

Assessment of Left Ventricular Systolic Function by Speckle Tracking Echocardiography in Young Hypertensive Patients

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Received for publication May 11, 2022; Accepted November 01, 2022;
Published online November 01, 2022.

Citation: Ahmed M., Samy H. and Attia M. Assessment of Left Ventricular Systolic Function by Speckle Tracking Echocardiography in Young Hypertensive Patients. AIMJ. 2022; Vol.3-Issue 11 : 1-8.

doi: 10.21608/aimj.2022.137902.1942

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ABSTRACT

Background: Tissue Speckle Tracking examines speckle aberrations in an echo picture to determine myocardial contractility and relaxation. Reflections, refraction, and dispersion of echo beams generate speckles.

Aim of the study: The purpose of this research was to use speckled tracking echocardiography to identify subclinical left ventricular failure in young hypertension individuals who had normal systolic function by 2D echocardiography.

Patients and Methods: The research included 30 hypertensive patients with preserved ejection fraction referred to cardiology department at AL azhar University hospital for echocardiography assessment and 20 controls. All patients were assessed clinically followed by M-Mode and 2D echocardiography assessment, pulsed wave Doppler mitral inflow as well as offline speckle tracking echocardiography.

Result: Demographic data, anthropometric measurements, blood pressure measurements, conventional tissue Doppler, and strain echocardiography were compared between the two groups. Body mass index, systolic and diastolic blood pressure, left ventricular end diastolic dimension, interventricular septal diameter, posterior wall diameter, relative wall thickness, left ventricular mass index, end diastolic volume, E/, left atrial diameter, aortic diameter, left atrial volume index, normal systolic velocity of tissue doppler mitral inflow, and GLS.

The control group had a considerably greater systolic velocity ($p=0.013$). In comparison to the hypertension group, GLS was considerably greater in the control group (-21.25 ± 1.18 vs -19.18 ± 1.66 , $p=0.001$).

Conclusion: The final result of the research recommends comprehensive longitudinal stress assessment and tissue Doppler imaging for all newly discovered arterial hypertension patients or patients with arterial hypertension who have normal ejection fraction and suffer from shortness of breath.

Keywords: Tissue Speckle tracking; Hypertension; ejection fraction; Left ventricular systolic function.

Disclosure: The authors have no financial interest to declare in relation to the content of this article. The Article Processing Charge was paid for by the authors.

Authorship: All authors have a substantial contribution to the article.

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INTRODUCTION

As a result, early identifying of left ventricular (LV) systolic failure in afflicted individuals is critical.¹

Conventional echocardiography, on the other hand, reveals LV anomalies.

Systolic activity occurs only in late stages of hypertensive heart disease (HHD), when there is apparent LV remodeling/hypertrophy; diastolic LV malfunction, on the other hand, develops early and is simpler to identify even with traditional approaches.² Recent study on HF patients with regular ejection fraction (EF) has shown that isolated diastolic dysfunction is uncommon.

Diastolic dysfunction is commonly coupled with a subclinical systolic dysfunction.³

Tissue Doppler imaging (TDI), strain echocardiography, and MRI have all shown preclinical changes in LV systolic function in individuals with essential hypertension and normal EF percent.⁴

Speckle tracking echocardiography (STE) has lately been found to yield more information than TDI, enabling non-invasive monitoring of total LV strain and twist.⁵

The purpose of this research was to use speckled tracking echocardiography (longitudinal strain pattern) to identify subclinical left ventricular failure in young hypertension individuals who had normal systolic function by 2D echocardiography.

PATIENTS AND METHODS

Between September 2020 and February 2022, 30 patients with systemic arterial hypertension were referred to the Cardiology department at AL azhar University Hospitals for Echocardiography estimation, and 20 control subjects were referred to the Echocardiography unit for analysis of symptoms such as breathlessness and palpitations.

The eligible patients consenting to participate after informed consent were enrolled in the research. Our cardiology department's ethics committee accepted the research plan.

Inclusion criteria: Apparently healthy thirty patients known to be hypertensive on medical treatment aged between 35-45 y. who were visiting Cardiology clinic for follow up. Twenty normal age-matched subjects will be enrolled as a control group after obtaining informed permission.

Exclusion criteria: Secondary hypertension, patients with age above 45 years, patients aged below 35 years, patients with left ventricular (LV) systolic malfunction ejection fraction (EF<55% detected by M-Mode and Simpson Biplane method), patients with resting segmental wall motion abnormalities (RSWMA), or with documented Myocardial infarction (MI), patients with valvular heart disorders, or any structural heart disorder, patients with Atrial fibrillation, patients with chronic obstructive pulmonary disorder, patients with diabetes mellitus, patients with poor image quality on echocardiography and patients with hematological and oncological disease.

Methods:

Proper history getting: with a focus on age, sex, and any prior hypertension complications.

Clinical evaluation: All individuals had their heart rates, weights, and heights measured, and their body mass index was computed as $BMI = \frac{weight(kg)}{height(m)^2}$ (6).

The Mosteller formula was used to compute the body surface area as $BSA = \frac{\sqrt{HT(cm) \times WT(kg)}}{3.600}$

Normal value was ≤ 1.6 m² in females, ≤ 1.8 m² in males (WT: weight in Kg, HT: height in cm). Measurements of ABP were taken according to ESC 2018 guidelines for management of ABP: Before starting BP readings, patients should sit comfortably in a calm area for 5 minutes. Three blood pressure readings should be taken 1–2 minutes apart, with extra readings taken only if the first two readings deviate by more than 10 mmHg. The median of the latest two blood pressure measurements is used to calculate blood pressure.⁷

ECG: Searching for voltage criteria or strain pattern with exclusion of any ischemic changes or arrhythmia.

Transthoracic Echocardiography: Standard transthoracic M Mode and two dimensional echocardiography Examinations.

Transmitral pulsed wave Doppler Echocardiography: The mitral E/A ratio and DT are utilized to diagnose mitral inflow patterns regular, impaired LV relaxation, pseudo normal LV filling (PNF), and limited LV filling are some of the options.

Tissue Doppler velocity of the mitral annulus was used to determine LV systolic and diastolic functioning. Mitral annular tissue Doppler velocity in longitudinal axis at septal and lateral annular points was measured and the average of both annuli measurements was calculated. The mitral annulus systolic velocity (S), early diastolic annular velocity (E'), and late diastolic annular velocity (A') were measured. Normal value of systolic velocity of mitral annulus (S) is 5.97 ± 1.14 at septal annulus and 6.26 ± 2.44 at lateral annulus. (8) Normal value of E' is ≥ 10 cm at septal annulus and is ≥ 14 cm at lateral annulus. Normal value of E/E' is ≤ 8 . The median ratio was less than 8 in patients with adequate LV filling pressures, while those with a ratio more than 13 have increased LV filling pressures. When the ratio is between 9 and 13, further measures such as the LA volume index are required.⁸

	Normal Diastolic filling pattern	DD grade II (pseudo-normal Pattern)
E' septal	>10	<8
E' lateral	>12	<8
E/E'	5-10	9-12
S/D	S>D	S<D
Ar-A(ms)	<0	>30
LA volume index (ml/m ²)	16-28	>28

E'(early Diastolic mitral velocity), E(mitral annular inflow velocity), S(systolic velocity of pulmonary venous flow), D(diastolic velocity of PV flow), Ar (Pulmonary vein A reversal), A (Atrial kick of mitral inflow velocity).⁽¹¹⁷⁾

Table 1: Different Echocardiographic Parameters to differentiate normal LV diastolic filling pattern from pseudo normal pattern

Speckle Tracking Echocardiography: 2D- speckle tracking echocardiography was used to image longitudinal strain.

The following points were considered during the image acquisition. ECG gates of excellent quality the images were taken from the apical four chamber, two-chamber, and three-chamber perspectives, and they were all taken at about the same heart rates. The gain settings were tweaked to perfection. The depth was decreased to the point that the LV took up the majority of the picture sector. The LV was carefully measured to prevent foreshortening. The gray-scale frame rate was maintained between 50 and 90 frames per second, and each loop received at least three cardiac cycles. This guaranteed that at least one full cardiac cycle (the middle one) was accessible for examination at all times. To eliminate breathing artifacts, all of the photographs were taken while holding your breath. All of the patients had a normal sinus rhythm. From three consecutive Beats, all photos were saved in cine-loop format, and data was transmitted to a computer for additional offline processing.

Statistical Analysis: The Statistical Package for Social Sciences (SPSS) was used (SPSS version 15.0). Continuous data was represented as mean

standard deviation, whereas categorical data was given as frequencies and percentages. To analyze categorical data, the Chi square or Fisher's exact test were employed, as appropriate. In the total study population and the hypertension group, univariate and multivariate logistic regression models were employed to look for predictors of subclinical systolic malfunction. In a multiple logistic regression model, factors having P values < 0.2 on univariate analysis or variables of clinical importance were listed. The 95 percent confidence interval (CI) and unadjusted and adjusted odds ratios (OR) were utilized to find predictive value. If the P value < 0.05, it was deemed substantial.

RESULTS

M-Mode and 2D echocardiography, pulsed wave Doppler mitral inflow, offline speckle tracking echocardiography evaluation of LV systolic functions (longitudinal strain pattern), and tissue Doppler echocardiography measurement of LV systolic and diastolic function were all performed on all patients.

		Range	M	±	S.	t.	p.
			ea	n	D	tes	val
			n			t	ue
LVE DD (cm)	Hypertensive	4 - 6	5.1	± 0.6	14.53	0.38	0.01
	Control	4 - 3	4.6	± 0.4	7.41	0.7	*
LVE SD (cm)	Hypertensive	2.1 - 3.8	2.9	± 0.8	0.41	0.38	0.467
	Control	2.4 - 3.1	2.9	± 0.1	0.30	0.6	*
IVS (cm)	Hypertensive	0.9 - 1.2	1.0	± 0.1	25.10	0.27	0.01
	Control	0.7 - 1.1	0.8	± 0.1	7.11	0.7	*
PW T (cm)	Hypertensive	0.9 - 1.2	0.9	± 0.2	30.09	0.98	0.01
	Control	0.6 - 1.3	0.8	± 0.1	6.12	0.6	*
RW T	Hypertensive	0.3 - 0.45	0.3	± 0.08	15.04	0.52	0.01
	Control	0.3 - 0.39	0.3	± 0.04	5.02	0.5	*
LV MI (G/M ²)	Hypertensive	66 - 109	89	± 9.7	16.19	0.19	0.01
	Control	62 - 96	77	± 10.88	0.1	0.1	*
EDV (ml)	Hypertensive	75 - 141	113	± 20.6	23.83	0.83	0.01
	Control	69 - 115	88	± 12.8	0.8	0.8	*
ESV (ml)	Hypertensive	20 - 49	37	± 7.98	1.654	0.205	0.2
	Control	20 - 50	34	± 9.00	0.4	0.4	*
LA (cm)	Hypertensive	2.8 - 4.3	3.7	± 0.52	12.53	0.53	0.01
	Control	2.4 - 3.7	3.2	± 0.33	4.4	0.4	*
Ao (cm)	Hypertensive	2.5 - 3.8	3.0	± 0.28	19.43	0.43	0.01
	Control	2.2 - 3.5	2.6	± 0.31	2.31	0.2	*
LAV I (ml/m ²)	Hypertensive	17 - 27	22	± 2.43	12.23	0.23	0.01
	Control	16 - 25	19	± 3.07	7.07	0.7	*
LV Geometry			Hypertensive	Control	Total		
NG	N	21	17	38			
	%	70.0%	85.0%	76.0%			
CR	N	3	0	3			
	%	10.0%	.0%	6.0%			
CH	N	2	0	2			

EcH	%	6.7%	.0%	4.0%
	N	4	3	7
Total	%	13.3%	15.0%	14.0%
	N	30	20	50
	%	100.0%	100.0%	100.0%
Chi-square	X ²	3.712		
	P-value	0.294		

DD (diastolic dysfunction), IVS (interventricular septum thickness), PWT (posterior wall thickness), LVEDD (left ventricular end diastolic diameter), LVMI (left ventricular mass index), RWT (relative wall thickness), EDV (LV end diastolic volume), ESV (LV end systolic volume), C-R (concentric remodeling), C-H (concentric hypertrophy), Ec-H (eccentric hypertrophy), N-G (normal geometry), LA (left atrium), AO (aorta), LAVI (left atrial volume index).

Table 2: M-Mode and Two-dimensional Echocardiography finding in Hypertensive group and Control group

The hypertension group had substantially greater LV relative wall thickness (RWT), posterior wall thickness (PWT), and interventricular septal thickness (IVS) (p < 0.001), as well as a substantially higher LV mass index (p = 0.001). The hypertension group had substantially greater LV end diastolic diameter (LVEDD) and volumes (LVEDV). The hypertensive group also had substantially greater LA diameter (LAD), Aortic root diameter, and LA volume index (p = 0.001, 0.001, and 0.001, respectively). Table 2

Diastolic function	Hypertensive	Control	Total	
Normal	N	16	18	34
	%	53.3%	90.0%	68.0%
D. Dysfunction	N	14	2	16
	%	46.7%	10.0%	32.0%
Total	N	30	20	50
	%	100.0%	100.0%	100.0%
Chi-square	X ²	7.412		
	P-value	0.006*		

Table 3: Comparison between hypertensive patients and control group in diastolic function

Alternation in diastolic function was detected in our study. Hypertensive group had substantially greater frequency of LV diastolic malfunction. Only 16 out of 30 (53.3%) hypertensive patients had normal diastolic function versus 18 out of 20 (90%) control group had normal diastolic function (p = 0.028). Table 3

		Range	Me	±	S.	t.	p.
			an		D	test	val
							ue
É (cm/s)	Hypertensive	6.7 - 13.2	9.2	± 1.6	7.3	0.91	0.009*
	Control	8.2 - 12.4	10.39	± 1.17			
Á (cm/s)	Hypertensive	3.9 - 10.4	7.0	± 1.8	0.0	0.31	0.862
	Control	3.8 - 10.2	6.9	± 2.09			
É / Á	Hypertensive	0.9 - 2.2	1.3	± 0.3	4.9	0.79	0.00*
	Control	1 - 2.5	1.6	± 0.4			

		3	8		Range	Mean	±	S. D	t. test	p. value
<i>E` (early mitral inflow velocity by tissue Doppler), A` (atrial component of mitral inflow velocity by tissue Doppler)</i>										

Table 4: Tissue Doppler mitral annular velocities in hypertensive group and control group:

E` velocity was substantially decreased in hypertensive group (*E`*9.24±1.63 vs 10.39±1.17 on control group, p=0.009) *E`/A`* was substantially reduced in hypertensive group (1.37±0.32 vs 1.63±0.48 on control group, p=0.030). Table 4

Systolic function	Hypertensive	Control	Total
Normal	N 16	18	34
	% 53.3%	90.0%	68.0%
S. Dysfunction	N 14	2	16
	% 46.7%	10.0%	32.0%
Total	N 30	20	50
	% 100.0%	100.0%	100.0%
Chi-square	X ²	4.851	
	P-value	0.028*	

Table 5: Systolic function in hypertensive and control group

The Simpson approach revealed no substantial variation in EF between the two groups (p=0.200). The control group had a substantially larger systolic velocity (p=0.013). The control group's MAPSE was substantially larger (p=0.004). In comparison to the hypertension group, GLS was considerably greater in the control group (-21.25±1.18 vs -19.18±1.66 p=0.001). Table 5

		Range	Mean	±	S. D	t. test	p. value
EF (%)	Hypertensive	57 - 76	64.97	± 4.71	1.687	0.200	
	Control	61 - 72	66.55	± 3.35			
S (cm/s)	Hypertensive	6 - 9	7.61	± 1.12	6.611	0.013*	
	Control	7.1 - 9.9	8.36	± 0.77			
GLS (%)	Hypertensive	- .21 - .15	- .19	± 1.66	23.12	0.01*	
	Control	- .23 - .19	- .21	± 1.18	8		

S (systolic velocity of mitral inflow), *GLS* (global longitudinal strain).

Table 6: Different LV systolic parameters in hypertensive and control groups

Different LV systolic parameters in hypertensive and control groups are shown in table 6.

		Range	Mean	±	S. D	t. test	p. value
Age (years)	Normal	35 - 45	40.19	± 3.25	2.344	0.137	
	S. Dysfunction	35 - 45	38.50	± 2.71			
BMI (Kg/m²)	Normal	19.2 - 31.2	26.35	± 3.77	11.054	0.02*	
	S. Dysfunction	26.6 - 37.3	30.74	± 3.40			
SBP (mm Hg)	Normal	14 - 17	155.56	± 8.74	2.672	0.113	
	S. Dysfunction	14 - 16	150.71	± 7.30			
DBP (mm Hg)	Normal	90 - 120	102.81	± 8.75	0.185	0.671	
	S. Dysfunction	85 - 115	101.43	± 8.86			
LVE DD (cm)	Normal	4 - 6	5.06	± 0.56	1.274	0.269	
	S. Dysfunction	4.4 - 6	5.28	± 0.47			
LVE SD (cm)	Normal	2.3 - 3.6	3.02	± 0.36	0.249	0.622	
	S. Dysfunction	2.1 - 3.8	2.94	± 0.48			
IVS (cm)	Normal	0.9 - 1.1	0.97	± 0.08	2.327	0.138	
	S. Dysfunction	0.9 - 1.2	1.03	± 0.11			
PWT (cm)	Normal	0.9 - 1.2	1.00	± 0.10	0.297	0.590	
	S. Dysfunction	0.9 - 1.1	0.98	± 0.08			
LA (cm)	Normal	2.8 - 4.5	3.68	± 0.59	0.587	0.450	
	S. Dysfunction	3.1 - 4.3	3.82	± 0.44			
Ao (cm)	Normal	2.5 - 3.5	2.94	± 0.29	2.672	0.113	
	S. Dysfunction	2.5 - 3.5	3.11	± 0.26			
EF (%)	Normal	57 - 75	64.69	± 4.64	0.117	0.735	
	S. Dysfunction	58 - 76	65.29	± 4.94			
S (cm/s)	Normal	6 - 9	7.40	± 1.23	1.249	0.273	
	S. Dysfunction	6.3 - 9	7.86	± 0.98			
LAV I (ml/m²)	Normal	17 - 27	21.69	± 2.57	1.558	0.222	
	S. Dysfunction	19 - 26	22.79	± 2.19			
LVM I (G/M²)	Normal	71 - 106	89.44	± 10.60	0.052	0.822	
	S. Dysfunction	66 - 109	90.43	± 13.25			
EDV (ml)	Normal	75 - 140	107.69	± 20.55	2.837	0.103	
	S. Dysfunction	87 - 14	120.00	± 19.			

	Dysfun ction	1	29					
ESV (ml)	Normal	20	– 49	35.00	± 9.4	3.2	0.0	
	S. Dysfun ction	32	– 48	40.07	± 5.0	0	53	82
RWT	Normal	0.3	– 0.4	0.39	± 0.0	3.1	0.0	
	S. Dysfun ction	0.3	– 0.4	0.36	± 0.0	4	04	89

LVESD: left ventricular end systolic dimensions, IVS: interventricular septal thickness, PWT: posterior wall thickness, LA: left atrium. AO: aorta, EF: ejection fraction, S: systolic wave of tissue Doppler of mitral annulus, LAVI: left atrial volume index, LVMI: left ventricular mass index, EDV: end diastolic volume, ESV: end systolic volume, RWT: relative wall thickness.

Table 7: Patients with and without LV systolic dysfunction in the hypertensive population were compared.

The BMI of hypertensive individuals with decreased GLS was substantially greater than that of those with normal GLS (statistically substantial p=0.006). PWT was greater in hypertensive individuals with lower GLS compared to those with normal GLS (statistically substantial, p=0.02). EDV & ESV were also substantially greater in hypertensive individuals with low GLS compared to those with normal GLS (p values were 0.01 & 0.04 respectively). Patients with impaired GLS showed lower S wave velocity values than those with normal GLS in the hypertension group (despite being statistically non-significant). Table 7

DISCUSSION

Hypertension is one of the most prevalent noncommunicable illnesses and a serious risk factor for early mortality.⁹

Left ventricular hypertrophy is seen in around 25% to 50% of those afflicted (LVH). In systemic hypertension, left ventricular hypertrophy is a potential risk for asymptomatic LV malfunction and congestive heart failure.¹⁰

While traditional echocardiography may identify changes in LV diastolic malfunction connected with LVH, global LV systolic function is generally intact until late in the disease's course, which can be overlooked in the early stages.¹¹

As a result, myocardial strain may be employed to determine subclinical changes in LV systolic function. A new echocardiographic approach for analyzing LV strains is two-dimensional speckle tracking.¹²

The following were the study's key findings:

As regards demographic characteristics among the studied groups, we found that the investigation group included 30 hypertensive patients, eight patients were males (27 %) and twenty-two were females (73 %) aged from 35 to 45 years with a mean of (39.40±3.08) years, hypertension lasted anywhere from 1 to 18 years. There was no statistically substantial variation between the analyzed groups regarding age, gender, or height, but statistically substantial differences were found in terms of weight, BMI, SBP, and DBP.

The current results can be supported by Daskalov et al.¹³. 150 people were enlisted, comprising 80 HTN patients (414 years old) and 70 age and gender matched healthy controls (378 years old). Regarding the research, there were no substantial variations between the analyzed groups regarding age or sex, however there were statistically substantial variations in terms of BMI, SBP, and DBP.

In addition, the research by Ayoub et al.¹⁴ agrees with our findings. 60 hypertensive patients (ages 21 to 49, duration of hypertension 1 to 18 years) and 30 healthy controls were included in the study. In terms of age or gender, the research showed no major variations between the two groups. BMI was substantially greater in patients with systemic hypertension than in the control group (P = 0.03). Patients with systemic hypertension had substantially greater systolic and diastolic blood pressure than the control group (P< 0.001).

Regarding the M-Mode, two-dimensional echocardiography findings, the current study showed that the hypertension group had substantially larger LV relative wall thickness (RWT), posterior wall thickness (PWT), and interventricular septal thickness (IVS) (p<0.001), as well as a substantially greater LV mass index (p= 0.001). LV end diastolic diameter (LVEDD) and volumes (LVEDV) were substantially greater in hypertensive group (p= 0.001), Also LA diameter (LAD), Aortic root diameter and LA volume index were substantially greater in hypertensive groups (p= 0.001, 0.001 and 0.001 respectively).

This was in harmony with the study by Baral et al.¹⁵. In this research, 240 patients were included, 158 of whom were hypertensive (average age 48.5±6.1 years, 50.6 percent female) and 82 of whom were healthy controls (average age 45.62±6.3 years, 51.2 percent female). Hypertensive patients had substantially greater posterior wall thickness and relative wall thickness (P< 0.001). In addition, the hypertensive group had a substantially greater Left Ventricular Mass Index. The worldwide LV ejection fraction (LVEF) did not vary substantially between the two groups.

Also, in accordance with Ayoub et al.¹⁴ In hypertension individuals, septal wall thickness (interventricular septum), PWT, and RWT were substantially larger (P < 0.001). In addition, the hypertensive group's LVMI was substantially larger. The worldwide LV ejection fraction (LVEF) did not vary substantially between the two groups.

In the present study regarding LV geometry, we found that in hypertensive group, 21 patients (70%) had normal LV geometry versus 9 hypertensive patients had abnormal LV geometry in the form of 3 patients having concentric remodeling (10%), 2 patients having concentric hypertrophy (6.7%), and 4 patients having eccentric hypertrophy (13.3%). However, in control group 17 subject (85%) had normal LV geometry versus 3 subjects had abnormal LV geometry in the form of eccentric hypertrophy (15%), none of the control group subject had concentric hypertrophy nor concentric remodeling. We also found that abnormal LV geometry was more common

in hypertensive group (30%) versus (15%) only in control group.

In line with our results Shehata et al.¹⁷ reported that 16 percent of hypertension patients had normal LV geometry, 18 percent had concentric remodeling, 40% had concentric hypertrophy, and 26% had eccentric hypertrophy. Concentric hypertrophy had a greater EF ($62.8 \pm 8.3\%$) than other forms of changed LV shape ($p = 0.037$).

Furthermore, the study by Silangei et al.¹⁷ reported that abnormal LV geometry was more frequent in hypertensive patients and significantly correlated with the duration of HTN.

Regarding LV diastolic function in hypertensive and control group, the current study showed that hypertensive group had significantly higher frequency of LV diastolic dysfunction. Only 16 out of 30 (53.3%) hypertensive patients had normal diastolic function versus 18 out of 20 (90%) control group had normal diastolic function ($p=0.028$).

In accordance with Daskalov et al.¹³ reported that hypertensive group had substantially higher frequency of LV diastolic dysfunction. Only 15% hypertensive patients had normal diastolic function versus 18 out of 96.9% control group had normal diastolic function ($p=0.038$).

Regarding the Tissue Doppler mitral annular velocities in hypertensive group and control group, the current study showed that E' velocity was substantially lower in hypertensive group ($E' 9.24 \pm 1.63$ vs 10.39 ± 1.17 on control group, $p=0.009$) E'/A' was substantially lower in hypertensive group (1.37 ± 0.32 vs 1.63 ± 0.48 on control group, $p=0.030$).

In line with our findings Ayoub et al.¹⁴ E' , E'/A' , and S' velocities were all substantially lower in hypertension individuals, according to the study. In the hypertensive group, however, the E/E' ratio was much larger.

Also, the study by Shehata et al.¹⁶ reported that in hypertension individuals, E' velocity was substantially decreased. In the hypertensive group, however, the E/E' ratio was much larger.

In terms of LV Systolic Function in Hypertensive and Control Groups, the Simpson technique revealed no substantial variation in EF between the two groups ($p=0.200$). The control group had a considerably greater systolic velocity ($p=0.013$). In comparison to the hypertension group, GLS was considerably greater in the control group (-21.25 ± 1.18 vs -19.18 ± 1.66 , $p=0.001$).

In line with our findings Daskalov et al.¹³ reported that The Simpson approach revealed no substantial variation in EF between the two groups ($p=0.11$). The control group had a substantially larger systolic velocity ($p=0.046$). The GLS in the control group was substantially larger than in the hypertensive group ($p < 0.001$).

Also, in harmony with our findings Ayoub et al.¹⁴ reported that there By the Simpson approach, there was no substantial variation between the two groups in terms of EF ($p > 0.05$). In individuals with systemic hypertension, GLS was substantially reduced (-20.75 ± 1.56 in the control group vs. -19.54 ± 2.43 in the hypertensive group)

Also, the study by Baral et al.¹⁵ reported that GLS was substantially reduced in individuals with systemic hypertension compared to normal controls (-19.575 ± 1.1 in the control group vs. -18.6 ± 2.06 in the hypertensive group).

In hypertensive group 14 patients (46.7%) had reduced systolic function by 2-D speckle tracking echocardiography {had $GLS > -19.1$ } (subclinical systolic dysfunction) versus 16 (53.3%) patients had normal systolic function. However, in control group 18 (90%) subject had normal systolic function by 2-D Speckle tracking echocardiography versus 2 (10%) subjects had reduced systolic function.

Also, our results were supported by Baral et al.¹⁵ who reported that in the hypertension group, 54 of 158 patients (34.2%) exhibited subclinical LV systolic dysfunction (defined as GLS less than 18%), while only 1 of 82 controls (1.2%) had preclinical LV dysfunction. This result was comparable to that of research by Saghir et al.¹⁸ when compared to control participants, hypertensive individuals with LVH showed substantially lower systolic longitudinal strain and strain rate values.

Similarly, Ayoub et al.¹⁴ reported that Only 3 of 30 controls (10%) showed subclinical LV systolic impairment, while 23 of 60 hypertension patients (38.3%) had preclinical LV systolic impairment defined as $GLS < -19.1\%$.

In the present study we found that among hypertensive group it was found that with increasing age, systolic velocity of tissue Doppler mitral annulus decreased (substantial negative association $r = -0.592$, $p=0.001$).

It was found that as LV mass index increased, Ejection fraction decreased (substantial negative association $r = -0.643$, $p=0.001$).

Also, it was found that as Body mass index increased, GLS became less negative (worsen).

The current results were supported by Ayoub et al.¹⁴ who reported that GLS and BMI had a strong positive connection ($r = 0.43$, $P = 0.0001$) in the whole investigation sample (90 participants). GLS and LVMI had a strong positive connection ($r = 0.27$, $P = 0.009$), indicating that when BMI or LVMI grew, GLS became less negative (worsen). Tissue Doppler imaging (TDI) revealed a negative but statistically insignificant connection between GLS and S' wave mitral annular velocity ($r = -0.18$, $P = 0.08$). Similarly, Simpson's approach revealed a negative statistically non - significant association between GLS and LVEF ($r = -0.04$, $P = 0.7$), eg, as S' velocity declined, GLS became less negative (worsen).

Comparison between subjects with and without LV systolic dysfunction among hypertensive group, showed that among hypertensive group, it was found that regarding age, sex distribution, and hypertension duration, there was no variation between hypertensive individuals with normal GLS and hypertensive patients with lower GLS.

The BMI of hypertensive individuals with decreased GLS was substantially larger than that of those with normal GLS (statistically substantial $p=0.006$).

Posterior wall thickness (PWT) was non substantially larger among hypertensive patients with decrease GLS than those with regular GLS (non substantial $p=0.59$).

Also, EDV & ESV were non substantially larger among hypertensive patients with reduced GLS than those with normal GLS (p values were 0.01 & 0.04 respectively).

Among hypertensive patients with decreased GLS, it was found that 60.8 % had normal LV geometry and 39.2 had abnormal LV geometry. However, among hypertensive patients with normal GLS 75.6 % of them had normal LV geometry and 24.4 % of them had abnormal LV geometry.

RWT, LV mass index, and LA volume index were all greater in hypertension individuals with lower GLS compared to those with normal GLS, although these differences were not statistically substantial.

It was found that between hypertensive, patients with lower GLS 43.47% had normal diastolic function, and 56.53% had impaired diastolic function. However, among hypertensive patients with normal GLS 54% had normal diastolic function and 48 % had impaired diastolic function (despite being statistically non-significant).

It was discovered that individuals with decreased GLS had lower S wave velocity than those with normal GLS in the hypertension group (despite being statistically non-significant).

Only BMI was shown to be substantially linked with hypertension individuals with lower GLS in the present study.

This is in line with Daskalov et al.¹³ who found that greater BMI was associated with HTN and lower GLS in individuals with HTN ($p<0.001$).

Additionally, regarding our findings Baral et al.¹⁵ only BMI was substantially greater in individuals with lower GLS than in those with normal GLS ($P <0.003$).

Similarly, Ayoub et al.¹⁴ found that among hypertensive individuals with lower GLS, only BMI was substantially greater than in those with normal GLS.

CONCLUSION

The final result of the study recommends comprehensive longitudinal stress assessment and

tissue Doppler imaging for all newly discovered arterial hypertension patients or patients with arterial hypertension who have normal ejection fraction and suffer from shortness of breath to be able to identify subfunctional left ventricular systolic function.

Conflict of interest : none

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