that sex difference was not statistically significant for any of the internal criteria but that LVH prevalence were significantly higher in women when using external criteria for LV mass (P <0.001). (23) Simone et al, reported that, sex difference in cardiac size in adults may also mostly reflect differences in the hemodynamics and metabolic demands of men and women. (24)

The present study showed that, LVH distributed between old age (65.6 %) and middle age (26.2%) more than between younger ages (8.2 %).

The influence of aging on AHT should be taken into consideration, LV wall thickness has been showing to increase with progression aging, even when blood pressure remains in the normotensive range, however, age-dependent changes increase LV wall thickness by only about 25% between the second and seventh decade. In contrast to wall thickness, LV diastolic diameter does not correlate with age, aging may primary cause concentric LVH. (25)

Mania et al, stated that, even borderline isolated systolic hypertension at an elderly age was associated with increase LV wall thickness and impaired diastolic filling. (26)

Richard et al, estimated that, the disagreement over all age groups was highly significant (P < 0.001). LVH were estimated higher with prevalence in the younger age groups and lower in the older age groups (P = 0.046). (27)

In the present study, there were no significant differences between the two groups, but it should be noted that groups had LVH that differed in severity only.

ISSN : 2410-7727

In this study E/A revealed a significant change in velocity which consider that diastolic dysfunction accompanied LV filling showed different variation among our studied patients.

The test, showed an important variation in E and A waves index, and in the ratio between both waves (E/A) in view of changes induced by LV preload.

The finding of gradually increasing prevalence of LVH over the whole distribution of E/A ratio may thus have clinical implication for a substantial part of the population. This consider that increasing in diastolic dysfunction which usually accompanied by progressive increase in LV filling pressure which in turn have a major impact on the transmitted flow profile.

Simpson et al, observed in twenty outpatients with arterial hypertension in different period of time a significant change in E/A velocity ratio measured by transmittal flow with standard relaxation deficit (P value 0.01). (28)

Statistical analysis in this study revealed that EF no significant changes (0.18/0.08) in velocity although of significant diastolic dysfunction accompanied LV filling, as well as Kimand Sohn, found that majority of his studied group (>50%) with different period of AHT and LV dysfunction was with normal EF. (29)

The most important variable for categorizing subjects as having

LV H was the echocardiographic parameters (IVSD, LVEDD, PLVWD) period of hypertension as important variable had been previously documented in our selected subjects.

The synergistic association between LVEDD and period of hypertension with LVH has seldom been a focus of population – based studies. Slight association found between PLVWD, and the period of hypertension, while IVSDو EF and SF have no association.

Tatiana et al, studied samples of random of general population, the overall prevalence of LV diastolic dysfunction with LVEDD (69.1_+9.2), estimated that, echocardiographic measurements were as high as 27.3% and increased in frequency with duration of hypertension. (30)

ISSN : 2410-7727

Wangler et al, found that LV mass in the diastolic hypertension was increased significantly (P-value≤ 0,05) by 14% and 29% at 3,7 and 15 months respectively. (31)

Naoto, had been reported that, development of hypertrophy, particularly in elderly person, was closely related to the presence of hypertension, and that frequency of hypertrophy was also higher among young patients or those borderline hypertensions. (32)

Reference:

1. Ram CV, Gonzalez D, Kulkarni P, Sunderajan P, Corbett J et al. (1989) Regression of left ventricular hypertrophy in hypertension. Effects of prazosin therapy. Am Med J .86,pp 66-9
2. Jostein H , Turid L H , Aage T , Oddgeir L et al. (2016) Blood pressure changes during 22-year of follow-up in large general population. Cardiovasc Disord J .pp16: 94
3. Joint National Committee (1997) on Prevention, Detection, Evaluation and Treatment of High Blood Pressure. The sixth report of the Joint national committee on prevention, detection, evaluation and treatment of high blood pressure. Arch Intern Med J .157, pp 2413-46
4. Frohlich ED. (1999) Risk mechanisms in hypertensive heart disease. Hypertension J . 34, pp782-89
5. Stamler J. (1991); Implications of the INTERSALT study. Hypertension J.17, pp16 – 20
6. Monika Możdżan M , Wijerzbowska-Drabik K, Kurpesa M,et al. (2013) Echocardiographic indices of left ventricular hypertrophy and diastolic function in hypertensive patients with preserved LVEF classified as dippers and non-dippers. Arch Med Sci J. 9, pp 268– 75
7. Reichek N and Devereux RB. (1996) Left ventricular hypertrophy: relationship of anatomic, echocardiographic and electrocardiographic findings. Circulation J. 63, pp1391-1398
8. Agabiti-Rosei E and Muiesan L. (2005) Hypertension and left ventricular Hypertrophy. Cardiovascular Medicine J. 10, pp 1-61

ISSN : 2410-7727

9. Jacob K, Moller E, James B. (2000) Ratio of left ventricular peak E-wave velocity to flow progression velocity assessed by color M-Mode Doppler echogardiography. AM Cardiol J.35, pp363-370.
10. Liebson PR, Savage DD. (1986) Echocardiography in hypertension: a review. I. Left ventricular wall mass, standardization, and ventricular function. Echocardiography J. 3, pp 181-218
11. Cabezas M, Comellas A, Ramon Gomez J, et al. (1997) Comparison of the sensitivity and specificity of the electrocardiography criteria for left ventricular hypertrophy. Cardiol J. 50, pp31-5.
12. Chobanian AV, Bakris GL, Black HR, et al. (2003) The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. JAMA J. 289, pp 60-72.
13. Liao Y, Cooper RS, Mensah GA, McGee DL. (1995) Left ventricular hypertrophy has a greater impact on survival in women than in men. Circulation J. 92, pp 805-10
14. Murilo Foppa, Bruce B Duncan and Luis EP Rohde. (2005) Echocardiography-based left ventricular mass estimation. How should we define hypertrophy?Cardiovascular Ultrasound J. 3, pp 17
15. Kofflard M, Michels M, Krams R, Kliffen M et al. (2007) Coronary flow reserve in hypertrophic cardiomyopathy: relation with microvascular dysfunction and pathophysiological characteristics**.** Neth Heart J.15, pp 209 – 15
16. Paulus W,Tschope C,Sanderson J,Rosconi C et al .(2007) How to diagnose heart failure with normal left ventricular ejection fraction by the Echocardiography. Eur Heart J.28, pp 2539 - 50
17. Sagie A, Benjamin EJ, Galderisi M, et al. (1993) Echocardiographic assessment of left ventricular structure and diastolic filling in elderly subjects with borderline isolated systolic hypertension (the Framingham Heart Study) American Journal of Cardiology J. 72, pp 662–665
18. Grand A and Broogi S. (2001) Left ventricular changes in isolated office hypertension: a blood pressure – matched comparison with

ISSN : 2410-7727

normotension and sustained hypertension. Internal Medicine J. 161, pp 2677-81
19. Casale PN, Devereux RB, Milner M. (1986) Value of echocardiographic measurement of left ventricular mass in predicting cardiovascular morbid events in hypertensive men. Annals of Internal Medicine J. 105, pp 173–178
20. Moon JC, Fisher NG, McKenna WJ, Pennell DJ. (2004) Detection of apical hypertrophic cardiomyopathy by cardiovascular magnetic resonance in patients with non-diagnostic echocardiography. Heart J. 90, pp 645–649
21. Olivotto I, Maron BJ, Appelbaum E, et al. (2010) Spectrum and clinical significance of systolic function and myocardial fibrosis assessed by cardiovascular magnetic resonance in hypertrophic cardiomyopathy. American Cardiology J. 106, pp261–267
22. Post W.S, Larson M. G, and Levy D. (1994) "Impact of left ventricular structure on the incidence of hypertension: The Framingham Heart Study," Circulation J.90, pp 179–185b
23. Levy D, Garrison RJ, Savage DD, Kannel WB, Castelli WP. (1990) Prognostic implications of echocardiographic ally determined left ventricular mass in the Framingham Heart Study. N Engl Med J. 322, pp 1561-66
24. De Simone G, Devereux RB, Roman MJ, et al. (1994) Relation of obesity and gender to left ventricular hypertrophy in normotensive and hypertensive adults. Hypertension J. 23, pp 600-6
25. Dvila DF, Donis JH, Odreman R, Gonzalez M, et al. (2008) Landaeta A. Patterns of left ventricular hypertrophy in essential hypertension: should echocardiography guide the pharma -cological treatment? Int J Cardiol . 124, pp 134-8
26. Mancia G, De Backer G, Dominiczak A, et al. (2007) Cifkova R, Fagard R. G ESC and ESH Guidelines for the man -agement of arterial hypertension. Eur Heart J. 28, pp 1462-536
27. Richard E. Katholiand Daniel M. Couri .(2011) Left Ventricular Hypertrophy: Major Risk Factor in Patients with Hypertension: Update and Practical Clinical Applications. Int Hypertens J.20, pp 49-53

ISSN : 2410-7727

28. Simpson TE, Dansky HM, Buttrick PM. (1995) Molecular genetic mechanisms of cardiac hypertrophy. Cardiovascular J. 2, pp 93–108
29. Kim Y and Sohn D. (2000) Mitral Annulus velocity in the estimation of left ventricular pressure filling pressure prospective study in 200 patients . M Soc Echocardiogr J. 13, 980-85
30. Tatiana K, Lieven H, Tom R, Robert H et al. (2009) prevalence of left ventricular diastolic dysfunction in general population. Cir Heart Fail J.2, pp 105-12
31. Wangler RD, Peters KG, Marcus ML and Tomanek RJ.(1982) Effects of duration and severity of arterial hypertension and cardiac hypertrophy on coronary vasodilator reserve**.**Circ Res J.51,pp 10-18
32. Naoto A, Shinji S, Kouei K, Kyoko T,et al. (1989) Effects of blood pressure(changes on development and regression of electrocardiographic left ventricular hypertrophy.Journal of the American College of Cardiology J. 13,pp 165 – 72.

ISSN : 2410-7727

نتائج تخطيط صدى القلب لمرضى ارتفاع ضغط الدم ومدى تأثير فترة المرض على تضخم البطين الأيسر

الملخص باللغة العربية :

الهدف من هذه الدراسة : هو تقييم تأثير مدة ارتفاع ضغط الدم الشرياني على اختلالات البطين الأيسر

وقد شملت الدراسة ٦١ مريضاً مصاباً بارتفاع ضغط الدم الشرياني في مستشفى ٢٢ مايو في محافظة عدن. تم استخدام جهاز تخطيط صدى القلب بنوعيه (M وB) وكذلك جهاز دوبلار.

وقد اظهرت الدراسة **النتائج** التالية : ٤٨٪ من مجموعة المرضى مصابين بارتفاع ضغط الدم الشرياني لأكثر من ٥ سنوات، ٣٩.٣٪ مصابين بارتفاع ضغط الدم الشرياني لمدة ١- ٤ سنوات ، في حين أن فقط ١٣.١ منهم مصابين بارتفاع ضغط الدم الشرياني لأقل من ١ سنة. أكثر من نصف الحالات (٥٧.٤) مصابين بضعف الاسترخاء للبطين الايسر من النوع الخفيف (درجه I) بينما ٢ فقط من مجموع المرضى مصابين بضعف الاسترخاء الشديد(درجه III) وقد وجدت علاقة ذات دلالة إحصائية هامه بين مدة ارتفاع ضغط الدم الشرياني والبعد الانبساطي للبطين الأيسر القيمة الاحتمالية (- ٠.٠٤٩/٠.٢٢)، وأقل أهميه مع البعد الانبساطي للجدار الخلفي للبطين الأيسر (القيمة الاحتمالية ٠.٠٩/ ٠.١٧) في حين لم يكن هناك دلاله إحصائية مع انبساط الحاجزين البطينين (القيمة الاحتمالية ٠.٠٤٨/٠.٣٦)، كذلك لم توجد دلاله إحصائية مع المنسوب القذفي والقصير حيث إن القيمة الإحصائية (٠.١٨/٠.٠٨) و (٠.١١٤/٠.١٩٣) على التوالي.

الاستنتاج: مدة ارتفاع ضغط الدم الشرياني وحده ليس له دور كبير في تطور تضخم البطين الأيسر. عامل آخر مثل: العمر، الجنس، شدة ارتفاع ضغط الدم الشرياني والالتزام بالعلاج يجب أن تأخذ في الاعتبار الحاجة لمزيد من الدراسة الإلزامية لاتباع هذه المعايير والقلب صدى ثابت آخر - تقييم البطين الأيسر ضخم البطين الأيسر مثل كتلة مساحة سطح الجسم .