



Is there a latent left ventricular dysfunction in hypertensive patients with preserved ejection fraction ?

Y'a-t-il une dysfonction VG latente chez l'hypertendu avec une fraction d'éjection conservée à l'échocardiographie standard ?

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RÉSUMÉ

Introduction : Chez les patients hypertendus, la fraction d'éjection ventriculaire gauche (FEVG) est longtemps préservée. Cependant, cette dernière ne reflète que la fonction contractile globale et ne prend pas en considération les anomalies régionales de la contractilité.

Objectif : Déterminer chez les patients hypertendus asymptomatiques ayant une fraction d'éjection VG conservée, une atteinte latente de la fonction contractile du VG, non détectée par l'échocardiographie conventionnelle.

Méthodes : Etude prospective cas-témoin colligeant 121 hypertendus (67 M/54 F) et 39 sujets sains sans différence significative d'âge et de répartition selon le sexe. Une échocardiographie standard et une étude du strain longitudinal ont été réalisées chez tous les patients.

Résultats : La moyenne d'âge était de $60,48 \pm 10$ ans. L'épaisseur septale, la masse du VG ainsi que la surface de L'OG et le volume de L'OG étaient significativement plus élevés chez les patients hypertendus. Seulement 20 patients (16,5 %) avaient une hypertrophie ventriculaire gauche (HVG). L'architecture du VG était normale chez 57,8 % (n=70) des patients. Une différence statistiquement significative entre les deux groupes a été constatée pour tous les paramètres de la fonction diastolique, à l'exception du rapport Em /Ea et du DTEm. L'étude du strain longitudinal global (SLG) du VG en speckletracking a montré une baisse significative du SLG par rapport aux sujets sains (P=0.000), cette baisse est plus marquée chez les patients avec une HVG.

Conclusion : Une atteinte latente de la fonction systolique VG peut être démasquée par une étude du 2D strain longitudinal du VG, Ceci peut avoir comme conséquence un renforcement des mesures thérapeutiques pour prévenir l'évolution vers l'insuffisance cardiaque clinique.

Mots clés : Speckletracking, 2D strain, hypertension artérielle, échocardiographie, fonction ventriculaire gauche, pressions de remplissage du VG, facteur de risque.

SUMMARY

Introduction: Early detection of left ventricular (LV) dysfunction may represent a clinical finding that would justify aggressive treatment aimed to reduce cardiovascular morbidity and mortality.

Aim: To evaluate longitudinal contractility in patients with essential hypertension and preserved LV ejection fraction (EF), in an attempt to detect latent impairment of LV systolic function.

Methods: Prospective case-control study, carried out on 121 (67 male/54 female) hypertensive patients (HTN group) with preserved EF and without any symptoms of heart failure and 39 age- and gender-matched healthy subjects as a control group. Conventional echocardiographic study, as well as 2D Longitudinal strain imaging by 2D-speckle tracking echocardiography (2D-STE), were performed.

Results: Mean age of patients was $60,48 \pm 10.5$ years old. The LV end-diastolic diameter and LVEF were comparable between the two groups. Hypertensive patients had greater septal thickness, left ventricular mass, and maximum left atrium volume (p respectively at 0.02; 0.04; and 0.01). Only 20 patients (16.5%) had left ventricular hypertrophy (LVH). The architecture of LV was normal in 57.8 % (n=70) patients. A statistically significant difference between the two groups was found for all diastolic function parameters except Em /Ea ratio and DTEm.

In comparison with normal controls, GLS was significantly attenuated in patients with HTN (-17.69 ± 4.06 % versus -22.70 ± 5.02 % in controls (p=0.000) and 67 (55.4%) hypertensive patients had a GLS < -20% (in absolute value). The decrease of GLS was more marked in the hypertensive group with left ventricular hypertrophy.

Conclusion: The results of our study confirmed that GLS is a sensitive biomarker of subclinical myocardial dysfunction in hypertensive patients, this suggests that identifying patients at higher risk for heart failure and earlier intervention may be beneficial.

Keywords: Two-dimensional speckle-tracking, strain, arterial hypertension echocardiography, left ventricular function, left ventricular filling pressure, risk factor.

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INTRODUCTION

Arterial hypertension (HTN) is a major public health issue. According to a Tunisian survey in 2012 (1), the prevalence of hypertension was 30.6%. Only 38.8% of those with hypertension were aware of their diagnosis, of which 84.8% were receiving treatment. Blood pressure control was achieved in only 24.1% of treated hypertensive patients. The ongoing relationship between the level of high blood pressure and cardiovascular risk is ascertained. HTN prevalence is increasing steadily. It is the most common cardiovascular risk factor and is the 2nd leading cause of cardiac death after coronary artery disease. Clinical evaluation and assessment of hypertension-mediated organ damage is an essential step in the proper management of the hypertensive patient in the latest guidelines (2).

The progression of hypertension to congestive heart failure is well established. It goes from a reversible stage of left ventricular hypertrophy (LVH) and diastolic dysfunction to an irreversible stage of systolic dysfunction (3).

Although the study of diastolic function has been well codified by the European recommendations of 2015, the study of systolic function remains limited to two-dimensional echocardiography, in particular, the calculation of Simpson's left ventricular ejection fraction (LVEF), which remains preserved for a long time in hypertension (4,5).

Detection of subclinical left ventricular dysfunction still challenging, few studies have been published to confirm the usefulness of the longitudinal strain in preserved LVEF hypertensive patients by detecting latent left ventricular damage not found by conventional echocardiographic measurements (6) but recent developments using T1 mapping in CMR allow for noninvasive assessment. Studies using T1 mapping have shown an increase in extracellular volume fraction (ECV).

Our study aimed to evaluate longitudinal contractility in patients with essential hypertension and preserved LVEF, by studying the longitudinal two-dimensional strain, in an attempt to detect latent impairment of systolic function.

METHODS

Study Population

We conducted a prospective case-control study. We included 121 eligible subjects who are hypertensive

patients followed-up in our department of cardiology over 3 years from January 2015 to December 2017. All participants gave informed consent to participate in the study. Patients were excluded if they had secondary hypertension, diabetes, kidney failure, congestive heart failure or LVEF < 50%, chronic respiratory disease, suspected or known coronary artery disease (chest pain, stress angina), more than a moderate valvular disease, rhythm disorder, chronic inflammatory disease, poor echogenicity, or age over 75 years. Patients with HTN were compared, to 39 age- and sex-matched healthy patients as a control group.

Data collection: Full history taking and general and cardiac clinical examinations were done before selection. Data collection was achieved using a medical questionnaire containing information about medical history, blood pressure (BP) and heart rate measurements, physical examination, and transthoracic echocardiographic data. Measurements of blood pressure (BP) were taken according to the American Heart Association and American College of Cardiology 2017 guidelines for the management of BP (7).

Echocardiography :

All echocardiograms were performed by the same operator. The ultrasound machine used was a General Electric Vivid9. Left ventricular diameters and function were assessed according to the ASE/ECAVI guidelines (8). The examination was standardized and conducted as follows:

- 1- Connecting the patient to the EKG of the echograph and obtaining a good quality single-lead trace.
- 2- Long axis para-sternal view where we measure parietal thicknesses (posterior wall (PW) and interventricular septum (IVS)) and left ventricle (LV) diameters (end-diastolic diameter (EDD) and end-systolic diameter (ESD)) in TM mode.
- 3- The analysis of the left ventricular mass (LVM) is calculated automatically according to the linear method of the American Society of Ultrasound (2) using the formula integrated into the machine ($LVM = 1.04[(IVS + LV\ EDD + PW)^3 - (LV\ EDD)^3] + 0.6\ g$). The threshold values are defined at $95\ g/m^2$ in women and $115\ g/m^2$ in men according to current guidelines (8).

4- Small axis para-sternal view.

5- Apical views with :

- Acquisition of the LV cavity contours by end-diastole and end-systole in 4 and 2-chambers views to determine LVEF by the Simpson biplane method.

- Acquisition of the trans-mitral flow with pulsed Doppler (the Em wave and its deceleration time (TDEm), the Am wave, the Em/Am ratio).

- Tissue Doppler analysis of the left atrioventricular ring displacements (the Ea wave in the septal and lateral wall as well as their average, the lateral Aa wave, and the Em/Ea ratio).

- Acquisition of 3, 4, and 2-chambers views over 3 consecutive cardiac cycles at a rate of 70 images per second for the study of the longitudinal strain.

6- The classification of LV geometry into one of four patterns based on LVM and relative wall thickness (RWT=2PW/EDD) : normal geometry (Normal LVM and a RWT < 0.42) ; concentric hypertrophy (elevated LVM and RWT >0.42) ; eccentric hypertrophy (elevated LVM and RWT < 0.42) ; or concentric remodeling (normal LVM and RWT > 0.42)

7- Longitudinal Strain acquisition: Data was analyzed offline using dedicated automated software (EchoPAC PC, Version 113; GE Health VIVID 9). The image rate required is optimally between 50 and 70 MHz. The apical 3, 4, and 2-chambers views are performed successively over 3 cardiac cycles by asking the patient to perform an apnea for better image quality. The software is highly dependent on the quality of the high-resolution image and is applied in harmonic imaging. On end-systolic images, the LV myocardium is traced by manual or semi-automatic endocardial contouring depending on the software used. The software then automatically defines the epicardial and endocardial lines and processes all the images included in the acquired loop. The endocardial boundary is identified by edge detection, based on the recognition of the black-and-white transition in a single image. The myocardium is defined by the empirical estimation of myocardial thickening. One can choose to accept or readjust the edges. The analysis is done in the following order: 3, 4 then 2 chambers view. For each view, the result will be an average longitudinal Strain value as well as values for each segment. The final result will be in

the form of a bull's eye image, where the Strain values for each segment will be displayed. A global value or Global Longitudinal Strain (GLS) will be given representing the result of the values in the 3 apical sections (Figure 1a et 1b).

Normal values of GLS vary from 15.9 to 22.1% in different publications (3). We considered altered GLS for values <-20% (absolute value) according to the latest recommendation of the American society of echocardiography (8).

« Septal bulge » is when the thickness of the basal septum \geq 2mm compared to the middle septum. It is considered to be an early echographic morphological criterion in hypertensive patients (6) but recent developments using T1 mapping in CMR allow for noninvasive assessment. Studies using T1 mapping have shown an increase in extracellular volume fraction (ECV). This echocardiographic sign has been looked for in hypertensive patients and healthy controls.

We studied the correlation between the conventional echocardiography and Speckle tracking, comparing the echocardiographic parameters (IVS, LVM, LVEF and EDD) and GLS values in the evaluation of left systolic function in hypertensive patients with preserved LVEF.

Statistical analysis

Data entry and analysis were performed using SPSS 24.0 software. The results for the various parameters are expressed for continuous variables as a mean \pm standard deviation (SD).

Inter-group comparisons are performed using the t-Student test for continuous variables and the Chi-Student test for categorical variables.

Correlations between the different parameters were tested by Bravais Pearson linear correlation. Their significance threshold is then tested by the t-Student test.

RESULTS

General characteristics of the population:

The study group has an average age of 60.48 ± 10.5 years old, and the sex ratio is 1.24 (67male). The average duration of HTN was 8.36 ± 6.37 years. HTN group and

controls were comparable in terms of age, sex, body surface area, and heart rate.

Conventional echocardiographic characteristics (table 1) :

The EDD and LVEF were comparable between the two groups.

Hypertensive patients had the greater septal thickness and left ventricular mass (LVM) with a statistically significant difference. The maximum volume of the left atrium (LA) was significantly greater in HTN group.

In hypertensive patients, only 20 patients (16.5%) had left ventricular hypertrophy (LVH). The architecture of LV in hypertensive patients was normal in 57.8% (n=70). Concentric remodeling was seen in 10% (n=12), concentric hypertrophy in 5.8% (n=7) and eccentric hypertrophy in 10.7% (n=13).

There was also a statistically significant difference between the two groups for all diastolic function parameters (except Em /Ea ratio and DTEm).

Longitudinal strain study :

The global longitudinal strain was significantly lower in the hypertensive group compared to the control group, with values of $-17.69 \pm 4.06\%$ versus $-22.70 \pm 5.02\%$ ($p=0.000$) Figure 2. Results of the regional 2D strain study are summarized in Table 1.

In our series, 67 (55.4%) hypertensive patients had a $GLS < -20\%$ (in absolute value).

GLS impairment in hypertensive patients is more evident in the presence of left ventricular hypertrophy than in its absence, with values of -16.46 ± 4.19 and 19.36 ± 4.34 , respectively ($p=0.04$).

Septal thickness showed a significant negative correlation with GLS ($r=-0.4$ $p=0.000$). A significant negative correlation between GLS and systolic pulmonary artery pressure (SPAP) was also found ($r=-0.53$ $p=0.00$). A positive correlation was determined between GLS and Ea ($r=0.23$ $p=0.05$) and no correlation between GLS and LVM, EDD, or LVEF (Table 2).

In our series, we found a septal bulge in 34 (28%) hypertensive patients, versus eight in the control group, which is a statistically significant difference ($p=0.01$).

Table 1. Conventional and 2d strain echocardiographic data

	HTN (+)	HTN (-)	P value
LV EDD (mm)	48.08 ± 5.94	46.76 ± 4.07	0.82
LV ESD (mm)	30.28 ± 4.64	29.46 ± 4.86	0.44
IVS in diastole (mm)	9.58 ± 1.8	8.48 ± 1.6	0.02
PW (mm)	9.10 ± 1.53	8.66 ± 1.31	0.14
LVM indexed to body surface area (g/m ²)	91,28±29,47	75,98±18,23	0,04
LVEF (%)	66.2 ± 4.71	68.21 ± 5.22	0.25
LA volume indexed to body surface area (ml/m ²)	32.27 ± 10.87	26.5 ± 7.17	0.01
Diameter of the LA	40.45 ± 5.51	38.82 ± 4.67	0.16
Em (cm/s)	70.94 ± 17.88	85.00 ± 18.40	0.02
Am (cm/s)	81.32 ± 18.65	66.83 ± 16.77	0.001
Em/Am	0.92 ± 0.30	1.35 ± 0.46	0.000
DTm (ms)	219.37 ± 58.31	204.34 ± 48.48	0.25
Ea (cm/s)	10.08 ± 3.30	14.00 ± 4.54	0.000
Aa lateral (cm/s)	11.04 ± 2.87	9.86 ± 2.47	0.049
Em /Ea	7.52 ± 2.96	7.86 ± 6.38	0.74
PSAP	29.85 ± 5.11	23.07 ± 08.74	0.02
LV 4C Strain	-17.88 ± 4.36	-22.29 ± 3.4	0.000
LV 2C Strain	-18.01 ± 4.84	-22.56 ± 3.17	0.000
LV 3C Strain	-17.21 ± 3.25	-22.76 ± 5.04	0.000
Global LV Strain	-17.69 ± 4.06	-22.7 ± 5.02	0.000

LV= left ventricle ; EDD= end-diastolic dimension ; ESD= end-systolic dimension ; IVS= inter-ventricular septum ; PW= posterior wall ; LVM= left ventricular mass ; LVEF= left ventricular ejection fraction ; LA= left atrium ; Am=indicates transmitral atrial filling velocity; Aa=tissue Doppler late diastolic mitral annular velocity ; Em= transmitral Doppler early filling velocity; Ea= tissue Doppler early diastolic mitral annular velocity ; PSAP= pulmonary systolic arterial pressure, DT= deceleration time, HTN (+): hypertensive patients, HTN (-): Healthy controls 4C= 4 chambers; 2C= 2 chambers; 3C= 3 chambers HTN (+): hypertensive patients, HTN (-): Healthy controls

Table 2. correlations between echographic parameters and gls

		GLS
IVS	Pearson Correlation	-0,26
	P value	0,017
LVMi	Pearson Correlation	-0,123
	P value	0,29
EDD	Pearson Correlation	-0,06
	P value	0,58
LVEF	Pearson Correlation	0,03
	P value	0,83
E	Pearson Correlation	-0,33
	P value	0,004
A	Pearson Correlation	0,05
	P value	0,65
E/A	Pearson Correlation	0,23
	P value	0,15
E	Pearson Correlation	-0,23
	P value	0,05
E/é	Pearson Correlation	0,01
	P value	0,94
SPAP	Pearson Correlation	-0,53
	P value	0,000

IVS: interventricular septum; LVMi : left ventricle mass indexed to body surface ; EDD : end diastolic diameter ; LVEF : left ventricle ejection fraction. Posterior wall, SPAP: Systolic pulmonary artery pressure.

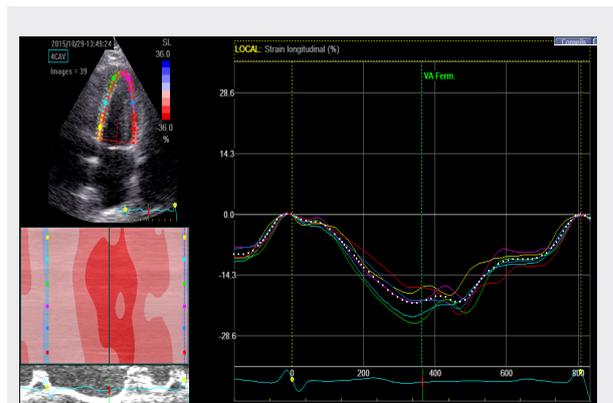


Figure 1a: Region of interest (ROI) and 2d strain curve in apical 4 chamber view

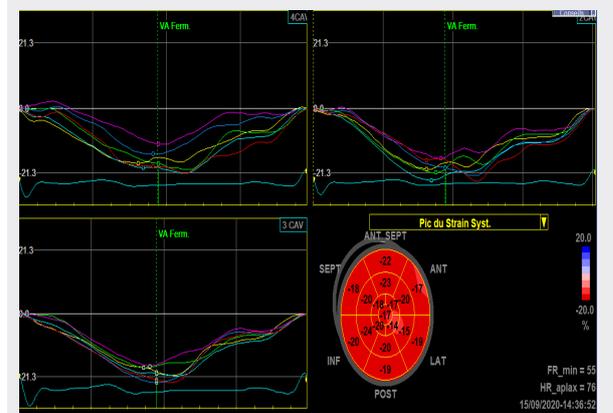


Figure 1b: Measurement of Global longitudinal 2 D strain: Bull's Eye Map in a patient with arterial hypertension.

Figure 1: Left ventricular global longitudinal strain

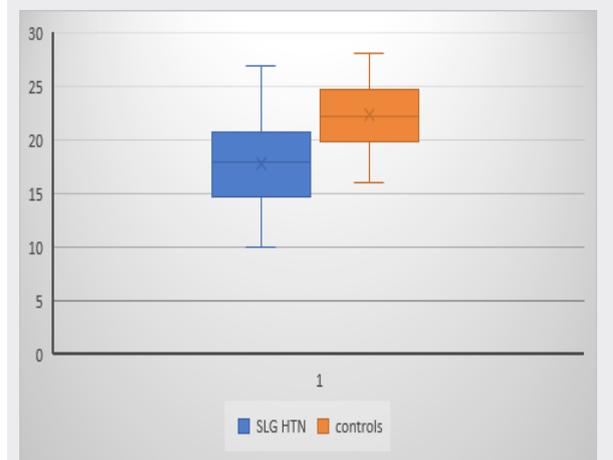


Figure 2: Mean of global longitudinal strain in hypertensive patients and in healthy controls

Table 3. Data of different published studies of literature

Study	Year	Population	Design study	Results	GLS threshold	Factors influencing the GLS	% of patients with GLS alteration	Software
Kand SJ et al. (39)	2008	56 HTN (+) VS 20 HTN (-)	HTN VS control	GLS is lower in HTN (+) group	-20.4±3%	-	-	Vivid7, EchoPacGE
Di Bello et al. (9)	2009	41 pre-HTN VS 33 HTN (+) VS 33 HTN (-)	Borderline pre-HTN VS Never-treated mild HT VS Healthycontrols	GLS is lower in pre-HTN and in mild HTN groups	-18±3.3%	SBP LVMi	-	EchoPac
Narayanan et al. (31)	2009	52 HTN (+) VS 52 HTN (-)	HTN VS control	No significant difference in GLS between the two groups	-20±3%	-	-	EchoPac, version 5.5
Kouzu et al. (32)	2010	74 HTN (+) VS 55 HTN (-)	LVH(+) VS LVH (-)	GLS was significantly reduced in LVH (+) groups compared with controls	Concentric LVH=-15.1±4% Eccentric LVH=-15.9±4.4%	E/E'	-	Echopac GE
Imblazano et al. (21)	2011	51 HTN (+) VS 51 HTN (-)	LVH(+) VS LVH (-)	-In LVH(+), GLS and SR are low. -In LVH (-), GLS is low	LVH+ :-15.9±3.3% LVH- :-18±1.9%	Systolic BP	-	Echopac v8
Afonso et al. (34)	2012	34 HTN (+) with LVH VS 56 HCM VS 27 professional athletes with LVH, VS 12 control	HTN Vs HCM Vs Athletes	HCM patients have lower GLS	-17.8±3.1%	-	-	Echopac GE
Sengupto et al. (35)	2012	34 HTN (+) VS 25 HTN (-)	HTN vs control	GLS is low while CS and RS are normal in HTN (+)	-13.4±5.8%	-	-	TomTecimaging system
Goncalves et al. (37)	2014	229 HTN(+) VS 20 HTN (-)	Ventricular geometry and HTN grades	GLS is low in HTN (+) who have concentric LVH	-19,49±2.89%	-	15,30%	Vivid7 , Echopac GE
Szelényi et al. (40)	2015	60 year old : 94 HTN (+) VS 18 HTN (-)	Diastolic dysfunction : DD	GLS is low while CS and RS are normal in HTN (+)	DD- :-15.66±1.75% DD+ :-15.35±1.88%	E/E'	-	QLAB 8.1 philips
Sun et al. (36)	2016	120 HTN (+) VS 120 HTN (-)	HTN vs control	GLS was lower in HTN (+) group	-21±3%	-	-	
Ayoub AM et al. (20)	2016	60 HTN (+) VS 30 HTN (-)	HTN vs control	Lower GLS in HTN (+) group compared to the control group	-19,10%	LVMi	38,80%	EchoPac V8
Minatoguchi Shingo et al. (33)	2017	54 HTN (-) VS 50 HTN (+) LVH (-) VS 40 HTN (+) LVH (+) VS 45 HHF(+)	-Presence of LVH -Presence of HF -LVEF	-GLS is lowest in HHF with HFREF -GLS in HTN (+) is lower compared to control -GLS in HTN(+) with LVH(+) is lower compared to LVH(-)	-	LVMi LVEF	-	Siemens Medical Solutions Inc., Mountain View, CA, USA)
Luo et al. (38)	2018	40 masked HTN (+) VS 40 HTN (-)	Masked HTN VS control	GLS is decreased in masked HTN compared to controls	-18.9±1.7%	-	-	Vivid E9 system echopac GE
Xu TY et al. (17)	2019	80 HTN (+) Vs 50 HTN (+)	HTN vs Controls	GLS in HTN(+) is lower for all 3 layers in concentric eccentric LVH(+) compared to HTN(-)	-25±2.5%NG -24.7±2.8%CR -22.8±4.7%CH -23±5.1% EH	LVMi	-	Vivid E9, echopac GE
Our study		121 HTN 39 Controls	HTN vs Controls		-18.16 ±3.97%			Vivid E9, echopac GE

HTN(+): hypertensive patient; HTN(-): normotensive patient; LVH: left ventricular hypertrophy; GLS: Global longitudinal strain; LVMi= left ventricular mass indexed to body surface; SBP: systolic blood pressure, CS: circumferential strain, RS: radial strain; HHF: hypertensive heart failure; LVEF: left ventricle ejection fraction; NG: Normal geometry; CR: Concentric remodeling; CH: Concentric remodeling; EH: Eccentric remodeling

DISCUSSION

In our present study, we used 2D strain to investigate left ventricular deformation in hypertensive patients with preserved ejection fraction. The main result is a decreased longitudinal 2D strain in hypertensive patients compared to normotensive patients, especially in the presence of left ventricular hypertrophy, and this despite normal global systolic parameters by conventional 2D echocardiography. In fact, in our series 67 hypertensive patients (65.6%) had GLS < -20%. Besides, a significant relationship had been found between GLS and LV wall thickness.

Physiopathology :

Complex changes occur in the heart adapting to the increased left ventricular (LV) workload leading to alterations in contraction and relaxation, and the evolution might go from a ventricle with concentric hypertrophy to a more dilated failing ventricle (often presenting as heart failure reduced ejection fraction (HF_rEF)) or to a heavily fibrotic and non-dilated ventricle (presenting as heart failure preserved ejection fraction (HF_pEF)), according to the three stages in the hypertrophic process (overload, hypertrophy, and failure) (3).

On a tissular level, it is demonstrated that the development of myocardial fibrosis with no growth of muscle mass is the major determinant of LV diastolic–systolic dysfunction and the occurrence of heart failure, confirming a relationship between myocardial fibrosis content and LV dysfunction (9).

These LV morphological alterations are classically detected on two-dimensional echocardiography. However, LVEF, the main parameter reflecting LV systolic function, often lead to overestimation of LV systolic performance yielding normal or even supranormal results not matching the individual's clinical situation and prognosis and do not accurately reflect the actual contractile function of the myocardium, and this is where speckle tracking echocardiography (STE) comes in (9).

Speckle Tracking :

STE is an advancement in echocardiographic techniques that allows a sophisticated better understanding of systolic function. STE is used to measure both global and regional strain, thus, STE has shown to be sensitive for the

detection of subclinical disease, including hypertensive heart disease (5).

The interest of speckle tracking in hypertensive patients :

The findings in our study extend previous observations on the topic (10-19).

Ayoub et al (20), in 2016, had conducted a study showing that LV longitudinal strain was significantly lower in hypertensive patients with preserved LVEF compared with controls; and in hypertensive patients with LVH compared to hypertensive patients without LVH.

In the same context, Imbalzano et al. (21) used STE to provide more insight into early hypertension-induced LV systolic dysfunction. In this caseseries, STE revealed an impairment of systolic longitudinal strain in all patients, including those without LVH

Moreover, in newly diagnosed hypertensive patients, GLS can unmask early subclinical systolic dysfunction even in the absence of LVH. This was demonstrated in 2010, by Di Bello, et al (9) who studied, by two-dimensional STE early left ventricular mechanics abnormalities in 74 consecutive newly diagnosed untreated hypertensive patients, and concluded that even considering only the hypertensive patients with normal LVM, a lower GLS has been found, but in a milder expression. This conclusion is also valid for pre-hypertensive stages when EF and other strain components are still normal. The « Cumulative Blood Pressure in Early Adulthood and Cardiac Dysfunction in Middle Age » study (22,23), has been demonstrated that long-time hypertension was not associated with alteration of LVEF, but with lower longitudinal systolic strain. This suggests that the use of STE surpasses the information provided by LV hypertrophy and allows identification of subclinical target organ damage that could make an individualized approach easier (23).

It is recalled that drug treatment in pre-hypertensive patients (high normal BP), is indicated only in very high-risk patients with cardiovascular disease, especially coronary artery disease (2).

In our study, we were interested mainly in longitudinal strain. In fact, among the different deformation (strain) components, the longitudinal strain has gained an important value in this context. The reason for this is

that longitudinal strain corresponds to the function of the endocardial layer of the myocardium, where longitudinal fibers are subjected to the negative impact of the early development of fibrosis in hypertensive heart disease (3). Another reason why the quantification of GLS, should be preferred in this clinical setting, is its easy feasibility and high reproducibility (24).

Hypertension is a common cause of heart failure with preserved EF (HFpEF) (25). The latest European guidelines of heart failure (HF) (26) define HF as « a clinical syndrome characterized by typical symptoms caused by a structural and/or functional cardiac abnormality, resulting in a reduced cardiac output and/ or elevated intracardiac pressures at rest or during stress ». This definition has been criticized for being restricted to symptomatic stages, while it is known, that patients can present with asymptomatic structural or functional cardiac abnormalities, which are precursors of HF, and which recognition is important as it's believed that early therapeutic control of etiologies of HF, such as hypertension, may delay or prevent the development of overt HF or prevent death before the onset of symptoms.

It is in this context that the STE is taking its full weight. Its clinical implication is various. It can be interesting in the early detection of organ damage. It is also useful as a monitoring tool predicting the response to hypertension treatment and the reversibility of structural anomalies. Beata et al (27) and Sodhi et al (28) and subsequent improvement, have on LV GLS. We hypothesized that in hospitalized patients presenting with uncontrolled hypertension (HTN, both demonstrated an improvement in GLS values in hypertensive patients who had adequate hypotensive treatment.

From a prognostic point of view, in the TOPCAT trial (29), a cohort with a high prevalence of HTN, GLS was associated with hospitalization for HF. However, this prognostic value for routine clinical use remains currently undefined. A recent study (30) concluded that GLS deterioration is associated with major adverse cardiac events in asymptomatic hypertensive patients. This finding warrants to assess in future studies the incorporation of GLS for predicting cardiovascular risk in hypertensive heart disease.

In table 3, there is a summary of the data of the literature compared to our results (9,17,20,21,31,32-34,35-40).

Limitations:

The limitations of our study include the small sample size. We also lacked close follow up, to have a thorough knowledge of medical treatment control of our hypertensive group, and correlations of their level of HTN control with their echocardiographic findings. Future studies are needed to assess the prognostic value of GLS to better manage patients with HTN.

CONCLUSION

Our study demonstrated the interest of the 2D strain in the evaluation of LV longitudinal strain in hypertensive patients with normal LVEF. A correlation between GLS and IVS thickness and between GLS and parameters of LV diastolic function has been demonstrated. These results suggest that the combination of standard and advanced echocardiography (speckle tracking echocardiography), notably the study of GLS, should be considered to improve the diagnostic accuracy, stratify the risk, and even guide the efficient course of treatment.

By demonstrating the possibility of detection of latent impairment of systolic function in hypertensive patients with preserved ejection fraction, we hope to build on this conclusion, to better detect patients at risk of developing heart failure, for closer monitoring and better control of treatment.

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