Assessment of Left Ventricular Myocardial Stiffness in Hypertension Patients Utilizing Conventional and Tissue Doppler Echocardiography

Hind Hasan Abed[*], *Basil Najeeb Aldileamy*[**]

ABSTRACT:

BACKGROUND:

It is predicted that changes in LV morphology in hypertensive patients will cause a reduction in compliance and an increase in myocardial stiffness. The E/E' to (LVEDD) ratio represents the pressure–volume relationship of the LV, which was used to quantify LV stiffness.

AIM OF THIS STUDY:

Assess myocardial stiffness in hypertensive patients by applying a combined conventional and (TDI) on the pressure volume relation [(E/E')/LVEDD].

METHODS:

The research included 81 hypertensive patients (48 females and 33 males) and 33 healthy individuals (23females&10males) as a control group . A complete TTE echocardiographic examination was performed on all patients, including (TDI) of the mitral annulus, measurement of (LV) septal and posterior wall thickness, LV mass, LVEDD, and transmitral Doppler peak early (E) and late (A) diastolic filling velocities. The septal and lateral sites of the mitral annulus were used to average TDI early diastolic myocardial velocities (E').

RESULTS:

In hypertensive group, mitral annular TDI early diastolic velocity E' was significantly lower than the control group's $P < 0.05$. Hypertensive group also showed a significantly higher late diastolic velocity compared with control group $p < 0.05$. The mean E' was significantly lower while the mean E/E' ratio was significantly higher and the LV stiffness index was insignificantly different in hypertensives compared with control subjects. In the hypertensive group, the LV stiffness was insignificantly correlated with IVS thickness.

CONCLUSION:

In hypertensive patients, LV myocardial diastolic stiffness index [(E/E')/LVEDD] is not so increased and is not correlated well with the LV wall thickness and LV mass index.

KEYWORDS: TDI, Hypertension patients, LV myocardial parameters

INTRODUCTION:

Hypertension can cause multiple adverse effects on both the heart and vascular system. It can increase cardiac muscle stiffness which in turn decreases compliance, as well as stiffen the peripheral arterial branches. In addition to age and gender, factors like increase in body weight, decrease physical activity, smoking, alcohol drinking , family history, elevated serum cholesterol, uncontrolled diabetes mellitus and pre-existing vascular disease can all affect blood pressure (BP) [1].

There are several reports demonstrating that left ventricular (LV) dysfunction can occur even in the absence of changes in LV systolic function in patients with hypertension[2]. In the left ventricle, functional impairment is a consequence of impaired systolic or diastolic function or both. Left ventricular Diastolic dysfunction results from conditions that alter the LV diastolic pressure-volume relation, which in turn affects the left ventricle's performance capability. With no or little systolic dysfunction, diastolic dysfunction might develop. Grade I diastolic dysfunction consider mildest form may manifest only as a slow or delayed pattern of relaxation and filling, with normal or only mild elevation of LV diastolic pressure [3]. It is possible to determine LV function based on diastolic performance. Doppler echocardiography is a simple and non-invasive method to undertake to determine left ventricular diastolic dysfunction. The velocity of the mitral annulus can be recorded throughout the cardiac cycle using the tissue Doppler method [4].

[*]Echocardiography Unit, AL-Fallujah Teaching Hospital, AL-Fallujah, Al-Anbar, Iraq.
[**]Department of Medicine, College of Medicine, University of Baghdad, Baghdad, Iraq.

This study was designed to assess LV myocardial stiffness in hypertensive patients non invasively utilizing an echo-Doppler and TDI parameters according to the pressure volume relation ((E/E')/LVEDD).

MATERIALS AND METHODS:

The study included 81 hypertensive patients (48 females and 33 males with a mean age of 47.7±8.59 years), with range of (30-59), and 33 healthy subjects (23females and 10 males with a mean age 41.6±6 years) with range of(30-59) as a control group. This study was conducted in the medical ward at Baghdad Teaching Hospital/ Medical City over the period February-June 2013. Patients with hypertension were having BP >140/90 mmHg as a mean of at least three different measurements at one-week intervals while the normal with BP <130/85&with HR between (60-90). All patients gave their informed consent for sharing in this study. Patients with valvular heart disease, cardiomyopathy, diabetes, smokers, hyperlipidemic patients or coronary artery disease were not included in the study.

Complete transthoracic echocardiography (TTE) was performed utilizing (SONOACE X8) equipped with a multiHertz sector probe (2-5 Hz). Standard views and measurements were obtained according to the American Society of Echocardiography's recommendations. The accompanying TTE measurements were made using a parasternal long-axis view using two-dimensional targeted M-mode tracings. All these parameters were measured (Diameter of LA , LV end-diastolic and end-systolic diameter, LV septal and posterior end-diastolic wall thickness, ejection fraction, Fractional shortening and LV mass index).

left ventricular mass index (LVMI)=LVM (left ventricular mass)/body surface area.

The known formula used to make an assessment of mass left ventricular is: **left ventricular mass = 1.04* {(inter ventricular septum (IVSD) + left ventricular interior dimension + posterior wall (PWD))^3 - left ventricular interior dimension)^3}- 13.6 g** [4].

Pulsed wave Doppler (PWD) mitral inflow signals were obtained from the apical four-chamber view, the Doppler beam aligned parallel to the direction of flow and the sample volume at the leaflet tips. These parameters were measured (The peak early (E) and late (A) diastolic velocities well, E deceleration time, and E/A ratio).

Assessment of TDI of the mitral annulus was obtained from the apical four-chamber view, using a 1-2 mm sample volume placed at the septal and lateral sides of the mitral valve annulus. Velocities of the TDI mitral annular were obtained including (Early (E'), late (A') diastolic and systolic (S') velocities). Whole velocities of TDI were taken as a rate of the septal and lateral readings. The median values of the E' velocities were used to calculate the E/E' ratio. Calculation of Myocardial diastolic stiffness index by the equation of Stiffness index= [(E/E')/LVEDD].

Statistical Analysis

Performation of statistical analysis was carried out by using SPSS software for windows version 18 .Continuous variables have been expressed as mean ± standard deviation. Statistical significance was set below the 0.05 level.

RESULTS:

Results show that LV wall thickness and LVMI were increased in the hypertensive group compared with control group (IVS1.25±0.25vs. 0.98±0.188, p-value <0.05, PWT 1.05±0.22vs. 0.94±0.16, p-value <0.05 and LVMI 114±35.38 vs. 79.6±17.6, p-value <0.05). EF is insignificant, while LVEDD and LA diameter showed significant difference between both groups, p- value <0.05, table (1).

Table 1:Conventional M mode and two dimensional echocardiographic findings.

Characteristics	Patient	Control	p-value
LA (cm)	3.43±0.45	3.2±0.46	<0.05
LVEDD (cm)	4.83±0.68	4.6±0.58	<0.05
EF%	67.9±8.05	68±6.1	>0.05
IVS (mm)	1.25±0.25	0.98±0.188	<0.05
PW (mm)	1.05±0.22	0.94±0.16	<0.05
LVMI (g/m^2)	114±35.38	79.6±17.6	<0.05

Mitral inflow Doppler indices of LV diastolic function are presented in Table (2). The hypertensive group showed a significantly higher A wave velocity, a lower E/A ratio and increased

E(DT) compared with control group (A wave 78.19±19.8vs. 59.7±19.6, p-value <0.05, E/A 0.95± 0.27 vs. 1.3±0.17, p-value <0.05, and E(DT) was (212±43.6 vs. 165.9±28.4, p-value <0.05, on the other hand, the E wave velocity showed insignificant difference between both groups, p-value >0.05.

Table 2: Mitral inflow Doppler Indices of LV diastolic function.

Characteristics	Patient	Control	P-value
E (cm/s)	72.4±22.06	78.4±26.7	>0.05
A (cm/s)	78.19±19.8	59.7±19.6	<0.05
E\A	0.95±0.27	1.3±0.17	<0.05
E(DT)	212±43.6	165.9±28.4	<0.05

Table 3: E/E' and Stiffness index and other tissue Doppler parameters of the study population.

Characteristics	Patient	Control	p-value
E′average (cm/s)	9.8±2.2	12.2±2.3	<0.05
A′average (cm/s)	12.3±2.7	8.8±1.1	<0.05
S′average	10±2.05	9.7±1.5	>0.05
E\E′	7.6±2.6	6.5±2.4	<0.05
E\E'\LVIDd	1.59±0.66	1.4±0.5	>0.05

E(DT)= E wave deceleration time

In Table (3), mitral annular TDI early diastolic velocity E' average was significantly lower in hypertensive group compared with control group (E' average 9.8±2.2 vs. 12.2±2.3, p-value<0.05), and A' average was significantly higher in hypertensive group compared with control group (A' average 12.3±2.7 vs. 8.8±1.1, p-value <0.05) The systolic velocity S ' average showed no significant difference between both groups, p-value >0.05. The mean E/E' ratio was significantly higher in hypertensive group compared with control subject (mean E/E '7.6±2.6 vs. 6.5±2.4 , p-value <0.05). The LV stiffness index was insignificant in hypertensive compared with control subject, and LV stiffness index (1.59±0.66 vs. 1.4±0.5 , p-value >0.05).

DISCUSSION:

Measurement of The transmitral flow velocity to annular velocity ratio (E/E' index) is included in the TDI, which estimate myocardial velocities during the cardiac cycle, is regarded to be more dependable for diagnosing diastolic dysfunction [4].

An indicator of diastolic stiffness ((E/E')/LVEDD) is produced by connecting the E/E' to the LV end diastolic diameter (LVEDD). This diastolic LV myocardial stiffness echo Doppler index was researched in hypertensive patients, with results published on its utility in assessing LV stiffness in hypertensive patients. [3].

In this research the E/E' ratio was significantly higher in patients with hypertension compared with the control group. This could be due to left ventricular diastolic dysfunction and increased afterload and hence increased LVDP and filling pressure of the LV. This is in agreement with Oh, et al. [5] who presented TDI and complete Doppler echocardiography as the most practicable and reliable tool for confirming or eliminating diastolic dysfunctionand also in agreement with Kasner N., et al., who was the first to compare the accuracy of several echocardiographic Doppler techniques with invasive pressure-volume loop data in detecting diastolic dysfunction in HFNLVEF, They discovered that no single diastolic index from conventional Doppler echocardiography was sufficient to make the correct diagnosis, and that indices correlated only weakly with diastolic relaxation anomalies and not at all with LV stiffness, but that combining the TDI parameters with the LV filling index E/E′ was well suited for detecting invasively proven diastolic dysfunction. [6].

In contrast, Maurer, et al. [7] found that Diastolic dysfunction cannot be detected by Doppler echocardiography because Doppler generated diastolic measures do not provide particular information on intrinsic passive diastolic features. Increases in ventricular preload are absorbed by a normal elastic LV in individuals with normal myocardial relaxation, resulting in an increase in stroke volume and maintenance of normal left atrial (LA) pressures, according to Starling's Law. The LV, on the other hand, is unable to accept increases in volume when relaxation is hindered, resulting in an increase in LA pressure[8]. Due to its ability to correctly quantify myocardial relaxation in patients with

cardiac disease, relatively independent of LV filling pressures, and dividing E by E' forms an indicator of volume load, corrected to myocardial relaxation [9]. In this study, the combined conventional and TDI index of diastolic stiffness was insignificant in hypertensive patients compared to healthy subjects. This may be due to that most of the hypertensive patients is still in grade 1 diastolic dysfunction This is in agreement with Nagueh, et al. who showed that in grade 1 diastolic dysfunction E/E' is less than 8 this cause the filling pressure being not elevated & not that different from normal, hence the stiffness index [10].

Another cause of insignificant are because more than half of the hypertensive group were females. This study is also in agreement with Grohe, et al [11]. who demonstrated that in general, women have a lower prevalence of left ventricular hypertrophy for a given level of blood pressure than men, even when their left ventricular mass has been corrected for body surface area or body weight. This gender difference appears to be limited to premenopausal women, revealing that estrogen may be involved in the pathogenesis of left ventricular hypertrophy [12].

Another cause was because of more than half of the control group & less than half of hypertensive group are obese, this is also in agreement with Iacobellis, et al[13] who showed that obesity increases circulatory blood volume and cardiac output as a compensatory mechanism from high metabolic demands of excessive adipose tissue. This is also in agreement with Pascual, , et al.(14) who showed global LV diastolic dysfunction occurs in otherwise healthy obese persons as demonstrated by abnormalities of LV filling and prolongation of isovolumic relaxation time [13,14].

This study is in agreement with Khalid Abd El Salam [15], Who showed that when comparing hypertensive patients to healthy people, the combined conventional and TDI index of diastolic stiffness was considerably higher in hypertensive patients. This could be because certain morphologic and structural alterations in the left ventricle, such as left ventricular hypertrophy and remodeling, reduce LV compliance and increase myocardial stiffness in hypertensive patients. This is also in agreement with Lorell, et al., who suggested that Chronic hypertension is the leading cause of diastolic dysfunction and failure, as it causes LV hypertrophy and an increase in connective tissue content, both of which reduce ventricular compliance [16].

The results of this study are also in agreement with Solomon, et al., who found increased diastolic stiffness in hypertensive patients and they also found that in response to intensive lowering of BP, The extent of systolic blood pressure decrease was linked to the degree of improvement in TDI annular relaxation velocity, and patients with the lowest achievable systolic blood pressure had the highest final diastolic relaxation velocities [17].

In the present study, the LV diastolic stiffness index showed in significant correlation with the degree of left ventricular hypertrophy (LVMI and IVS thickness). These findings do not support the previous assumption that certain morphologic changes do occur in hypertensive patients leading to left ventricular hypertrophy and increased LV mass that increase myocardial stiffness and LV filling pressure [15].

CONCLUSION:

This study showed that the LV myocardial diastolic stiffness index [(E/E')/LVEDD] is no significantly different in hypertensive patients compared to control group.

REFERENCES:

1. Walker, B. R., Colledge, N. R., Stuart, H.R, Ian D.P. "Cardiovascular disease". Chapter 16,In: Davidson's principles and practice of medicine, Edinburgh London and New yourk Oxford Philadelphia St Louis Sydney Toronto,23edition, 2018.
2. Safar, M. E., Asmar, R., Benetos, A., Blacher, J., Boutouyrie, P., Lacolley, P., Interaction Between Hypertension and Arterial Stiffness An Expert Reappraisal. *Hypertension*. 2018;72:796-805.
3. Mohammed, M. K., Kadam, S. M., & Essa, S. I. Evaluation of Left Ventricle Performance in Patients with Ischemic Heart Disease Using Single Photon Emission Computed Tomography (SPECT) in Comparison with Echocardiography. Iraqi Journal of Science. 2021; 62:3871-76.
4. Armstrong W.F., Ryan T. Feigenbaum's echocardiography, chapter 6, Philadelphia. Wolter kluwer; Lippincott Williams and Wilkins, 8edition, 2019.
5. Oh, J. K., Hatle, L., Tajik, A. J., & Little, W. C. "Diastolic heart failure can be diagnosed by comprehensive two-dimensional and Doppler echocardiography". J Am Coll Cardiol. 2006; 47:500-6.

6. Kasner, M., Westermann, D., Steendijk, P., Gaub, R., Wilkenshoff, U., Weitmann, K., ... & Tschöpe, C. "Utility of Doppler echocardiography and tissue Doppler imaging in the estimation of diastolic function in heart failure with normal ejection fraction: A comparative Doppler-conductance catheterization study". Circulation. 2007;116:637-47.
7. Maurer, M. S., Spevack, D., Burkhoff, D., & Kronzon, I. "Diastolic dysfunction: Can it be diagnosed by Doppler echocardiography?". J Am Coll Cardiol. 2004; 44:1543-49.
8. Nishimura, R.A., Tajik, A.J. "Evaluation of diastolic filling of left ventricle in health and disease: Doppler echocardiography is the clinician's Rosetta Stone". J Am Coll Cardiol. 1997; 30:8-18.
9. Sohn, D. W., Chai, I. H., Lee, D. J., Kim, H. C., Kim, H. S., Oh, B. H., ... & Lee, Y. W. "Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function". J Am Coll Cardiol. 1997;30:474-80.
10. Nagueh, S. F., Smiseth, O. A., Appleton, C. P.,et al . Recommendations for the evaluation of left ventricular diastolic function by echocardiography. European Journal of Echocardiography. 2009;10:165-93.
11. Grohe C, Kahlert S, Lobbert K et al. Modulation of hypertensive heart disease by estrogen. Steroids .1996; 61: 201–4.
12. Aryan, L., Younessi, D., Zargari, M., Banerjee, S., Agopian, J., Rahman, S., ... & Eghbali, M.The role of estrogen receptors in cardiovascular disease. International Journal of Molecular Sciences. 2020; 21:4314.
13. Iacobellis G, Ribaudo MC, Leto G, et al: Influence of excess fat on cardiac morphology and function: study in uncomplicated obesity. Obes Res . 2002;10:767-773.
14. Pascual M, Pascual DA, Soria F, et al: Effects of isolated obesity on systolic and diastolic left ventricular function. *Heart.* 2003*;* 89:1152-56.
15. Ommen, S.R., Nishimura, R.A., Appleton, C.P., et al. "Clinical utility of Doppler echocardiography and tissue Doppler imaging in the estimation of left ventricular filling pressures: A comparative simultaneous Doppler-catheterization study". Circulation. 2000;102:1788-94.
16. Lorell, B.H., Carabello, B.A. "Left ventricular hypertrophy: Pathogenesis, detection and prognosis". Circulation. 2000;102:470-79.
17. Solomon, S.D., Verma ,A., Desai, A., et al. "Effect of intensive versus standard blood pressure lowering on diastolic function in patients with uncontrolled hypertension and diastolic dysfunction". Hypertension. 2010;55:241-48.