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Echocardiographic diastolic dysfunction among hypertensive patients

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Abstract

Background and objective: Hypertension is a very common medical disorder, affecting nearly 40% of our population. Diastolic dysfunction is one of complications that happens and is regarded as a predictive of subsequent heart failure. The aim of this study was to determine the frequency of left ventricular diastolic dysfunction by echocardiography among hypertensive patients.

Methods: This study involved 150 hypertensive cases (82 female and 68 male) age from 25-81 years old. Transthoracic echocardiography approach, using SIEMENS SONOLINE Omnia 2005 machine with the probe of p4-2 was used by the researcher. A comparative 150 normal non-hypertensive healthy adults with hypertensive patients are examined again by echocardiography. The parameter for the diastolic dysfunction measurement is E/A ratio.

Results: It was found that 88 (58.7%) of hypertensive patients have diastolic dysfunction, while 4.9% of non-hypertensive group have diastolic dysfunction with significant statistical differences between both groups (P <0.001), who were age >60years have 79%, while age 25-35years old have 21% (P <0.001). Among left ventricular hypertrophy patients, 87.5%, versus 12.5% have diastolic dysfunction (P <0.001). Females are more liable for diastolic dysfunction, who have 70.7% than males who have 44.1% (P <0.001).

Conclusion: Diastolic dysfunction is common among hypertensive patients, particularly among females, aged, and who have echocardiographic criteria of left ventricular hypertrophy, represent risk factors for diastolic dysfunction.

Keywords: Diastolic dysfunction, Left ventricular hypertrophy, Echocardiography, Hypertension.

Introduction

An appreciation of normal diastolic function permits a better understanding of clinical features of diastolic heart failure. Cardiac function is critically dependent upon diastolic physiologic mechanisms to provide adequate left ventricular filling (cardiac input) in parallel with left ventricular ejection (cardiac output). These processes must function under a variety of physiological conditions, both at rest and during exercise.[1] Events during diastole begin with the relaxation of the contracted myocardium. This is a dynamic, energy-dependant, process that includes two phases:

1- Isovolumic relaxation which is a period between aortic valve closure and mitral valve opening during which left ventricular pressure declines with no change in volume.

2- Autonomic relaxation which is a period between mitral valve opening and mitral valve closure during which the left ventricle fills at variable pressure.[2]

Diastolic dysfunction (DD) is characterized by elevated diastolic pressure in left ventricle despite normal or sub-normal diastolic volume. [3]Hypertrophy of cardiac cells, increased interstitial collagen deposition or infiltration of the myocardium with amyloid proteins causes decreased distensability of the cardiac tissue. The ventricle then behaves as a balloon made

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from abnormally thick rubber. Despite filling with high pressure, the volume cannot expand adequately. If the heart cannot be filled with blood easily, either the cardiac output becomes diminished or compensated increase the ventricular diastolic pressure to higher levels.[4] When the left ventricular diastolic pressure is elevated, venous pressure in the lungs must also become elevated to maintain forward flow. Increased pulmonary venous pressure results in alveolar oedema making the patient to become shortness of breath.[5] Echocardiography provide information regarding global diastolic function and is not affected by increasing heart rate or atrial fibrillation.[6] Hypertensive patients in our locality, they were poorly compliance to the treatment, so we want to study the effect of this poor compliance on DD. Same thing detected in near developing countries.[7,8]

The aim of this study was to study the prevalence of left ventricular diastolic dysfunction detected by echocardiography among hypertensive patients in Erbil city.

Methods

The study was performed during 1st of February2012 till 1st of March 2013 in Rozh-halat Emergency Hospital, Erbil city, Kurdistan Region, Iraq.

Subjects:

One hundred fifty hypertensive patients (68 males and 82 females) compared with one hundred fifty non hypertensive patients were involved in this study, age ranges from 25-81 years old.

Exclusion criteria:

Ischemic heart disease, poor Echo window, valvular heart disease, cardiomyopathy (specially hypertrophic cardiomyopathy), congenital heart disease, diabetes mellitus, chronic Kidney disease, systolic heart failure, atrial fibrillation, severe chronic obstructive pulmonary disease, pericardial disease and those who take more than one antihypertensive drugs are excluded from the study. All the exclusion criteria's were excluded by history, physical examination, echocardiography, and other blood investigations.

Physical examination and investigation:

History and clinical examination, vital signs were taken by the researcher (specialist physician). Blood pressure was measured by Mercury sphygmomanometer, systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg on the average of two or more readings regarded as hypertension.[9] All our patients were previously diagnosed and treated for hypertension, and control group were totally free of any medical disease. Pulse rate, respiratory rates were taken once. SIEMENS SONOLINE Omnia 2005 Echo machine with p2-4 probe Echocardiography was used. Definition for left ventricular hypertrophy in diastole, that depended in this study was septal thickness >10.9mm and posterior wall thickness>11.9mm.[10] All patients and control group were examined in the left lateral position, pulse waved Doppler Mitral inflow velocities were recorded by placing sample volume at the tips of the Mitral valve. The transmitral peak early diastolic velocity (E), peak late diastolic velocity (A), E wave deceleration time (DT) and E/A ratio were measured. Isovolumic relaxation time (IVRT) was recorded from apical 5- chamber view by simultaneously recording of the mitral and aortic flows. Tissue Doppler Imaging was performed by activating the TDI function, to assess the diastolic function, two velocities; peak early diastolic velocity E and peak late diastolic velocity A at Mitral annulus was determined. Four different sites on the mitral annulus i.e. Lateral, Anterior, Septal and Inferior were selected. For lateral and septal sites apical 4-chamber view and for anterior and inferior sites apical 2-chamber views were utilised. Mean values from above four sites were used to assess global diastolic left ventricular function. Normal peak E-wave is (m/sec)0.68±0.15, peak A-wave is (m/sec)0.69±0.17, E/A ratio is 1.04±0.34, IVRT(m/sec) 80±20, DT (m/sec) 184±24.[11]

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Ethical considerations:
Approval was taken from the Research Ethics Committee of the Medical Research Centre, Hawler Medical University. Participants were fully informed about the methods and objectives of the study.

Statistical Analysis:
Statistical package for the social sciences (version 20) was used for analysis of the data. Student's t-test was used to compare between means. Unpaired t-test was used to compare between readings of case and control group. Chi square test of association (or McNemar test) was used to compare between proportions. A p-value of ≤0.05 was considered as statistically significant.

Results

It was found that 88 (58.7%) of hypertensive cases have DD, while 7 (4.7%) of control group have DD with statistical significance (P <0.0001) as shown in Table 1.

The proportion of DD increases with the progression of patients' age, with significantly association between them (P <0.001) as shown in Table 2.

Female gender cases showed more DD (70.7%) as compared with nearly same matched age group male cases (44.1%) with a significant difference between male and female (P <0.001) as shown in Table 3.

Mean body mass index in male hypertensive group was 28.2; females were 29.7, while among control group 24.1, and 24.98 respectively. Morbid obesity was found in 13 patients, and only two in control group.

Regarding the types of antihypertensive drugs that given to the patients, its shown that patients on angiotensin converting enzyme inhibitors, have the lowest proportion of DD (49%) in comparison with other treatments, while beta-blocker groups have the highest proportion (81%) but the difference statistically was not significant, Table 4.

Table 1: Proportion of diastolic dysfunction among hypertensive cases as compare to matched age control group.

Group	DD[%]		Total	p
	Present No. [%]	Absent No. [%]		
Hypertensive	88[58.7]	62[41.3]	150	0.001
Control	7[4.7]	143[95.3]	150	
Total	95[31.7]	205[68.3]	300	

Table 2: Age and diastolic dysfunction.

Age (years)	DD[%]		Total	p
	Present No. [%]	Absent No. [%]		
25-35	7[21]	26[79]	33	0.001
36-45	6[43]	8[57]	14	
46-55	9[56]	7[44]	16	
56-65	29[72.5]	11[27.5]	40	
66-75	21[78]	6[22]	27	
>75	16[80]	4[20]	20	

Table 3: Gender and diastolic dysfunction.

Gender	DD[%]		Total	p
	Present No. [%]	Absent No. [%]		
Male	30[44.1]	38[65.9]	68	0.001
Female	58[70.7]	24[29.3]	82	
Total	88[58.7]	62[41.3]	150	

Table 4: Medication used by the patients.

Drugs	DD[%]		Total	p
	Present No. [%]	Absent No. [%]		
Angiotensin receptor blocker	20[49]	21[51]	41	0.3
Angiotensin converting enzyme inhibitor	17[47.5]	19[52.5]	36	0.3
Calcium channel blocker	14[53.5]	12[46,5]	26	0.8
Beta-blocker	13[81]	3[19]	16	0.13
Diuretics	24[77]	7[23]	31	0.08

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Left ventricular hypertrophy which could be seen in hypertensive cases and detected by echocardiography are found to be significantly increases the proportion of DD (87.5%) compared to (37.2%) with normal ventricular group that has DD (P <0.001) and shown in Table 5.

Selected wall thickness in diastole, specifically interventricular septal thickness increased to 86 (76%) among hypertensive with DD compared to only two (5.5%) of normal septum with DD (P <0.001). Posterior wall thickness increased to 57 (74%) of cases with DD compared to 31 (42.5%) of normal posterior wall with DD (P <0.001) as shown in Table 6.

Table 5: Left ventricular hypertrophy and diastolic dysfunction.

Group	DD[%] Present No. [%]	Absent No. [%]	Total	p
Left ventricular hypertrophy	56[87.5]	8[12.5]	64	
Normal ventricular wall	32[37.2]	54[62.8]	86	0.001
Total	88[58.7]	62[41.3]	150	

Table 6: Diastolic dysfunction according to septal and posterior wall thickness.

Wall thickness	DD[%] Present No. [%]	Absent No. [%]	Total	p
Increase septal thickness	86[76]	27[24]	113	
Normal septal thickness	2[5.5]	35[94.5]	37	0.001
Increase posterior wall	57[74]	20[26]	77	
Normal posterior wall	31[42.5]	42[57.5]	73	0.001

Discussion

The study shows that among hypertensive patients, 58.7% of cases have DD, while only 4.9% of non-hypertensive patients have DD. Levy *et al* showed that DD is more common in hypertensive patients by more than three folds in comparison to normotensive group, that's nearly the same result was found in the present study.[12] The occurrence of DD in hypertensive patients increases with age, in which 80% of patients with DD their age was more than 70 years old, Donald *et al* showed that the incidence of DD in the 80 years of age is double risk than in the 40 years of age.[13] In this study female patients have more DD (70.7%) than male patients (44.1%) with a significant difference (P<0.001), Masoudi *et al* showed 79% of DD among female patients versus 21% among male patients.[14] A greater decline in diastolic function is seen in hypertrophied hearts, the result shows that increase in the septal thickness is a risk for development of DD, since 76% of those with increased septal thickness have DD and74% of those with increased posterior wall thickness have DD (P<0.001). Drazner *et al* showed 75% DD for both septal and posterior wall hypertrophy.[15] Among patients with left ventricular hypertrophy, 87.5% have DD, while only 12.5% have no DD, the reason may be due to the fact that 12.5% have no DD probably due to lack in estimation of left ventricular mass for diagnosis of left ventricular hypertrophy which is more accurate (P <0.001). Koren *et al* proved that upon 10years of follow up of hypertensive patients with left ventricular hypertrophy, and hypertensive patients without left ventricular hypertrophy re-vealed that 76% have DD in left ventricular hypertrophy group, while only 12% have DD in non-left ventricular hypertrophy group.[16] Drugs which were taken by the patients, for controlling hypertension, beta-blockers associated with 81% of DD, while only 19% of them have no DD, the difference was not significant statistically, same result founded by Che *et al*.[17]

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Among hypertensive patients receiving angiotensin converting enzyme, 47.5% of them have DD, while 52.5% of them have no DD, the difference was not significant statistically. Angiotensin receptor blocker groups showed that 49% of patients have DD, while 51% have no DD, again with non-significant differences; Warner *et al* noticed an improvement of DD with losartan treatment.[18] Calcium channel blocker group showed that 53.5% of patients have DD, while 46.5% of them have no DD, with non-significant difference, William *et al* showed certain calcium channel blockers improve DD.[19] This difference is possibly because of patient's noncompliance to the treatment. The study shows that 77% of patients using thiazide diuretics have DD, while 23% of them have no DD, with no significant differences (P <0.08), but Mottran *et al* showed that hypertensive patients with DD were given spironolactone 25mg/day for 6 months showed clinical improvement and echocardiographic criteria by decreasing in the septal and posterior wall thickness.[20]

Conclusion

Diastolic dysfunction is common among hypertensive patients and regarded as a risk factor, especially if this associated with echocardiographic criteria for left ventricular hypertrophy, and more common among older age and female sex patients.

Conflicts of interest

The author reports no conflicts of interest.

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