Al-Azhar Med. J.(Cardiology). *Vol. 50(3), July, 2021, 2107-2126*
DOI: 10.21608/amj.2021.178674
https://amj.journals.ekb.eg/article_178674.html

VALUE OF DIFFERENT ECHOCARDIOGRAPHIC MODALITIES IN THE ASSESSMENT OF LEFT VENTRICULAR HYPERTROPHY IN HYPERTENSIVE PATIENTS WITH TYPE II DIABETES MELLITUS

By

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ABSTRACT

Background: Left ventricular hypertrophy (LVH) is the response of myocytes to various stimuli leading to myocytes' hypertrophy, which occurs as a compensatory response to increased after-load. It is defined as an increase in LV mass assessed by postmortem measurements, electrocardiographic (ECG), echocardiographic and cardiovascular magnetic resonance (CMR) criteria.

Objective: To investigate the reproducibility of different echocardiographic modalities in the assessment of LVH in hypertensive patients and patients with type II DM in comparison to the golden standard CMR measurements.

Patients and Methods: We prospectively investigated 40 patients with LVH, 20 patients being hypertensive and diabetic, 20 patients being diabetic only and 20 healthy control subjects (normal ventricular function and ECG and no cardiac risk factors). In addition to clinical and conventional echocardiographic parameters, LV mass and volumes were analyzed.

Results: The comparison between new echocardiographic techniques and CMR showed that the assessments of LV volumes/LVEF by echocardiography and CMR have good correlations. However, the inter-technique agreement of absolute LV volumes revealed considerable differences, with significant underestimation of volumes and LVEF with respect to CMR. The LV mass was less in patients assessed by different algorithms of 2D echo rather than 3D echo and CMR, correlated positively with EDV and ESV.

Conclusion: 3D echocardiography attains more solid results in patients diagnosed having LVH by 2D echocardiography. CMR would be preferable for research and specific clinical conditions requiring higher accuracy and reproducibility.

Keywords: Echocardiographic, LVH, Hypertensive Patients, Type II Diabetes Mellitus.

INTRODUCTION

Left ventricular hypertrophy (LVH) is the response of myocytes to various stimuli leading to myocytes' hypertrophy, which occurs as a compensatory response to increased afterload. It is defined as an increase in LV mass, assessed by postmortem measurements, electrocardiographic (ECG), echocardiographic and Cardiovascular Magnetic Resonance (CMR) criteria. Early echocardiographic studies defined

LVH as an absolute LV mass (LVM) exceeding 250 g *(Drazner et al., 2011)*.

Regression of LVH reduces the risk of stroke, myocardial infarction and all-cause mortality. There are two main patterns of LVH: a) concentric and b) eccentric LVH. Concentric LVH is considered, when LV mass increases by wall thickening in response to pressure overload, as often in middle aged and elderly patients, is associated with lower cardiac output and predicts poor prognosis. There is a pathway from hypertension to concentric LVH without focal scar *(Yoneyama et al., 2012)*, hypertension to concentric LVH with focal scar *(Ambale-Venkatesh et al., 2014)*, concentric remodeling with myocardial infarction assessed by replacement fibrosis *(Turkbey et al., 2015)*, and concentric LVH with symptomatic vascular events and heart failure either with replacement scar *(Schelbert et al., 2012)* or without *(Chahal et al., 2015)*.

Diastolic dysfunction and/or heart failure with preserved ejection fraction (HFpEF), due to remodeling of the extracellular matrix and increase in LV filling pressures, are common in concentric LVH *(Liu et al., 2013)*. In eccentric LVH, there is an increase in LV mass without increased concentricity and is associated with higher cardiac output *(Chahal et al., 2015)*. It has not been fully clarified why patients develop a specific LVH pattern, as a response to different stimuli. Factors such as pressure, volume overload, ethnicity, gender, obesity and plasma renin levels, all seems to play a role *(Drazner, 2011)*.

Various non-invasive techniques have been used to elucidate the pattern of LVH. ECG and echocardiography were for many years the only techniques for evaluation of LVH. Although ECG measures of LVH were associated with cardiovascular disease risk in the Framingham study, the ECG evaluation of LVH lacks sensitivity and specificity, particularly in young male patients *(Bratincsák et al., 2015)*.

Discrepancy documented in diagnostic performance and agreement on predictive ability suggests that LVH by ECG and LVH by CMR are likely to be two distinct phenotypes *(Bacharova et al., 2015)*.

CMR, due to its excellent reproducibility, unrestricted field of view and non-invasive, non-radiating tissue characterization, became a powerful player for early diagnosis and treatment assessment of LVH and gender-specific values according to age and body surface area have been already published. The comparison between new echocardiographic techniques and CMR showed that the assessment of LV volumes/LVEF by echocardiography and CMR have good correlations. However, the inter-technique agreement of absolute LV volumes revealed considerable differences, with significant underestimation of volumes and LVEF with respect to CMR *(Aurich et al., 2014)*.

Another study evaluating if LVM by real-time, 3-dimensional echocardiography (RT-3DE) corresponded to CMR in patients with LVH, showed that LVM by RT-3DE correlated with that determined by CMR better than that determined by 2DE, which means that RT-3DE can overcome some of the disadvantages of 2DE in the evaluation of LVM. However, another

study, evaluating the accuracy of LVM calculation using new echocardiographic techniques in comparison with CMR in ischemic (IC) and nonischemic cardiomyopathy (non-IC), documented that although more accurate and reliable echocardiographic measurement of LVM was achieved by 3DE, underestimation and variability remained challenges in IC *(Kusunose et al., 2013)*.

Another study, evaluating 40 patients by echocardiography using 4 imaging modalities (M-mode fundamental imaging [FI], M-mode harmonic imaging [HI], two-dimensional [2D] FI and 2D HI) and CMR, showed that HI overestimates LVM, compared with FI and CMR leading to overestimation of prevalence of LVH in hypertensive patients. HI improves inter-observer reproducibility of LVM measurements, compared with FI, leading to a significant decrease in the number of patients required for clinical trials of LVM regression. Finally, the accuracy of LVM measurements by echocardiography is affected by LV geometry *(Park et al., 2014)*.

Cardiac magnetic resonance imaging (CMR) is an accurate and reliable means of evaluating cardiac morphology, and therefore very well suited for identifying and characterizing patients with various manifestations of left ventricular hypertrophy (LVH) *(Brouwer et al., 2011)*.

Within the latest 10 years, research in LVH as cardiac target organ damage has uncovered its prognostic importance. Consequently, LV mass should be accurately calculated as mass size may have important clinical implications *(Westenberg et al., 2010)*.

Brumback et al. (2016) sought for new accurate indices of LV mass. The main purpose of their study was to develop allometric indices for LV mass measured by CMR and to compare estimates of the prevalence and predictive value of LVH.

In this study we aim to evaluate the effect of type II diabetes mellitus in hypertensive patients on LV mass index and geometry by conventional echocardiography in comparison with CMR.

PATIENTS AND METHODS

This study included 60 Patients referred to the echocardiography unit of Al-Hussein University hospital for conventional echocardiographic assessment in the period from October 2017 to October 2019 and were divided into three groups. Forty patients were found out to have LVH as documented by ECG and echocardiographic criteria, according to the diagnostic criteria of American Society of Echocardiography 2017. Twenty patients had the clinical diagnosis of hypertension made according to AHA guidelines 2017, and on medical treatment. Twenty patients were diabetics, diagnosed according to ADA guidelines 2015 with documented evidence of LVH and on medical treatment; another twenty subjects were taken as a control group, (normal ventricular dimensions and function and normal ECG with no history of cardiac disease).

Exclusion criteria:

All patients with congenital heart defects, valvular heart disease, atrial fibrillation, flutter or other arrhythmias, infectous disorders, malignant tumors, as well as patients with evidence of any type

of pulmonary hypertension, chronic kidney disease, glycogen storage diseases and other hereditary disorders were excluded from the study.

All patients were studied along the following scheme:

A. Informed consent taken from all patients.

B. Complete history taking: highlighting onset, duration and complications of both hypertension and diabetes mellitus and medications received.

C. Clinical examination.

Full clinical examination was carried out on every patient with special emphasis on the following data: (1) Pulse: rate and rhythm, (2) Blood pressure, (3) Height in meters, (4) Chest and heart examination for heart sounds, additional heart sounds and murmur and the back for lung crackles.

D. Laboratory investigations: Including fasting and post prandial blood sugar, kidney functions tests and HbA1c.

E. Resting 12 lead Electrocardiography: Resting standard 12-leads electrocardiogram searching for rate, rhythm, and chamber hypertrophy. Chamber hypertrophy was concluded according to Sokolov-Lyon criteria.

F. Transthoracic Echocardiographic Examination (ASE 2015)

Echocardiographic examination to all patients was done in the left lateral decubitus position between 20 and 45 degrees using Phillips i3 device with 3.0 MHz phased array transducer. Images were acquired from the standard views (parasternal long-axis, parasternal short axis at the mid cavity level, apical four-chamber and apical five-chamber).

The transducer was placed at the third to fifth left inter-costal space and sweeps were made from the aortic root to the mitral valve and toward the apex of the left ventricle.

The following measurements were taken:

The end-diastolic left ventricular posterior wall thickness (LVPWd), inter-ventricular septal thickness (IVS,), and left ventricular internal dimension (LVIDd) were measured at the level of the chordae of the mitral valve at the peak of the R wave of the electrocardiogram using standard methods. The standard measurement convention includes the thickness of the right and left septal endocardial echoes in the IVSd and includes the posterior wall endocardial echoes in the LVPWd.

Normal left ventricular end-diastolic dimensions were considered to be the following: Left ventricular posterior wall (LVPWd) = 6-11 mm, inter-ventricular septal thickness (IVSd) = 6-11 mm, left ventricular internal dimension (LVIDd) = 35-57 mm.

Left ventricular mass was estimated from measurements using the standard convention as follows: Mass (g) = 0.77 × 10–3 × [(LVIDd + LVPWd + IVSd)3 - (LVIDd)3] + 2.4 where the various dimensions are given in millimeters.

Left ventricular mass was also estimated from measurements using the Penn convention. Measurements with the Penn convention excluded the right and left septal endocardial echo thickness from the IVSd and this convention

excludes the posterior wall endocardial echo thickness from the LVPWd. Left septal endocardial echo thickness and posterior wall endocardial echo thickness are thus included in the LVIDd by this method. Using the Penn convention, left ventricular mass is estimated as: Mass (g) = 1.04 [(LVIDd + LVPWd + IVSd)3-(LVIDd)3] - 13.6 where the dimensions are given in millimeters.

These equations permit an estimate of left ventricular mass by assuming that the ventricle is ellipsoidal during end diastole. The internal volume of the ventricle is subtracted from the external volume, which gives the volume of the ventricular muscle. Mass is estimated from the specific gravity of ventricular muscle, which is assumed to be 1.05 g/cm3.

G. CMR measurements.

A set of contiguous short axis slices covering the entire LV from the atrioventricular ring down to the apex, acquired from a cine sequence. A combination of body matrix/torso radio frequency coils was used for the acquisition, using a 2D cardiac gated pulse sequence. Ideally, images were acquired at resting lung volume. Myocardial volume is the area occupied between the endocardial and epicardial border multiplied by the interslice distance. By convention, LVM was measured at end diastole. Similar to echocardiography, LVM is the product of this volume and the density of the myocardium.

Statistical analysis:

Data was analyzed using Statistical program for Social Science (SPSS) version 25.0 for Windows. Quantitative data was expressed as mean ± standard deviation (SD). Qualitative data was expressed as frequency and percentage. Probability (p-value): p-values <0.05 was considered significant, p-values <0.001 was considered as highly significant and p-values >0.05 was considered insignificant.

RESULTS

There was no statistically significant difference between the three groups and 2D echocardiographic data regarding 2D LVEF, 2D EDV, 2D ESV, 2D SV while there was highly statistically significant difference as regard 2D IVSd, 2D PWd, 2D LV mass **(Table 1)**.

Table (1): Comparison between the studied groups regarding the 2D echocardiographic data

Parameters \ Groups		Control group No. = 20	HTN group No. = 20	DM group No. = 20	P-value
2D IVSd	Mean ± SD	9.21 ± 0.85	12.07 ± 0.25	11.98 ± 0.26	0.000
	Range	7.5 – 10.8	11.7 – 12.6	11.5 – 12.4	
2D PWd	Mean ± SD	8.95 ± 0.69	11.45 ± 0.48	11.29 ± 0.51	0.000
	Range	8 – 10	10.3 – 12	10.2 – 12.3	
2D LVEF	Mean ± SD	65.5 ± 2.7	64.20 ± 2.46	64.50 ± 2.54	0.255
	Range	62 – 70	60 – 68	60 – 69	
2D EDV	Mean ± SD	100.25 ± 7.4	101.45 ± 7.52	104.10 ± 5.38	0.199
	Range	88 – 112	89 – 112	93 – 112	
2D ESV	Mean ± SD	34.85 ± 8.15	36.20 ± 8.59	38.70 ± 4.90	0.256
	Range	22 – 52	23 – 51	30 – 46	
2D SV	Mean ± SD	65.4 ± 4.47	65.25 ± 2.99	65.40 ± 3.44	0.989
	Range	57 – 72	59 – 70	58 – 72	
2D LV mass	Mean ± SD	93.11 ± 10.91	115.91 ± 10.63	114.19 ± 8.83	0.000
	Range	83.6 – 134.5	97.4 – 131.7	100.8 – 132.6	
ASE	Mean ± SD	95.63 ± 11.45	120.05 ± 10.80	118.79 ± 10.61	0.000
	Range	81.3 – 136.7	101.5 – 133.9	100.8 – 133.7	

Variables	Post hoc analysis		
	Control Vs HTN group	Control Vs DM group	HTN Vs DM group
2D IVSd	0.000	0.000	0.616
2D PWd	0.000	0.000	0.376
2D LV mass	0.000	0.000	0.595
ASE	0.000	0.000	0.718

There was no statistically significant difference between the three groups and 3D echocardiographic data regarding 3D LVEF, 3D EDV, 3D ESV, 3D SV, while there was highly statistically significant difference as regard 3D IVSd, 3D PWd, 3D LV mass **(Table 2)**.

Table (1): Comparison between the studied groups regarding the 3D echocardiographic data

Parameters \ Groups		Control group No. = 20	HTN group No. = 20	DM group No. = 20	P-value
3D IVSd	Mean ± SD	9.21 ± 0.85	12.07 ± 0.25	11.98 ± 0.26	0.000
	Range	7.5 – 10.8	11.7 – 12.6	11.5 – 12.4	
3D PWd	Mean ± SD	8.95 ± 0.69	11.45 ± 0.48	11.29 ± 0.51	0.000
	Range	8 – 10	10.3 – 12	10.2 – 12.3	
3D LVEF	Mean ± SD	66.42 ± 2.32	65.29 ± 2.44	65.72 ± 2.25	0.314
	Range	62.6 – 70.1	62.1 – 70.4	61.4 – 69.3	
3D EDV	Mean ± SD	130.6 ± 10.9	129.70 ± 6.97	133.85 ± 4.72	0.230
	Range	98 – 142	117 – 141	125 – 140	
3D ESV	Mean ± SD	48.3 ± 6.82	49.40 ± 5.56	45.00 ± 5.43	0.061
	Range	31 – 57	39 – 58	32 – 54	
3D SV	Mean ± SD	64.25 ± 4.46	64.40 ± 3.38	64.45 ± 3.12	0.984
	Range	56 – 72	59 – 70	58 – 69	
3D LV mass	Mean ± SD	93.18 ± 12.8	123.04 ± 10.63	121.48 ± 11.64	0.000
	Range	75.7 – 140.6	102.7 – 136.6	100.9 – 136.5	

Variables	Post hoc analysis		
	Control Vs HTN group	Control Vs DM group	HTN Vs DM group
3D IVSd	0.000	0.000	0.616
3D PWd	0.000	0.000	0.376
3D LV mass	0.000	0.000	0.676

There was no statistically significant difference between the three groups and CMR data regarding CMR LVEF, CMR ESV, CMR SV, while there was highly statistically significant difference as regard CMR IVSd, CMR PWd, CMR EDV, CMR LV mass **(Table 3)**.

Table (2): Comparison between the studied groups regarding the CMR data

Parameters \ Groups		Control group No. = 20	HTN group No. = 20	DM group No. = 20	P-value
CMR IVSd	Mean ± SD	9.17 ± 0.72	12.11 ± 0.35	12.34 ± 0.27	0.000
	Range	7.6 – 10.7	11.3 – 13.1	11.8 – 12.9	
CMR PWd	Mean ± SD	9.03 ± 0.7	11.31 ± 0.45	11.64 ± 0.42	0.000
	Range	7.6 – 10.4	10.5 – 12.1	10.9 – 12.4	
CMR LVEF	Mean ± SD	65.42 ± 3.79	65.97 ± 2.84	65.85 ± 2.72	0.846
	Range	57.9 – 72.2	58.6 – 71	58.8 – 71.5	
CMR ESV	Mean ± SD	55.9 ± 7.61	51.39 ± 5.43	53.58 ± 5.83	0.089
	Range	41 – 65.9	42.4 – 59.8	43.3 – 62.4	
CMR EDV	Mean ± SD	132.99 ± 8.28	134.87 ± 5.43	120.95 ± 9.14	0.000
	Range	112.4 – 143.2	122.6 – 143	107.5 – 134.6	
CMR SV	Mean ± SD	67.18 ± 10.45	66.04 ± 5.13	67.21 ± 4.27	0.841
	Range	48.7 – 88.7	53.8 – 78.3	58.6 – 75.3	
CMR LV mass	Mean ± SD	98.6 ± 15.31	130.79 ± 8.95	125.70 ± 10.12	0.000
	Range	83 – 156	112.7 – 148	108 – 146	
Variables	**Post hoc analysis**				
	Control Vs HTN group		**Control Vs DM group**		**HTN Vs DM group**
CMR IVSd	0.000		0.000		0.142
CMR PWd	0.000		0.000		0.057
CMR EDV	0.447		0.000		0.000
CMR LV mass	0.000		0.000		0.178

There was no statistically significant difference between the three measurements in control group data regarding IVSd, PWd, LVEF, SV, while there was highly statistically significant difference as regard ESV, EDV, LV mass **(Table 4)**.

Table (3): Pairwise comparison among the control group regarding the 2D, 3D and CMR measurements

Parameters \ Control group		2D	3D	CMR	P-value
IVSd	Mean ± SD	9.21 ± 0.85	9.21 ± 0.85	9.17 ± 0.72	0.687
	Range	7.5 – 10.8	7.5 – 10.8	7.6 – 10.7	
PWd	Mean ± SD	8.95 ± 0.69	8.95 ± 0.69	9.03 ± 0.7	0.347
	Range	8 – 10	8 – 10	7.6 – 10.4	
LVEF	Mean ± SD	65.5 ± 2.7	66.42 ± 2.32	65.42 ± 3.79	0.435
	Range	62 – 70	62.6 – 70.1	57.9 – 72.2	
ESV	Mean ± SD	34.85 ± 8.15	48.3 ± 6.82	55.9 ± 7.61	0.000
	Range	22 – 52	31 – 57	41 – 65.9	
EDV	Mean ± SD	100.25 ± 7.4	130.6 ± 10.9	132.99 ± 8.28	0.000
	Range	88 – 112	98 – 142	112.4 – 143.2	
SV	Mean ± SD	65.4 ± 4.47	64.25 ± 4.46	67.18 ± 10.45	0.312
	Range	57 – 72	56 – 72	48.7 – 88.7	
LV mass	Mean ± SD	93.11 ± 10.91	93.18 ± 12.8	98.6 ± 15.31	0.000
	Range	83.6 – 134.5	75.7 – 140.6	83 – 156	

Variables	Post hoc analysis		
	2D Vs 3D	2D Vs CMR	3D Vs CMR
ESV	0.000	0.000	0.000
EDV	0.000	0.000	1.000
LV mass	1.000	0.001	0.000

There was no statistically significant difference between the measurements among the HTN group using 2D and 3D echocardiography and CMR regarding IVSd, SV, and there was statistically significant difference regarding PWd, LVEF, while there was highly statistically significant difference as regard ESV, EDV, LV mass **(Table 5)**.

Table (4): Pairwise comparison among the hypertension group regarding the 2D, 3D and CMR measurements

Parameters \ HTN group		2D	3D	CMR	P-value
IVSd	Mean ± SD	12.07 ± 0.25	12.07 ± 0.25	12.11 ± 0.35	0.759
	Range	11.7 – 12.6	11.7 – 12.6	11.3 – 13.1	
PWd	Mean ± SD	11.45 ± 0.48	11.45 ± 0.48	11.31 ± 0.45	0.042
	Range	10.3 – 12	10.3 – 12	10.5 – 12.1	
LVEF	Mean ± SD	64.20 ± 2.46	65.29 ± 2.44	65.97 ± 2.84	0.026
	Range	60 – 68	62.1 – 70.4	58.6 – 71	
ESV	Mean ± SD	36.20 ± 8.59	49.40 ± 5.56	51.39 ± 5.43	0.000
	Range	23 – 51	39 – 58	42.4 – 59.8	
EDV	Mean ± SD	101.45 ± 7.52	129.70 ± 6.97	134.87 ± 5.43	0.000
	Range	89 – 112	117 – 141	122.6 – 143	
SV	Mean ± SD	65.25 ± 2.99	64.40 ± 3.38	66.04 ± 5.13	0.410
	Range	59 – 70	59 – 70	53.8 – 78.3	
LV mass	Mean ± SD	115.91 ± 10.63	123.04 ± 10.63	130.79 ± 8.95	0.000
	Range	97.4 – 131.7	102.7 – 136.6	112.7 – 148	

Variables	Post hoc analysis		
	2D Vs 3D	2D Vs CMR	3D Vs CMR
LVEF	0.007	0.052	0.903
ESV	0.000	0.000	1.000
EDV	0.000	0.000	0.037
LV mass	0.000	0.000	0.000

There was no statistically significant difference between DM group regarding the 2D and 3D echocardiography and CMR data regarding LVEF, SV, while there was highly statistically significant difference as regard IVSd, PWd, ESV, EDV, LV mass **(Table 6)**.

Table (5): Pairwise comparison among the diabetes group regarding the 2D, 3D and CMR measurements

Parameters \ DM group		2D	3D	CMR	P-value
IVSd	Mean ± SD	11.98 ± 0.26	11.98 ± 0.26	12.34 ± 0.27	0.000
	Range	11.5 – 12.4	11.5 – 12.4	11.8 – 12.9	
PWd	Mean ± SD	11.29 ± 0.51	11.29 ± 0.51	11.64 ± 0.42	0.000
	Range	10.2 – 12.3	10.2 – 12.3	10.9 – 12.4	
LVEF	Mean ± SD	64.50 ± 2.54	65.72 ± 2.25	65.85 ± 2.72	0.109
	Range	60 – 69	61.4 – 69.3	58.8 – 71.5	
ESV	Mean ± SD	38.70 ± 4.90	45.00 ± 5.43	53.58 ± 5.83	0.000
	Range	30 – 46	32 – 54	43.3 – 62.4	
EDV	Mean ± SD	104.10 ± 5.38	133.85 ± 4.72	120.95 ± 9.14	0.000
	Range	93 – 112	125 – 140	107.5 – 134.6	
SV	Mean ± SD	65.40 ± 3.44	64.45 ± 3.12	67.21 ± 4.27	0.076
	Range	58 – 72	58 – 69	58.6 – 75.3	
LV mass	Mean ± SD	114.19 ± 8.83	121.48 ± 11.64	125.70 ± 10.12	0.000
	Range	100.8 – 132.6	100.9 – 136.5	108 – 146	

Variables	Post hoc analysis		
	2D Vs 3D	2D Vs CMR	3D Vs CMR
IVSd	1.000	0.000	0.000
PWd	1.000	0.000	0.000
ESV	0.003	0.000	0.000
EDV	0.000	0.000	0.000

DISCUSSION

LVM is strongly influenced by body size. However, even after adjustment for anthropometric variables, males have larger LVM than females *(Marwick et al., 2015)*. Similarly, athletes have increased LVM compared to non-athletes *(Poppe et al., 2015)*, and black men and women have larger LVM than their white or Asian counterparts *(Lang et al., 2015)*. Likewise, obesity is associated with increased LVM. The aforementioned body size- , ethnic-, and exercise-related factors are associated with increased LVM, as well as proportional increases in left ventricular (LV) volume, which initially maintains normal LV wall stress *(Poppe et al., 2015)*. Consequently, LV relative wall thickness (RWT), defined as the ratio of twice the LV infero-lateral wall thickness to the LV internal diameter measured at end-diastole, initially remains unchanged. Other factors to be considered are age and blood pressure.

Normal values for LVM are derived from studies of the general population without hypertension or obesity *(Chirinos et al., 2010)*. Separate cutoff values for body size-adjusted LVM have been used for men and women *(Su et al., 2012)*. In order to allow comparison of LVM among subjects of different body sizes, different allometric approaches have been suggested to normalize LVM *(Chirinos et al., 2010)*. However, there is controversy about the best method for indexing LVM.

Body surface area (BSA) was the first anthropometric variable used to index LVM and has shown a stronger statistical correlation than height with LVM and

better identification of hypertension-related LVH. However, indexing by BSA has been noted to minimize the effect of obesity on LVM, and, therefore, it underestimates the prevalence of obesity-related LVH. Consequently, height has also been used for indexing (either height alone or height raised to an allometric power of 1.7 or 2.7. Indexation of LVM to height raised to an allometric exponent of 2.7 (LVM/height2.7), in comparison to BSA or height alone, has shown better predictive value for CVD outcomes, better detection of obesity-related LVH, and less variability of LVM among normal individuals. *Chirinos et al.* demonstrated that indexation to LVM/height1.7 was the best method, in comparison to BSA and height2.7, to identify obesity-related LVH and was more consistently associated with CVD outcomes and all-cause mortality *(Chirinos et al., 2010)*. In a population with a low prevalence of obesity, there was no significant difference in the risk attributed to LVH regardless of the method of indexation. BSA has been widely adopted by the American Society of Echocardiography (ASE) and European Association of Cardiovascular Imaging as the preferred method for indexing LVM *(Chirinos et al., 2010)*.

There is evidence suggesting that type II DM could be associated with increased LV mass, concentric geometry/remodeling and impaired diastolic function. Type II DM can cause LVH through metabolic and not hemodynamic pathways *(Su et al., 2012)*. Over time, these structural and functional changes result in impaired systolic function and symptomatic heart failure, which are associated with worse clinical outcomes.

The presence of LVH is a strong independent predictor of cardiovascular morbidity and mortality. Direct cardiac effects of LVH include increased risk for developing congestive heart failure, an increased risk of arrhythmic events and a reduced coronary flow reserve promoting myocardial ischemic episodes. In LVH there is an association between cerebro-vascular disease and increased LV mass.

Different cardiac modalities are used for the evaluation of cardiac function, among these techniques, Echocardiography is still the commonly used in clinical practice thanks to its simple protocol and availability. In the past years, several studies have shown that the functional features computed from echocardiography are influenced by numerous factors such as heart rate and reduced image quality *(Chahal et al., 2015)*. Moreover, the main limitation of echocardiography lies in the fact that it is an operator-dependent technique, which may lead to inter and intra-observer variability as well as to measurement errors.

While echocardiography remains the most used technique in daily cardiology practice, CMRI allows an accurate global and regional assessment of cardiac wall motion abnormalities and provides additional information regarding cardiac structure and function. For the computation of global LV features using MRI, myocardial contours' (endocardial and epicardial contours) delineation through a stack of cine MRI images in short axis view is needed. Then, the software available in the acquisition console can provide the following

parameters: CO, EDV, ESV, SV, and LVEF.

Despite the diversity of techniques dedicated to the measurement of cardiac parameters and the increased number of research in this field, there is no consensus about the most reproducible and accurate method for the measurement of LV volumes and LVEF. Some studies have established a comparison of LV volumes and LVEF measurements using MRI, 2D and 3D echocardiography. Most of them have demonstrated that CMRI technique is more reproducible than 2D echocardiography since it is able to compute these functional parameters without geometric assumption and it is less dependent from the operator. In addition, they reported that the measurements obtained by 3D echo are similar to those obtained by MRI with little variation *(Augustine et al., 2018)*.

CMR tissue tagging allowed the non-invasive assessment of intra-myocardial displacement / strain by monitoring motion of specific material points spread in the myocardium *(Bratincsák et al., 2015)*. The application of this technique in large epidemiologic studies such as the Multi-Ethnic Study of Atherosclerosis (MESA) *(Bacharova et al., 2015)*, has enabled to investigate the nature of atherosclerosis in a total of 1184 asymptomatic participants (aged 45–84 years). Regional LV function was quantified by evaluating peak systolic circumferential strain (Ecc). The study proved that higher diastolic blood pressure (DBP) was associated with decreased regional LV function in asymptomatic individuals and was significantly attenuated after controlling for LVM. Furthermore, LV torsional deformation was greater in hypertensive patients, despite that they had lower circumferential shortening, because torsion in hypertension with concentric remodeling is a compensatory mechanism to maintain LVEF.

In our study, patients with type II DM and no hypertension, free of ischemic heart disease, had significantly larger LVMI in comparison to patients with both type II DM and hypertension and those with only hypertension. In the real clinical setting, patients with type II DM usually have co-existing hypertension, both known to contribute to the increase of the LVMI. Therefore, we expected to find the highest LVMI in that group, which was not the case. HTN per se is a well-known cause of LV mass increased *(Levelt et al., 2016)*. However our study investigated patients with only hypertension and had smallest LVMI. Our results were in contrast to the strong heart study which showed that the combination of type II DM and HTN lead to the highest LVMI, followed by patients with only type II DM or HTN. Largest LVMI in the type II DM group indicates the possible negative effect of hyperglycemia on LV mass increase, even before overt type II DM.

The present study demonstrated that both 2D and 3D echocardiography allowed the estimation of LV EF. However, evaluation with 2D echo and 3D Echo results in significant underestimation of LV EF. In our study, reliability of LV EF and LV measurements was superior with 3D echo. As compared with CMR, ECHO systematically overestimates LVM.

Underestimation of LVM was seen in another study involving healthy subjects.

A major contributor to the inaccuracy in LVM measurement by ECHO relates to the presupposition of an ellipsoid shaped heart. The endocardial border may not be well defined on ECHO, and left ventricular wall thickness is not uniform across all myocardial segments. Furthermore, the ECHO calculation for LVM is based on a formula that relies heavily on the internal diameter of the left ventricle. As a result, LVM determined by ECHO can vary considerably, because of fluctuations in intravascular volume and the resulting change in intracardiac volume *(Augustine et al., 2018)*. By contrast, CMR measures LVM via direct mathematical integration using 3-dimensional data that does not involve assumptions regarding cardiac geometry or reliance on left ventricular diameter *(Chirinos et al., 2010)*. CMR, unlike ECHO, is therefore less sensitive to changes in intra-vascular and by extension intra-cardiac volume. The limitations of 2-dimensional ECHO may be mitigated by the use of 3-dimensional ECHO, which correlates better with CMR.

Our study expanded on previous work by evaluating paired ECHO and CMR studies in a comparator group, the exclusion of papillary muscle mass measurement from LVM determination by CMR may have inflated the mean difference in LVM by ECHO and CMR. However, given that ECHO-based LVM measurements universally exclude papillary muscles, a direct comparison excluding papillary muscles from the CMR protocol is actually more appropriate. Nevertheless, we tried to account for this by performing a sensitivity analysis in which LVM values were adjusted with the best estimate available for papillary muscle.

Despite its limitations, ECHO is advantageous in terms of accessibility and cost. Nevertheless, when it comes to the measurement of LVM and assessment of LVH, the use of ECHO may overestimate the prevalence of LVH.

By contrast, CMR which is less sensitive to volume changes provides a more accurate LVM measurement, in recognition of this major drawback of ECHO, clinical trials in which LVM is used as an outcome measure are increasingly relying on CMR. Hence, it is clear that CMR is beginning to emerge as the reference standard in research, particularly in those with left ventricular alterations that may not conform to ECHO geometric assumptions. This study also provides further support for the use of CMR in clinical practice when accurate measurements of LVM are needed.

Our findings demonstrated a significant degree of LVM overestimation and increased LVH prevalence by ECHO in patients with type II DM compared with normal individuals, while ECHO continued to be used in routine clinical practice, evidence was mounting that CMR may afford a more accurate evaluation of cardiac remodeling and associated LVM changes in type II DM patients, thus providing a superior alternative for cardiac assessment in this population.

CONCLUSION

In the assessment of LVM, no superiority between echocardiography and

CMR may be stated at this time due to the absence of studies directly comparing the methods. Assessed by both echocardiography and CMR, LVM and LVH are reliable cardiovascular event predictors. LVM assessed by echocardiography is more practical on a clinical basis. CMR would be preferable for research and specific clinical conditions requiring higher accuracy and reproducibility.

Although detailed guidelines determining the clinical indications of CMR in hypertension are still missing, CMR can provide early and highly reproducible evaluation of LVH and remodeling, not available by any other non-invasive technique; furthermore, the capability to perform tissue characterization facilitates the early diagnosis and better risk stratification of micro-, macro-vascular ischemia and fibrosis, commonly found in hypertensive patients, with potentially high impact on their treatment and also on health care costs.

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قيمة الموجات فوق الصوتية للقلب في تقييم تضخم البطين الأيسر في المرضي المصابين بارتفاع ضغط الدم من داء السكري من النوع الثاني

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خلفية البحث: تضخم البطين الأيسر هو استجابة الخلايا العضلية لمحفزات مختلفة تؤدي إلى تضخم الخلايا العضلية، والذي يحدث كاستجابة تعويضية للزيادة بعد التحميل. يتم تعريفه على أنه زيادة في كتلة البطين الايسر يتم تقييمها من خلال قياسات ما بعد الوفاة وتخطيط القلب الكهربائي وتخطيط صدى القلب ومعايير الرنين المغناطيسي للقلب والأوعية الدموية.

الهدف من البحث: كان الهدف من هذه الدراسة هو التحقيق في إمكانية استنساخ طرق مختلفة لتخطيط صدى القلب في تقييم تضخم البطين الأيسر في مرضى ارتفاع ضغط الدم والمرضى المصابين بالنوع الثاني من الداء السكري مقارنةً بالقياسات القياسية الذهبية للرنين المغناطيسي للقلب.

المرضي وطرق البحث: قمنا بفحص مستقبلي لـ 40 مريضًا مصابًا بتضخم عضلة القلب، و 20 مريضًا يعانون من ارتفاع ضغط الدم والسكري، و20 مريضًا يعانون من مرض السكري فقط و20 مريضًا يتمتعون بصحة جيدة (وظيفة البطين الطبيعية وتخطيط القلب ولا توجد عوامل خطر على القلب). بالإضافة إلى معدلات تخطيط صدى القلب السريرية والتقليدية، تم تحليل كتلة وأحجام البطين الايسر.

نتائج البحث: أظهرت المقارنة بين تقنيات تخطيط صدى القلب الجديدة والرنين المغناطيسي للقلب أن تقييم أحجام البطين الايسر/ الكسر القذفي للبطين الايسر عن طريق تخطيط صدى القلب والرنين المغناطيسي للقلب له علاقة جيدة. ومع ذلك، كشف الاتفاق بين التقنيات لأحجام البطين الايسر المطلقة عن اختلافات كبيرة، مع تقليل كبير في تقدير الأحجام والكسر القذفي للبطين الايسر. فيما يتعلق بالرنين

المغناطيسي للقلب، كانت كتلة البطين الايسر أقل في المرضى الذين تم تقييمهم بواسطة خوارزميات مختلفة من الصدى ثنائي الأبعاد بدلاً من صدى ثلاثي الأبعاد والرنين المغناطيسي للقلب، مرتبطة بشكل إيجابي مع الكم النهائي الانبساطي والكم النهائي الانقباضي.

الاستنتاج: يحقق تخطيط صدى القلب ثلاثي الأبعاد نتائج أكثر صلابة في المرضى الذين تم تشخيص إصابتهم بتضخم عضلة القلب بواسطة تخطيط صدى القلب ثنائي الأبعاد. سيكون الرنين المغناطيسي للقلب مفضلاً للبحث والظروف السريرية المحددة التي تتطلب دقة أعلى.

الكلمات الدالة: تخطيط صدى القلب، تضخم عضلة القلب، مرضى ارتفاع ضغط الدم، داء السكري من النوع الثاني.