

The correlation between longitudinal strain and increased left ventricular mass in hypertensive patients: a 2D speckle tracking study

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Abstract

The geometry of the left ventricle (LV) can be altered by many pathologies such as primary structural disorders or secondary to hemodynamic conditions, with consequences to ventricular relaxation and contractility. Arterial hypertension is one of the most common causes of the left ventricular mass increase, secondary to afterload increase. Structural changes lead to subclinical alterations of contractility detectable through advanced echocardiographic techniques even in the absence of systolic dysfunction. In our study, we assessed the correlation between the changes of myocardial deformation and left ventricular mass evaluated by longitudinal strain. We performed transthoracic echocardiography to a cohort of 64 patients and assessed the LV mass and longitudinal strain using speckle tracking technique. Subsequently, all patients underwent coronary angiography to exclude significant coronary lesions. The results showed that patients with increased left ventricular mass had a more altered global longitudinal strain (GLS) compared to those with normal LV mass, regardless of the presence of coronary lesions.

Keywords: LV mass, hypertension, myocardial deformation, speckle tracking, longitudinal strain

Introduction

The left ventricle mass can be increased either by secondary hemodynamic conditions, by increasing left ventricular afterload, as it happens in aortic stenosis or hypertension, or because of primary genetically

determined diseases, such as hypertrophic cardiomyopathy or systemic diseases such as amyloidosis. High blood pressure with its high prevalence, is one of the most common causes of increased ventricular mass. Pressure overload increases parietal stress and triggers compensatory mechanisms. According to Laplace's law, parietal stress can be normalized with the thickening of the heart muscle. So, parietal stress activates neurohormonal systems which cause the release of hormones, growth factors and cytokines, promote muscle cell growth but also fibrotic inter-

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stitial changes, contributing to myocardial dysfunction (1).

Impaired systolic function appears late but contractility changes can be found earlier, with more sensitive techniques than ejection fraction. Thus, changes of LV strain may be a marker of subclinical target organ damage in hypertensive patients.

Our study aimed to highlight the correlation between LV mass and strain alteration, assessed by speckle tracking method in hypertensive patients, correlation that can play an important role in the therapeutic decision in patients with myocardial hypertrophy.

Methods

We studied a cohort of 64 patients presented in our clinic for anterior chest pain. Patients were enrolled in the study after we had ruled out cardiovascular emergencies or obvious causes of non-cardiac pain.

Their inclusion and exclusion criteria are detailed in Table 1 .

Patients were evaluated clinically with both invasive and non-invasive and techniques such as routine blood tests, electrocardiogram, 2D echocardiography and coronary angiography. Medical history was focused both on the current symptoms and on the detection of risk factors associated with cardiovascular pathologies. Life threatening pathologies were excluded.

Arterial blood pressure was measured in both arms with a sphyngomanometre at least twice, 1-2 minutes apart, after patients had rested for 3-5 minutes, and the values were reported as an average of the measurements. Arterial hypertension was defined as systolic values over 140 mmHg or diastolic values over 90 mmHg. The clinical examination has also included the calculation of BMI, obesity being defined as values above 30 kg/m².

Laboratory tests have ruled out cardiovascular emergencies (positive myocardial necrosis enzyme) and detected the associated risk factors (blood glucose, lipid profile, etc.).

After performing the electrocardiogram, we excluded patients with ischemic ECG pattern, left bundle branch block and those with arrhythmias.

Transthoracic echocardiography was performed with Vivid E9 machine, M5S probe. We used 2D echocardiography to measure chambers, wall thickness and LV mass through area-length method from short axis view. Color and spectral Doppler techniques were used to assess the heart valves and tissue Doppler was used to assess the systolic and diastolic function.

LV ejection fraction was measured with the Simpson method. The study excluded patients with difficult echo window, those with severe systolic dysfunction, pericarditis, aortic dissection or severe valvular diseases.

Images for speckle tracking evaluation we acquired (3 beats acquisitions at 50-70 FPS in 2, 3 and 4 chambers) which were later processed offline in Echo PAC workstation software. The strain was reported as global longitudinal strain GLS 18 (all 18 segments), GLS 12 (basal and medium segments) and BLS (6 basal segments).

After the non-invasive evaluation, all patients underwent coronary angiography to exclude ischemic

Table 1. Inclusion and exclusion study criteria

Inclusion criteria
Chest pain > 5 minutes in the last 24 hours
Exclusion criteria
Suboptimal echo window
ACS (STEMI, NSTEMI)
Systolic dysfunction
Myocarditis
Dilated cardiomyopathy
Severe/moderate valvulopathies
Pericardial effusion
Aortic dissection/aneurism
LBBB
Arrhythmias
Obvious non-cardiac cause

Table 2: Correlations between LV mass and GLS (entire cohort)

		Masa VS	SLG18_0	SLG12_0	SLG6_0
LV mass	Pearson Correlation	1	.473**	.523**	.495**
	Sig. (2-tailed)		.000	.000	.000
	N	64	64	64	64
GLS18	Pearson Correlation	.473**	1	.892**	.790**
	Sig. (2-tailed)	.000		.000	.000
	N	64	64	64	64
GLS12	Pearson Correlation	.523**	.892**	1	.961**
	Sig. (2-tailed)	.000	.000		.000
	N	64	64	64	64
GLS 6	Pearson Correlation	.495**	.790**	.961**	1
	Sig. (2-tailed)	.000	.000	.000	
	N	64	64	64	64

** . Correlation is significant at the 0.01 level (2-tailed).

causes for the chest pain. Significant coronary artery lesions were defined as stenosis >70%.

The data was statistically analyzed using SPSS software for Windows and was reported as mean ± standard deviation for quantitative variables and as percentages for qualitative variables. Comparisons between groups were made using the Student t test (quantitative variables) and Pearson's correlations (qualitative variables). A value of 'p' of less than 0.05 was considered statistically significant.

Results

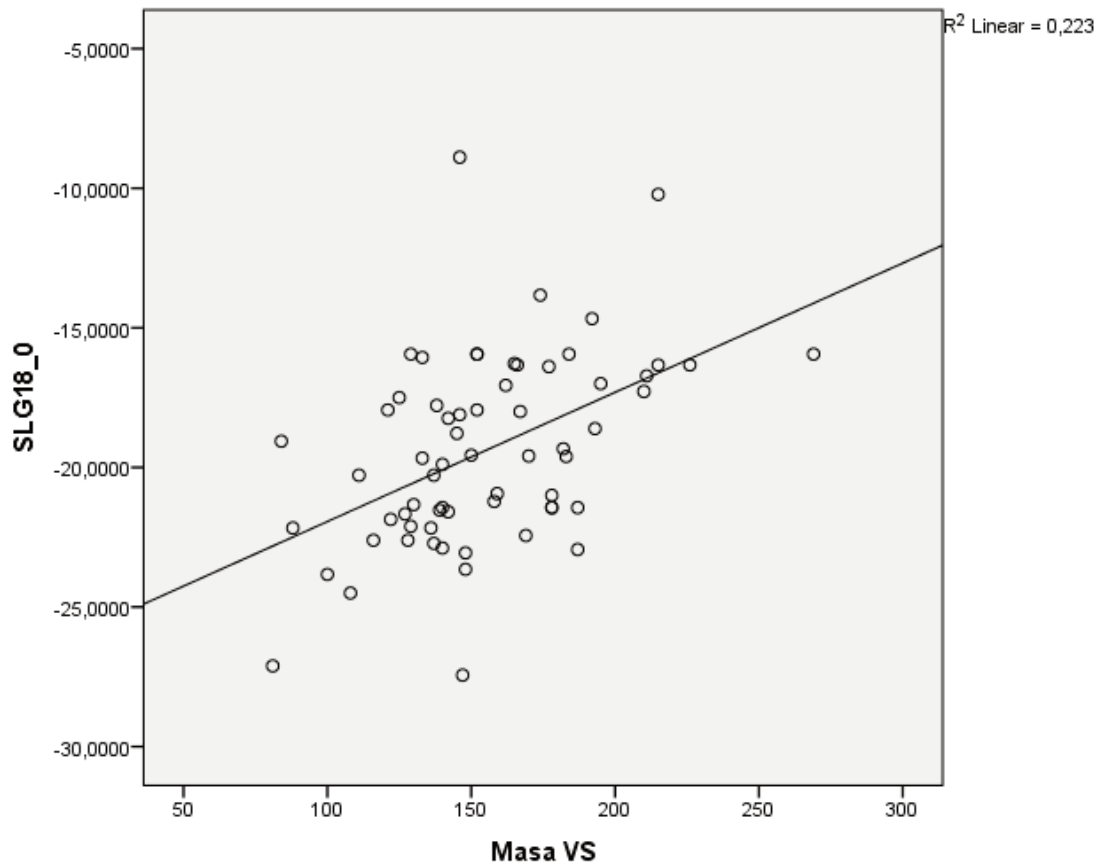
The cohort included 64 patients, average age 56.4 ± 10 years of which 32 were women and 32 were men. 17 patients (26.5%) were smokers, 18 patients (28.1%) had diabetes mellitus, 20 patients (31.25%) were obese (BMI >30 kg/m²), 47 patients (73.4%) had dyslipidemia (hypercholesterolemia or hypertriglyceridemia)

and 47 patients (73.4%) were hypertensive (SBP >140 mmHg or DBP > 90 mmHg).

Analysing the data obtained by standard 2D echocardiography, we found that hypertensive patients had a greater LV mass compared with normotensive patients: 153.7 g ± 29.5 g vs. 149 ± 34.5; but the correlation did not reached the statistical significance (t (39)-0.429, p, 0.67), probably due to the small number of patients.

In the hypertensive patients group, not only was the LV mass increased but also diabetes (31% vs. 16.6%) and dyslipidemia (85.1% vs. 38.8%) were more common.

When we studied the correlation between left ventricular mass and longitudinal strain, we obtained decreased GLS which was statistical significant for the entire cohort and for all the GLS segments studied: GLS18 (r= 0.473, p <0.001), GLS12 (r= 0.523, p <0.001) and GLS6 (r= 0.495, p <0.001) (Table 2, Graphic 1).



Graphic 1: Correlation between LV mass and GLS 18 (entire cohort).

After performing coronary angiography, patients were divided into two groups: patients with significant coronary lesions: 23 (36%) and without significant lesions: 41 (64%). We excluded patients with significant coronary lesions and we analyzed the correlation between longitudinal strain and LV mass in patients with normal epicardial coronary arteries. The statistical significant correlation maintained for all the analyzed segments: GLS18 ($r= 0.483$, $p= 0.001$), GLS12 ($r= 0.552$, $p <0.001$) and GLS6 ($r=0.564$, $p<0.001$) (Table 3).

Discussions

The study of myocardial deformation brings superior information compared to standard echocardiography and highlights subtle changes in contractility even in patients with a normal ejection fraction.

Left ventricular hypertrophy (LVH) is one of the echocardiographic signs of target organ damage but not the first. LVH is an independent marker of cardiovascular morbidity and mortality and therefore, therapeutic measures should be intensified before this stage in patients at risk.

Myocardial fibers are arranged in three layers with different orientation (longitudinal-subendocardial layer, radial and obliq subepicardial layer), that generates a complex structure of fibers orientation. This changes continuously from right handed helix in the subendocardium to left handed helix in subepicardium (2), and increases the pump function during systole (the twist). This arrangement makes possible the delimitation of three spatial layers of myocardial deformation (3).

Subendocardial layer is the most likely to be altered because it is affected by interstitial fibrosis and hypoperfusion induced by pressure overload (4) and thus, the longitudinal strain can be used for assess-

Table 3: Correlation between LV mass and GLS in patients without significant coronary lesions

		Masa VS	SLG18_0	SLG12_0	SLG6_0
LV mass	Pearson Correlation	1	.483**	.552**	.564**
	Sig. (2-tailed)		.001	.000	.000
	N	41	41	41	41
GLS18	Pearson Correlation	.483**	1	.907**	.828**
	Sig. (2-tailed)	.001		.000	.000
	N	41	41	41	41
GLS12	Pearson Correlation	.552**	.907**	1	.959**
	Sig. (2-tailed)	.000	.000		.000
	N	41	41	41	41
GLS 6	Pearson Correlation	.564**	.828**	.959**	1
	Sig. (2-tailed)	.000	.000	.000	
	N	41	41	41	41

** . Correlation is significant at the 0.01 level (2-tailed).

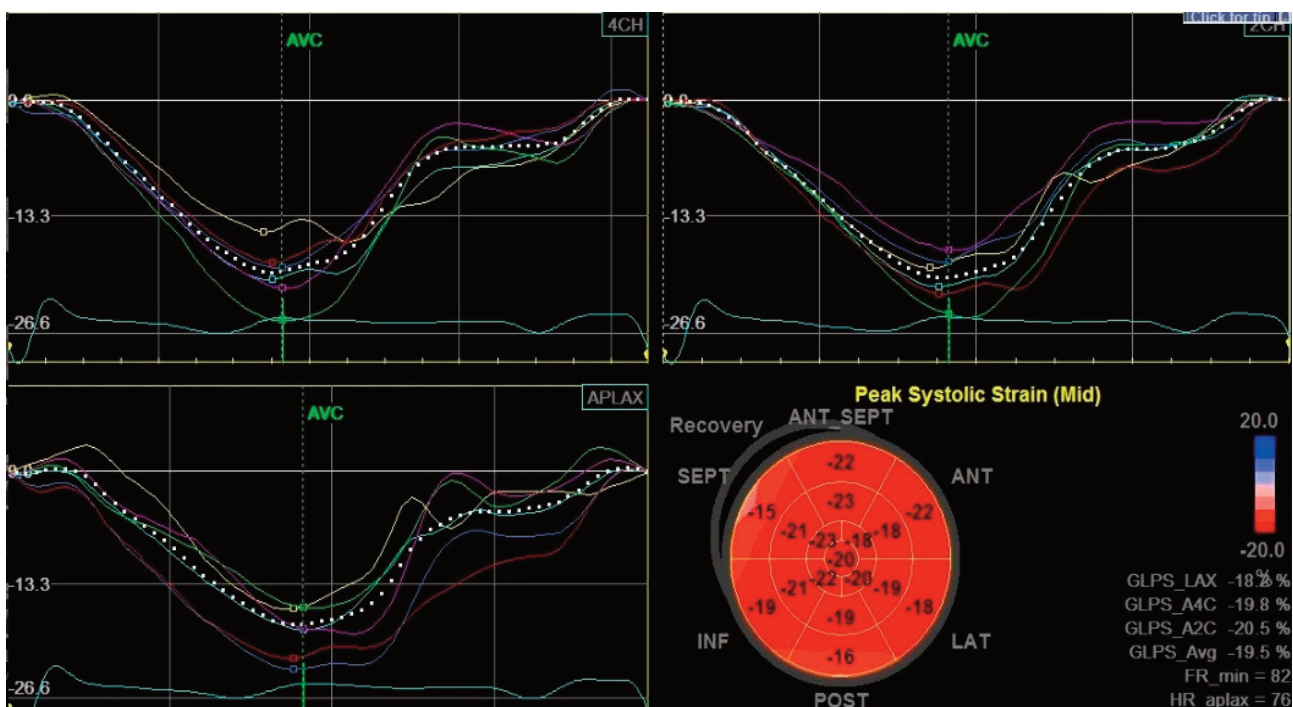


Fig 1. Bull's eye pattern in hypertensive LVH: altered strain in basal septal segments.

ment of early subclinical changes of myocardial contractility (5). Myocardial strain alteration depends on LV adaptation to blood pressure, fact that was suggested in a study based on 3D wall motion tracking analysis (6). Longitudinal strain alteration does not appear only in hypertension but also in patients with hypertrophic cardiomyopathy, diabetes mellitus or diastolic dysfunction (7, 8). In hypertensive patients, longitudinal strain alteration appears in early remodeling stage and radial strain alterations appears in more advanced stages (9, 10). Radial and circumferential strain are preserved in patients without LVH, and this supports the hypothesis that only transmural damage is correlated with reduction of EF (11). Also, in the early stages there is a compensatory increase in LV twist (10).

The data obtained by us regarding the correlation between LV mass and GLS alteration, are backed by the existing literature. Many studies have confirmed that in hypertensive patients with hypertensive left ventricular hypertrophy, the strain was more altered compared to hypertensive patients without hypertrophy (12–15). The advantage of our study is that we've ruled out a possible ischemic etiology that would have interfered with the changes secondary to hypertension.

Because the changes in longitudinal strain are not specific for a pathology, the etiology is a challenge. For this reason it has been tried various pathologies differentiation on certain criteria including the bull's eye pattern or the GLS 18.

Automatic results of the longitudinal strain analysis AFI (automated function imaging) are reported as bull's eye, a color-coded map with numeric values for pick systolic longitudinal strain of all LV segments (16). Thus, in the case of hypertensive patients, it has been found that the alterations are located at the basal septal level (septal bulge) in early stages (17, 18). When the concentric hypertrophy occurs (with normal ejection fraction), GLS is slightly altered but with significant reduction in longitudinal strain in the basal and medium segments. When ejection fraction decrease, GLS and segmental strain are clearly reduced. Similar changes can be identified in the athletic heart (16).

Bull's eye pattern obtained in our patients corresponds with the pattern described in the literature,

with a more altered strain in basal septal segments (figure 1).

In contrast, in patients with hypertrophic cardiomyopathy with asymmetrical pattern the strain is affected in hypertrophied segments. Also, in amyloidosis there's a specific pattern, with prominent alteration of the strain in the basal segments and normal strain in apical segments, with a base-to-apex gradient significantly higher than in other causes of LVH (19). Alteration of the strain in the middle segments of the posterior and lateral walls can appear in Fabry disease. (19). Also, strain changes in ischemic pathology are limited to a coronary territory. However, differential diagnosis requires additional investigation and cannot be done solely on the bull's eye pattern. This method can only suggest a possible pathology.

Also, the degree of strain alteration can provide clues for some etiologies. Afonso et al. compared in a study the longitudinal strain alteration in 56 patients with hypertrophic cardiomyopathy, 34 patients with hypertensive LV hypertrophy and 27 athletes with LV hypertrophy, and they identified that hypertensive LVH can be differentiated from cardiomyopathy using GLS 18, with a cut-off of 14.3% for GLS (with a sensitivity of 77% and a specificity of 97%), with a significant lower average GLS in patients with hypertrophic cardiomyopathy compared with hypertensive LVH (20).

Other studies have demonstrated besides the correlation between the GLS and LV mass a correlations between the strain and RWT (relative wall thickness), left atrium, MAPSE and S' wave velocity (20), so the assessment must cover all echocardiographic aspects and offer a vision of the whole. In addition, there are studies which have demonstrated correlations between GLS and clinical parameters: BMI (body mass index) (20) and between GLS and laboratory tests: the level of BNP (12). All these data from the literature supports the value of the GLS in the risk assessment of hypertensive patients.

Conclusions

Hypertensive patients tend to associate more risk factors that accumulates, increasing exponentially the cardiovascular risk. So the detection of contractility alterations before the appearance of LVH is important

in cardiovascular risk assessment and can be found by evaluating the myocardial deformation. These changes are directly proportional to the myocardial remodeling. Our study confirmed the correlation between strain alteration and LV mass, before and after the exclusion of a possible ischemic causes. In hypertensive patients with chest pain, speckle tracking method is less specific in the detection of coronary artery lesions in the absence of angiographic evaluation especially when the LV mass increases.

Acknowledgements

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Conflict of interests

There are no conflicts of interests.

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