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ROLE OF SPECKLE TRACKING ECHOCARDIOGRAPHY IN DETECTING EARLY LEFT ATRIAL DYSFUNCTION IN HYPERTENSIVE PATIENTS WITH NORMAL LEFT ATRIAL DIMENSION

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ABSTRACT

Introduction: Although conventional echocardiography is considered to be reliable for ventricular wall motion analysis and assessment of regional myocardial function, the visual estimation of wall motion are very subjective and therefore highly operator dependent. It also has high interobserver and intraobserver variability and allows only limited evaluation of radial displacement and deformation, without the possibility of assessing myocardial shortening and twisting.

Aims: Strain rate imaging (SRI) for accurate quantification of LA function in hypertensive patients and to detect an early LA dysfunction in hypertensive patients before changes in LA size represented in LA volume index. We will use 2D speckle tracking echocardiography for estimation of LA function.

Materials and Methods: This was a Observational study conducted in Institute of Cardiovascular Sciences, R.G. Kar Medical College and Hospital, Kolkata from 1 YEAR 6 MONTHS. 100 patients were included in this study.

Result: In our study though, hypertensive group had larger LA volume compared to the control group. Furthermore, LA function measured in conventional methods named total LA stroke volume and LA expansion index was more affected in the hypertensive group.

Conclusion: Asymptomatic pharmacologically treated hypertensive patients with preserved LVEF and normal diastolic function have early compromise of LA strain despite normal cavity size, suggesting an evidence for preclinical LA myocardial dysfunction.

Keywords: Echocardiography**,** blood pressure, Left atrial dysfunction and Hypertension.

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INTRODUCTION

Although conventional echocardiography is considered to be reliable for ventricular wall motion analysis and assessment of regional myocardial function, the visual estimation of wall motion are very subjective and therefore highly operator dependent. It also has high interobserver and intraobserver variability and allows only limited evaluation of radial displacement and deformation, without the possibility of assessing myocardial shortening and twisting.

During recent years, velocity imaging, displacement imaging and deformation imaging (strain and strain-rate imaging) have emerged as valuable tools for more comprehensive and reliable echocardiographic assessment of myocardial function.

For a better understanding of different echocardiographic modalities available for the assessment of myocardial contractile function, it is important to make a distinction between myocardial wall motion and wall deformation. Whereas velocity and displacement characterize wall motion, strain and strain- rate describe wall deformation. Over time a moving object will change its position (displacement) but does not undergo deformation if all its parts move with the same velocity. If, however, different parts of the object move with different velocities, the object will undergo deformation and will change its shape. Thus wall motion measurements (displacement and velocity) cannot differentiate between active and passive movement of a myocardial segment, whereas deformation analyses (strain and strain-rate imaging) allow discrimination between active and passive myocardial tissue movement. The term “strain”, which in everyday language can mean “stretching”, is used in echocardiography to describe “deformation”.

The instantaneous deformation is thus expressed relative to the initial length (Lagrangian strain). The deformation can also be expressed relative to the length at a previous time instance (natural strain) and in this definition of instantaneous strain the reference value is not constant over the time but changes during the deformation process. For small deformations the Lagrangian and natural strain are approximately equal whereas for large deformations which can occur during ventricular contraction and relaxation the difference between Lagrangian and natural strain are relevant. For myocardial strain measurements it appears more appropriate to measure the natural strain because the measured values are less dependent on the definition of the initial length L0.

For two-dimensional (2D) objects, the deformation is not limited to lengthening or shortening in one direction. A 2D object can lengthen or shorten along the x or y axis (normal strain) and can also distort (shear strain) by the relative displacement of the upper to the lower border or the right border to the left border. Thus, in two dimensions strain has four components, two normal strains and two shear strains. More complex is the deformation of three dimensional (3D) objects such as myocardial segments. In this case there are three normal strains (along the x, y and z axes) and six shear strains. To completely define the deformation of 3D objects, all nine strain components must be defined. Today, echocardiographic deformation imaging allows 1D

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measurements based on tissue Doppler imaging and 2D strain measurements based on speckle-tracking imaging.

The amount of deformation (positive or negative strain) is usually expressed in %. Positive strain values describe thickening, negative values describe shortening, of a given myocardial segment related to its original length. During myocardial contraction, as the wall shortens it also thickens and thus assessment of all parameters, radial thickening (positive strain), circumferential shortening (negative strain) and longitudinal shortening (negative strain), is useful for the evaluation of contractile function.

AIMS AND OBJECTIVE

1. Strain rate imaging (SRI) for accurate quantification of LA function in hypertensive patients.

2. To detect an early LA dysfunction in hypertensive patients before changes in LA size represented in LA volume index. We will use 2D speckle tracking echocardiography for estimation of LA function.

3. To find a correlation between different echocardiographic parameters found in hypertensive patients and LA function.

MATERIALS AND METHODS

STUDY AREA: Institute of Cardiovascular Sciences, R.G. Kar Medical College and Hospital, Kolkata

STUDY POPULATION: This study included 50 patients with arterial hypertension and 50 age matched non hypertensive control subjects. Cases are patients attend in the Cardiology department of R.G.Kar Medical College and Hospital fulfilling the inclusion and exclusion criteria. Controls will be taken from patient relatives.

Inclusion criteria:

1. Patients with systemic arterial hypertension; defined as: Systolic BP $\geq$ 140 mmHg and/or diastolic BP $\geq$ 90 mmHg or Antihypertensive treatment with a documented history of hypertension.

2. All patients should have an echocardiographic evidence of normal LA size; defined as: LA volume index < 28 ml /m2.

Exclusion criteria:

A. Cardiac conditions that affect LA size and Function:

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1-Documented coronary artery disease (defined as ; history of MI or revascularization , RWMA on echocardiography or any positive stress test) 2-Any type of cardiomyopathy (e.g Dilated,ischemic,hypertrophic)

3- Structural heart disease.

4 -Atrial fibrillation.

5- Atrial flutter

B. Others.

Diabetes mellitus, smoking, dyslipidemia and positive family history of cardiac disease, chronic kidney disease

STUDY PERIOD: 1 YEAR 6 MONTHS

STUDY DESIGN: Observational study.

RESULT AND DISCUSSION

Arterial hypertension is one of the common diseases associated with the increased incidence of heart failure and is one of the independent risk factors for atrial fibrillation through perpetual structural and functional changes in the left atrium which is in turn responsible for increased cardiovascular mortality. Hypertension alters atrial dynamics significantly and so hypertensive patients are at risk of structural and functional changes in the LA.

Choice of controls:

We used a control group of 50 healthy subjects whom we chose to be matching with the hypertensive group regarding their criteria ; age, gender, heart rate, BMI and BSA, this aimed at minimizing the impact of these factors on LA function while comparing it between both groups.

Many studies didn't match all these criteria between the diseased and control groups, in a study done by Mondillo S[1] et al.; both groups were age matched but not matching in BMI.

Regarding LV measurements:

Both groups were matching for LVEF only, but the hypertensive group had greater LV mass index and worse LV diastolic function and this was expected as a result of hypertension.

This was in concordance with a recent Egyptian study in Cairo University carried by Hassanin N[2] in 2015.

In another study in 2014, both groups had no significant difference in LVEF but more LV mass and worse diastolic function in hypertensive group.

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Regarding LA parameters:

The key point in our study was to ensure that all the studied subjects had normal LA size, and the best to express this is LA volume index. This was important for early detection of LA dysfunction before LA enlargement.

In our study though, hypertensive group had larger LA volume compared to the control group. Furthermore, LA function measured in conventional methods named total LA stroke volume and LA expansion index was more affected in the hypertensive group.

This was similar to a study done by Eshoo S[3] et al in 2009 in which hypertensive group had larger LA volume index than the control group.

This was different than a study by Cameli[4] et al in which both groups had LA volume indices with no significant difference.

A possible explanation for this difference is that the number of hypertensive patients was double the number of the control group.

Regarding global peak atrial longitudinal strain:

In our current study, global PALS were impaired in hypertensive group compared to the control group. Global PALS represents LA function, mainly the reservoir function which is affected early in patients with arterial hypertension.

In 2007, a study was conducted by Kokubu N[5] et al to test the effect of RAS inhibition on LA function, they concluded that LA deformation indices were reduced in hypertensive patients and RAS inhibitors could preserve or improve LA reservoir function.

The correlation of different risk factors and studied parameters with global LA strain:

Risk factors that were found to affect LA strain adversely are older age and higher BMI; this could be explained by that these factors have a negative impact on LA function.

Age:

This could be due to that the mean age in this study (54 years) was far below the elderly age group (above 65 years old).

Body mass index:

A recent study in 2015 stated that overweight and obese hypertensive patients had worse LA function (measured by 2D strain) compared with hypertensive patients with normal BMI.

Corresponding results were found by Miyoshi H[6] et al, BMI independently affected LA strain by 2D speckle tracking echocardiography.

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Other parameters that were related adversely to global LA strain in the current study were systolic blood pressure, LV diastolic function, LV mass index, LA volume index and LA expansion index.

Systolic blood pressure:

In a study conducted in 2014, systolic blood pressure adversely affected LA strain in hypertensive patients.

LV diastolic function:

Tsai W et al conducted a study that showed a negative impact of LV diastolic dysfunction on LA conduit function.

LV mass index:

This is compared hypertensive groups with and without high LV mass index.

LA volume index:

This correlation is somewhat logic because changes in both LA size and function are the 2 arms by which systemic hypertension affects LA hemodynamics.

LA expansion index:

In a study done by Saraiva RM[7] et al, they found that LA expansion index correlated well with global LA strain representing LA reservoir function.

This could be explained by that both LA expansion index and global LA strain represent LA reservoir function which is affected early in hypertensive patients.

LA stroke volume:

In our study, LA stroke volume didn't correlate significantly with global peak atrial longitudinal strain which could be explained by that LA stroke volume represents LA booster pump function which is not expressed fully in the global PALS.

LV ejection fraction:

Regarding LVEF, it didn't affect LA strain significantly. A possible explanation for this is that all the studied subjects in the current study had normal LVEF and so it may be of significant impact in the reduced LVEF values.

In contrary, Dogan[8] et al found a significant impact of LVEF on LA strain.

Hypertensive patients have high prevalence of LA enlargement, which is associated with impaired LA function, as measured by the STE strain imaging technique. In the absence of LA

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structural enlargement, hypertension was still associated with LA functional impairment, especially during LV systole and LA contraction, even after accounting for LV remodeling and diastolic dysfunction. Our finding on the impaired LA function as assessed by STE strain imaging during LV systole and LA contraction in the absence of LA enlargement and in the presence of hypertension .There is consensus that hypertension is associated with impaired LA function, regardless of the measurement techniques, such as the phasic/volumetric measurements, indexes of mitral and pulmonary vein flows, and tissue Doppler imaging or STE deformation indexes by strain imaging techniques. After accounting for these apparent cardiac abnormalities, we found that hypertension was still associated with LA functional impairment, suggesting that LA strain imaging is particularly sensitive in assessing LA function in hypertension. It is possible that these 2 dynamic measurements, respectively, in the heart and systemic circulation, though separated by LV, may share common or similar mechanisms of pathogeneses.

STE strain imaging therefore might be useful in the assessment of target organ damage and in the initiation of antihypertensive treatment on several conditions, such as white-coat or masked hypertension. In the presence of impaired LA function, even white-coat or masked hypertension might be treated with hypertensive drugs. Using multiple echocardiographic techniques, we found that hypertensive patients with mildly elevated blood pressure might have multiple structural and functional LA and LV abnormalities. Among others, LA enlargement had high prevalence and was closely related to LA and LV functions. This confirmatory finding might be clinically relevant.

Current standard echocardiography only measures LA diameter, and can only unveil abnormalities at a late and probably irreversible stage. In hypertension, a more thorough echocardiographic evaluation of LA may be necessary and useful in the choice of antihypertensive drugs and target blood pressure. There is some evidence that inhibitors of the renin-angiotensin system may provide more protection against LA diseases, such as atrial fibrillation. Our observation on the close association between LA enlargement and functional impairment is in line with the results of numerous previous studies involving various measuring techniques. This consistency to some extent validates the STE strain imaging technique in general and our measurements in particular. This 2-dimensional STE strain imaging technique allows simple and rapid evaluation of 3 phases of LA function, and may be used in the clinical setting.

CONCLUSION

The reproducible compromised LA myocardial function may guide towards optimizing hypertension treatment, even in the absence of clear evidence for LV dysfunction. LV diastolic dysfunction, shown by long axis amplitude and velocities should provide more accurate assessment of compromised segmental function compared with the conventionally used E/A ratio. Even in the absence of LV diastolic dysfunction LA compromised strain function should

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be considered as an early myocardial embarrassment which needs aggressive management of HTN in order to avoid perpetual deterioration of LA function and irreversible cavity remodeling.

Asymptomatic pharmacologically treated hypertensive patients with preserved LVEF and normal diastolic function have early compromise of LA strain despite normal cavity size, suggesting an evidence for preclinical LA myocardial dysfunction.

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Table: Distribution of mean LA EXPANSION INDEX: Group

		Number	Mean	SD	Minimum	Maximum	Median	T Statistic	p-value
LA EXPANSION INDEX	HTN	50	219.4000	31.8062	154.0000	278.0000	223.0000	6.1184	<0.0001
	NON HTN	50	253.5600	23.3869	198.0000	284.0000	258.5000		

Table: Distribution of mean Global PALS: LVDD

		Number	Mean	SD	Minimum	Maximum	Median	T Statistic	p-value
GLOBAL PALS	None	26	28.7500	3.4421	21.0000	34.0000	29.0000	9.6228	<0.0001
	Present	24	17.9231	4.4084	11.0000	30.0000	18.0000		

Table: Correlation of GLOBAL PALS in all parameters

		GLOBAL PALS	Remarks
AGE	Pearson Correlation Coefficient (r)	-.575	Negative Correlation
	p-value	<0.0001	Significant
	Number	50	
SBP	Pearson Correlation Coefficient (r)	-.495	Negative Correlation
	p-value	<0.0001	Significant
	Number	50	
DBP	Pearson Correlation Coefficient (r)	-.295*	Negative Correlation
	p-value	.037	Significant
	Number	50	
HR	Pearson Correlation Coefficient (r)	-.153	Negative Correlation
	p-value	.287	Not Significant
	Number	50	

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BMI	Pearson Correlation Coefficient (r)	-.751	Negative Correlation
	p-value	<0.0001	Significant
	Number	50	
LVEF	Pearson Correlation Coefficient (r)	.043	Positive Correlation
	p-value	.985	Not Significant
	Number	50	
LV MASS INDEX	Pearson Correlation Coefficient (r)	-.809	Negative Correlation
	p-value	<0.0001	Significant
	Number	50	
LA VOLUME INDEX	Pearson Correlation Coefficient (r)	-.573	Negative Correlation
	p-value	<0.0001	Significant
	Number	50	

LA STROKE VOLUME	Pearson Correlation Coefficient (r)	-.163	Negative Correlation
	p-value	.259	Not Significant
	Number	50	
LA EXPANSION INDEX	Pearson Correlation Coefficient (r)	.588	Positive Correlation
	p-value	<0.0001	Significant
	Number	50	